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Review

Wildfire-Related Air Pollution and Infectious Diseases: Systematic Review and Meta-Analysis

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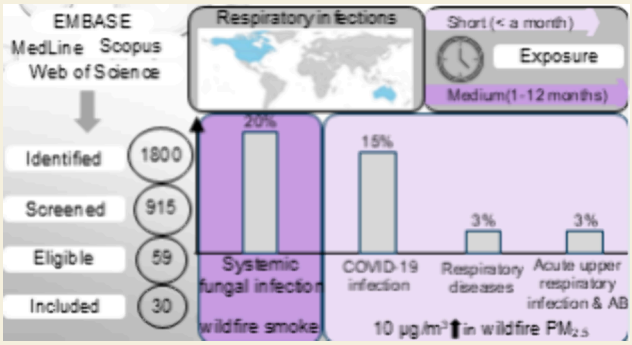
ABSTRACT: Amid the global rise in wildfire events, the health impacts of wildfire-related air pollution are increasingly scrutinized. While numerous reviews have examined the link between air pollution and infectious diseases, reviews specifically focusing on wildfire-related air pollution and infectious diseases remain scarce. To address this gap, we conducted a comprehensive search in MEDLINE, EMBASE, Scopus and Web of Science databases up to December 31, 2023, using PRISMA (Preferred Reporting Items for Systematic Reviews & Meta-Analyses) guidelines. Search terms included synonyms of wildfire and infectious diseases. Peer-reviewed epidemiological studies that reported any association or trend between wildfire air pollution and infectious diseases were selected against eligibility criteria. Risk of bias and quality of included studies were assessed using modified risk of bias and quality assessment tools. Our review included 30 studies, predominantly from developed countries including the United States (USA), Australia, and Canada. Most focused on respiratory infectious diseases (n = 29), including 9 specifically on the coronavirus disease 2019 (COVID-19). The majority examined short-term wildfire air pollution (n = 27) (exposure of one month or less). Twenty-three studies reported effect estimates for the meta-analysis. We found that a 10 $\mu g/m^3$ increase in short-term wildfire $PM_{2.5}$ (particulate matter with a diameter of 2.5 micrometer of less) exposure was associated with a 15% increase in COVID-19 infections (relative risk [RR] = 1.15; 95% confidence interval [CI]: 1.09−1.21; heterogeneity (I^2): 83%), a 3% increase in respiratory diseases (RR = 1.03; 95% CI: 1.01−1.05; I^2: 0%) and a 3% increase in acute upper respiratory infection combined with acute bronchitis (RR = 1.03; 95% CI: 1.02−1.05; I^2: 62%). Medium-term exposure (more than a month but less than a year) to wildfire smoke was associated with 20% rising hospitalization for systemic fungal infections like coccidioidomycosis (95% CI: 5−38%). The current research exclusively examines respiratory infections in developed countries. Future high-quality primary studies should prioritize understanding the impact of wildfire-related air pollution on various infectious diseases.



KEYWORDS: *Infectious disease, respiratory infection, wildfire, wildfire air pollution, wildfire $PM_{2.5}$, wildfire smoke*

1. INTRODUCTION

In recent years, the duration, frequency and severity of wildfires have been increasing worldwide in the changing climate. According to the 2024 Lancet countdown report, the population under an extremely high wildfire risk increased in 124 of 187 countries (66%) between 2003−2007 and 2019−2023.[1] Regional wildfire hotspots such as Australia, the western USA, Canada and Brazil generally experience frequent seasonal wildfires, but the wildfire seasons of 2019−2020 witnessed unprecedented numbers and severity of wildfires, particularly in Australia, USA and Brazil.[2,3] The United Nations Environment Programme projected an increase of extreme wildfires up to 30% in 2050 and 50% by 2100.[3] This increasing trend of frequency and intensity of global wildfires in the future, coupled with the capacity of wildfire smoke to travel long distances,[4] could elevate the wildfire-related air pollution in regions that were previously unaffected by wildfires.

Apart from the hazards of flame and heat, wildfires can impact human health through air pollution.[5,6] Not only in regions prone to wildfires but also in regions without wildfires people are impacted by wildfire-related air pollution, as smoke can travel long distances through atmospheric transport.[4] For example, the highest concentrations of wildfire $PM_{2.5}$ were recorded in regions typically not prone to extensive wildfires,

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such as Central Africa, Southeast Asia, South America and Siberia during 2000−2019.[7] Compared with ambient $PM_{2.5}$, previous evidence indicated that wildfire-related $PM_{2.5}$ may be more likely to increase respiratory hospitalization.[8] Considering the increasing trends of frequency and severity of wildfires, the health burden brought by wildfires will be elevated in the future. Current evidence suggests exposure to wildfire-related air pollution increases the risk of all-cause mortality, respiratory and cardiovascular morbidities, mental health disorders[9−11] and dermatological diseases.[12]

Wildfires may directly affect the spatiotemporal distribution of wildlife which serve as reservoir hosts for arboviruses and impact mosquito populations, thereby affecting the risk of mosquito-borne diseases.[13] Moreover, living bacteria and human pathogens including the fungi *Aspergillus penicillioides* found in wildfire smoke[14] traveling long distances[4] could also increase the risk of bacterial and fungal infections in the human population. Given the increasing frequency and severity of wildfires,[2] along with the rise of re-emerging (dengue, malaria)[1] and emerging infectious diseases (COVID-19),[18] the role of wildfire-related air pollution in shaping the infectious disease risk has become a growing public health concern. However, reviews on how wildfire-related air pollution affect mortality and morbidity from infectious diseases are relatively limited. A recent systematic review studied the impact of wildfire smoke exposure on respiratory infectious diseases[11] but did not explore other infectious diseases. While previous studies investigated the impacts of climate change[15,16] and air pollution[17−19] on infectious diseases, to the best of our knowledge, the association between exposure to wildfire-related air pollution and infectious diseases has not been systematically reviewed or assessed. Therefore, we aimed to systematically review and summarize the current findings on the association between wildfire-related air pollution exposure and infectious disease morbidity and mortality risks and to perform a meta-analysis for these findings.

2. METHODS

This systematic review followed the PRISMA guidelines[20] when planning, implementing and reporting this review (Table S1). The protocol for this systematic review was developed and registered at PROSPERO and can be found under the registration number CRD42023456967.

2.1. Search Strategy

We conducted a comprehensive systematic search in Ovid MEDLINE, Ovid Embase, Web of Science Core Collection and Scopus (Elsevier) for articles published from the inception of the database to 31 December 2023. We also searched for references of similar reviews and manually scanned the references of included studies, to include all the potentially relevant articles.

In the search strategy, search terms included a combination of text words and subject headings on wildfire and infectious diseases to cover all the relevant studies. We used the following synonyms for wildfire and infectious diseases and later combined those search terms: “bushfire”, “forest fire”, “wildland fire”, “wildfire”, “peat fire”, “vegetation fire”, “woodland fire”, “grassland fire”, “landscape fire”, “wildfire smoke”, “infectious disease”, “communicable disease”, “infection”. We also used terms for cause-specific infectious diseases such as “viral infection”, “bacterial infection”, “parasitic diseases”, “fungal infection”, “vector borne diseases”, “malaria”, “dengue”, “tuberculosis”, “leishmaniasis”, “influenza”, “pneumonia”, “COVID-19”, “common cold”. The detailed search strategy is described in Supporting Information (Table S2).

2.2. Selection Criteria and Screening

We considered infectious disease outcomes associated with wildfire air pollution in a general human population of all ages without restricting the population. Outcomes due to infectious diseases included: hospitalization, emergency department (ED) visit, outpatient visit. For respiratory infectious diseases, we selected pneumonia, acute upper respiratory infection, acute bronchitis and influenza that are infectious in nature and excluded noninfectious conditions such as chronic obstructive pulmonary disease (COPD) and asthma. Exposure to wildfire-related air pollution was defined as exposure to air pollutants from wildfire or wildfire events. Wildfire smoke mainly includes particulate matter, gaseous pollutants (CO, NO, NO_2), volatile organic compounds and O_3 (ozone) generated as a secondary pollutant.[5,6] We included studies reporting wildfire exposure (bush fire, forest fire, grassland fire, vegetation fire, woodland fire, landscape fire, peat fire) while excluding the studies that reported exposure to nonwildfire. Peer-reviewed epidemiological studies that reported any trends or quantitative measures of the association between wildfire-related air pollution and infectious disease related outcomes were included with a restriction to studies published in the English language. In addition, nonhuman and animal studies were excluded. Eligible study designs included cohort study, case-control study, cross-sectional study, and ecological study but excluded reviews, commentaries, editorials, letters, book chapters, conference abstracts and case reports.

The screening of identified records was conducted in two stages against the eligibility criteria. Two authors (R.M. and K.J.) screened the title and abstract of identified records in the first stage. Following the retrieval of full-texts of selected studies from first stage screening, the same authors comprehensively reviewed the full-text articles in stage two. Any disagreements were resolved by consulting the third author (R.X.).

2.3. Data Extraction

Two authors (R.M., K.J.) extracted the following data: names of authors, publication year, title, study location, study design, study period, study population, exposure (wildfire-related air pollution), exposure window (duration of exposure measured), outcomes (types of infectious diseases), ICD (International Classification of Diseases) codes for infectious diseases (if reported), sources of exposure and outcome data, statistical analysis methods, confounding variables and main findings (estimates of association or trend). Effect estimates were extracted from the best-fitted model. Since studies did not consistently use the same lag days, we focused on extracting effect estimates for lag days 0 or 1 from studies that reported effects estimates for various lag days.[21]

2.4. Quality and Risk of Bias Assessment

The included studies were critically assessed for their quality and risk of bias using relevant modified tools. Two authors (R.M., K.J.) performed the quality and risk of bias assessment independently and consulted a third author (R.X.) to resolve any disagreements. The Newcastle-Ottawa Scale (NOS) for cohort studies was used to assess the quality of the cohort study in three domains, namely, selection, comparability and outcome.[22] A total score for NOS ranged from 0 to 9 (good quality: ≥8, fair quality: 5−7, poor quality: ≤4). The quality of other observational studies (time-series, case-crossover, ecological design, longitudinal study, quasi experimental method) was assessed using a modified version of the validated quality assessment framework developed by Zaza et al.,[23] previously used in similar systematic reviews.[9,24] This framework assesses study design, sampling, validity and reliability of exposure and outcome measures, generalizability, risk of bias and reporting and studies were scored out of 24. Studies with a score of 18−24 were classified as “high quality”; a score of 10−17 was regarded as of “medium quality”; and a score of less than 10 considered as of “low quality”.

A modified version of the National Toxicology Program Office of Health Assessment and Translation (OHAT) risk of bias rating tool was used to assess the risk of bias of each included study.[9,25] This OHAT tool evaluates risk of bias in five domains including selection,

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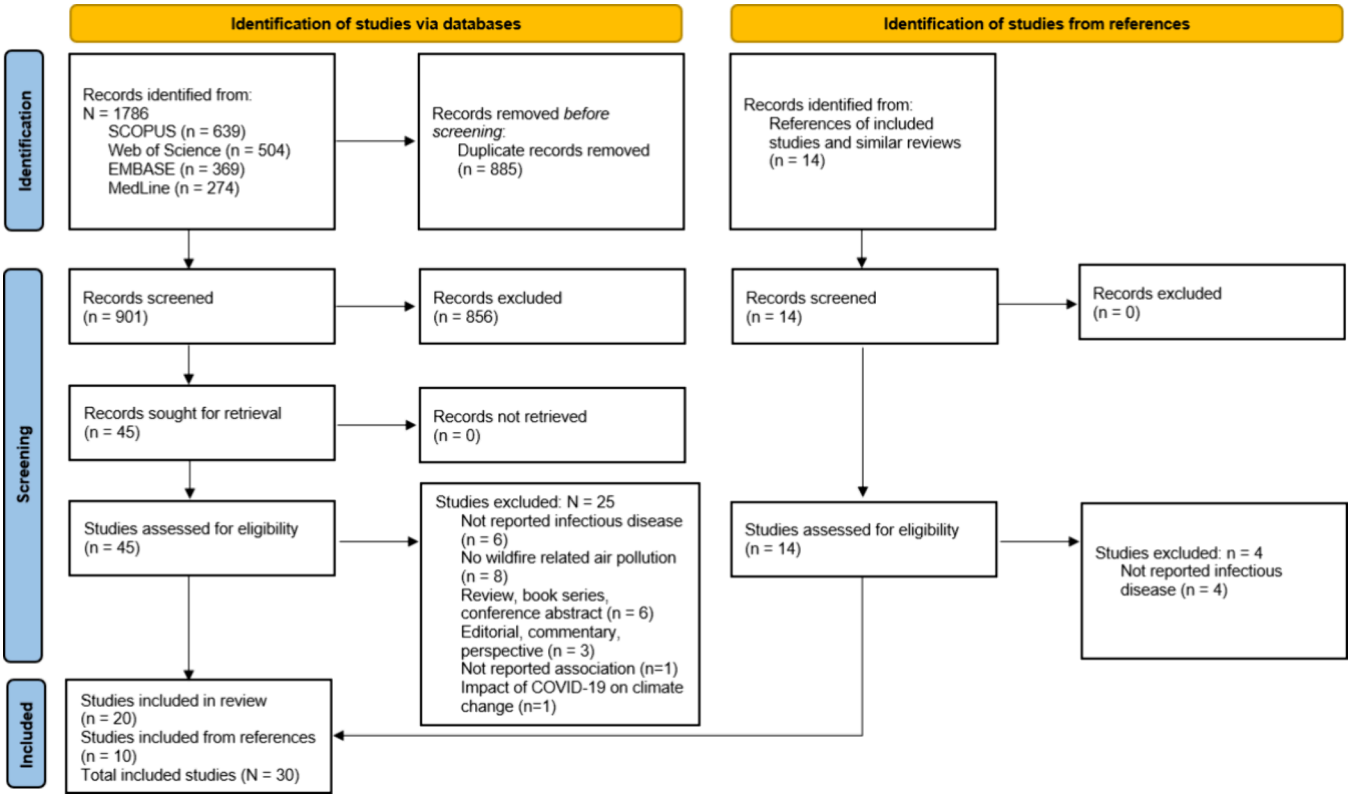

Figure 1. PRISMA flow diagram used in the study selection process.

confounding, attrition/exclusion, detection and selective reporting. The rating for each domain was classified as "definitely low", "probably low", "probably high", "definitely high", and "not reported". Studies were excluded in current systematic review if they were "definitely high" or "probably high" in more than four domains in the OHAT tool.

2.5. Data Analyses

Studies were grouped into short-term (one month and below), medium-term (above one month but less than a year) or long-term exposures (one year and above) according to the length of exposure period to wildfire air pollution. A quantitative analyses (meta-analysis) was conducted only when two or more effect estimates were available for the association between each cause-specific infectious disease and specific wildfire-related air pollution exposure, regardless of the study area and infectious disease outcomes. We grouped studies that reported signle effect estimate formultiple respiratory infections (two or more) and a specific respiratory infectious disease with a single study into one group named respiratory diseases, when performing analyses. In other cases, a descriptive analyses was performed. The quantitative measure of association reported included odds ratios (ORs), RRs, risk ratios and rate ratios; for consistency, risk ratios and rate ratios were treated as RRs. For studies on wildfire $PM_{2.5}$, all effect estimates were mathematically converted to the change in risks per 10-$\mu g/m^3$ increase in the exposure to wildfire $PM_{2.5}$. For wildfire smoke and other wildfire air pollutants, the estimates were taken as reported in their original studies. A unit of 10 $\mu g/m^3$ was considered for the exposure of $PM_{2.5}$, while a unit of 1 $\mu g/m^3$ was used for all other pollutants. The pooled effect estimates and their corresponding 95% CI for smoke and per 10 $\mu g/m^3$ $PM_{2.5}$ were calculated based on data from all eligible studies.

Random-effects meta-analysis models were used to pool the effect estimates. Heterogeneity among studies was evaluated using I^2 statistics, with a value greater than 50% indicating substantial heterogeneity across included studies.[26] When more than three studies were available for each exposure−outcome pair, publication bias was assessed using funnel plots in which RR estimates were plotted against standard error and *p*-value was derived from Egger's test. All analyses were conducted using the 'meta' (version 7.0−0) and 'Metafor' (version 4.6−0) packages and the R software version 4.0.3.[27]

3. RESULTS

3.1. Search Results

The initial search results identified 1,786 titles (Figure 1). Following the removal of 885 duplicate records, the title and abstracts of 901 records were screened against our eligibility criteria. After the exclusion of 856 irrelevant records, 45 articles were identified for full-text screening. Additionally, 14 full-text articles were obtained through the reference list searches. A total of 59 full-text articles underwent full-text screening, resulting in 30 studies being retained for the quantitative and descriptive synthesis after excluding 29 studies that did not meet our inclusion criteria. The main reasons for the exclusion of 29 studies were not reporting infectious diseases (n = 10), not reporting wildfire-related air pollution (n = 8), reviews, book series, conference abstracts (n = 6), editorials, commentaries, perspectives (n = 3), not reporting association nor trend (n = 1), and impact of COVID-19 on climate change (n = 1).

3.2. Quality and Risk of Bias Assessment

Of the studies included, only one cohort study was identified, assessed using NOS, and rated as good quality (Table S3). The rest of the 29 studies were divided into two groups (time-series studies and other observational studies) and assessed using a modified version of the previously validated quality assessment framework developed by Zaza et al.[23] (Tables S4 and S5). According to the quality assessment scores from this tool, 11 studies were rated as high quality; 18 studies were medium

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Table 1. Detailed Study of Characteristics of the Included Studies[a]

Author	Study location	Study design	Study period	Study population	Sample size	Wildfire-related air pollution exposure	Outcome	Confounders	Statistical analysis
Yadav et al., 2003[28]	Brunei Darussalam	Time-series study	7.5 months (April 7 to December 31, 1998)	Outpatient Department records of Civil hospitals of Brunei for Asthma and Acute Respiratory Infections and Influenza	N/A	PM_{10} and CO	Outpatient visits for Acute Respiratory Infections and Influenza	N/A	Technique of canonical correlation (multivariate method of analysis)
Hanigan and Morgan, 2008[30]	Australia	Time-series study	8 months for 10 years (between April 1 and Nov 30 each year from 1996 to 2005)	All persons admitted to the Royal Darwin Hospital for respiratory and cardiovascular conditions	Total admission: 109,478; Respiratory: 4836; Cardiovascular: 3443	PM_{10}	Hospital admissions for Respiratory Diseases (ICD-9:460-519)	Mean daily relative humidity and temperature, weekly influenza (influenza epidemic), school holidays, population estimates of indigenous and non-indigenous Darwin residents, change between ICD editions	Overdispersed Poisson generalized linear models with natural cubic splines
Delfino et al., 2009[31]	United States	Time-series study	2.5 months (October 1 to November 15, 2003)	Individuals who lived in affected counties and were diagnosed with the respiratory and cardiovascular illnesses	Hospital admissions: 40,856	$PM_{2.5}$	Hospital admission for Acute Bronchitis, Influenza and Pneumonia	Relative humidity, temperature, surface pressure gradient, percent of non-Caucasians (race), percent of females (sex), median household income, age distribution, weekend vs weekday, day of the week, county	Generalized estimating equations for Poisson data
Rappold et al., 2011[32]	United States	Time-series study with comparison group	1.5 months (June 1−14 July 2008)	ED visits for selected cardiovascular and respiratory outcomes by adults throughout the eastern portion of the state	Total exposed counties: Respiratory = 4702; Cardiovascular = 6078	Smoke (Aerosol Optical Depth)	ED visits for Acute Upper Respiratory Infection, Acute Bronchitis and Pneumonia	Flooding, humidity, rainfall, average daily temperature	Poisson log−linear regression
Martin et al., 2013[33]	Australia	Case-crossover design	14 years (1994−2007)	Those who reside in statistical local areas of Sydney, Newcastle, and Wollongong metropolitan areas who admitted to hospital during the study period	Total admission: Sydney = 3,141,017; Wollongong = 273,034; Newcastle = 345,736	Smoke (Event vs nonevent days)	Hospital admissions for Acute Bronchitis and Pneumonia	Average temperature and humidity, binary variable for influenza epidemic, public holidays, school holidays	Conditional logistic regression models
Johnston et al., 2014[34]	Australia	Case-crossover design	11 years July 1 1996 to June 30 2007)	Residents of statistical local areas corresponding with the Sydney metropolitan area who were recorded at the ED attendances in Sydney during the study period	Total nontrauma attendances: 4,655,639; Respiratory: 663,333; Cardiovascular: 368,423	Smoke (Event vs nonevent days)	ED visits for Acute Bronchitis and Pneumonia	Daily ambient temperature and dew point, average of the study population of Sydney, influenza epidemic, public holidays, school holidays	Conditional logistic regression models
Alman et al., 2016[35]	United States	Case-crossover design	1 month (5.6.2012 to 6.7.2012)	Patients of all ages living in Colorado with hospitalization and ED visits for cardiorespiratory disease during study period	Respiratory: 6610; Cardiovascular: 3219	$PM_{2.5}$	ED visits and hospital admissions for Pneumonia, Acute Bronchitis and Acute Upper Respiratory Infection	Day-of temperature, day of week, daily 8-h maximum ozone	Conditional logistic regression
Reid et al., 2016[36]	United States	Time-series study	4 months (May 6 to September 15, 2008)	Counts of hospital admission visits and ED visits for 66 zip codes of Northern California	Total counts: 102,311 Daily mean respiratory: Hospitalization = 145.2; ED visit = 752	$PM_{2.5}$	ED visits and hospital admissions for Pneumonia, Acute Upper Respiratory Infection	Holiday, day of week, smoking prevalence, median income, percent of population over 65 years of age, ozone, heat index (temperature and relative humidity)	Poisson generalized estimating equations
Tinling et al., 2016[37]	United States	Time-series study	1.5 months (May 5, 2011 to June 19, 2011)	Same data as Rappold et al. 2011 (ED visits for selected cardiovascular and respiratory outcomes by adults throughout the eastern portion of the state)	Total ED visits: 57,650; Total respiratory: 19,884	$PM_{2.5}$	ED visits for Acute Bronchitis and Pneumonia	Age, gender, temperature, relative humidity, weekends, Memorial Day holiday, socioeconomic status (county population in poverty)	Quasi-Poisson regression model

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Table 1. continued

Author	Study location	Study design	Study period	Study population	Sample size	Wildfire-related air pollution exposure	Outcome	Confounders	Statistical analysis
Yao and Henderson, 2016[38]	Canada	Time-series study	6 months for 8 years (April 1 to 30 September 2003–2010)	Population of 29 local health area in British Columbia in 2006 (daily counts of pharmaceutical dispensation for each LHA retrieved from BC PharmaNet database	Total population in 2006: 2,681,840; Mean daily physician visits for Upper respiratory infection: 62 per 100,000 individuals; mean daily lower respiratory infection: 6 per 100,000 individuals	$PM_{2.5}$	Outpatient visits for Acute Bronchitis and Acute Upper Respiratory Infection	Temperature using a natural cubic spline of daily maximum apparent temperature with 3 df, short- and long-term temporal trends controlled using a factor variable that indicated the year, month, and day of week	Generalized linear Poisson regression model + random effect meta-regression
Gan et al., 2017[39]	United States	Case-crossover design	4 months (July 1–October 31, 2012)	Persons who were admitted to a hospital in Washington State and were recorded in the Washington State Department of Health Comprehensive Hospital Abstract Reporting System (CHARS) for the year 2012	248,647 total admissions, of which 26,835 hospital ED admissions	$PM_{2.5}$	Hospital admissions for Respiratory Diseases (ICD-9:460-519)	Temperature, relative humidity, wind speed, precipitation	Conditional logistic regression models with patient specific strata
Sheldon & Sankaran, 2017[40]	Singapore	Time-series study	6.5 years (Jan 2010–June 2016)	Polyclinic attendances for acute upper respiratory tract infections (ARTIs), acute conjunctivitis (AC), acute diarrhea and chickenpox in Singapore	Maximum (minimum) average daily polyclinic visits for ARTIs: 4241 (1839); AC: 168 (62)	Pollution Standard Index	Outpatient visits for Acute Upper Respiratory Tract Infection	Daily rainfall, mean maximum and minimum temperature and mean and max wind speed	Two-stage least-squares approach
Hutchinson et al., 2018[41]	United States	Case-crossover design	5 months (August 1–December 31, 2007)	Medi-Cal (public health insurance program) beneficiaries who resided in San Diego county	Medi-Cal beneficiaries: 345,257; total of 5,454,360 Medi-Cal claims for San Diego beneficiaries derived from 217,067 residents with at least one claim	$PM_{2.5}$	ED visit, Outpatient visit and hospital admissions for Acute Bronchitis, Acute Upper Respiratory Infection, Influenza and Pneumonia	Temperature and humidity, day of the week	Bidirectional case-crossover analysis
Reid et al., 2019[42]	United States	Time-series study	4.5 months (May 6–September 26, 2008)	Daily counts of hospitalizations and ED visits by residential ZIP code from the California Department of Public Health Environmental Health Investigations Branch for the state of California for respiratory outcomes	All respiratory during wildfire: Hospitalization = 5579, ED visit = 27,934	O_3 and $PM_{2.5}$	ED visits and hospital admissions for Acute Bronchitis, Pneumonia	Daily heat index, temporal trend modeled with a natural cubic spline with 3 df, estimated ZIP code-level smoking prevalence, percent of the ZIP code aged 65 or older, percent of ZIP code aged 5 or younger, percent of nonwhite, ZIP code level median income, day of week and holidays	Poisson generalized estimating equations
Stowell et al., 2019[43]	United States	Case-crossover design	4 months for 4 years (May–August of 2011 to 2014)	Individualized-level health data on daily hospitalizations and ED visits at all public and private hospitals from the Colorado Department of Public Health & Environment	Total records: 490,368	$PM_{2.5}$	ED visits and hospital admissions for Acute Bronchitis and Acute Upper Respiratory Infection	Age, sex, relative humidity, boundary layer height, heat index, wind speed	Conditional logistic regression models
Landguth et al., 2020[44]	United States	Time-series study	10 years (from 2009 to 2018)	Influenza counts in Montana were obtained from the Montana Department of Public Health and Human Service	N/A	$PM_{2.5}$	Influenza records from Department of Public Health	Maximum daily temperature, population in each county, seasonality	Seasonal quasi-Poisson model with generalized estimating equations

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Table 1. continued

Author	Study location	Study design	Study period	Study population	Sample size	Wildfire-related air pollution exposure	Outcome	Confounders	Statistical analysis
Meo et al., 2020[45]	United States	Time-series study	6 months (March 20–September 16, 2020)	Daily COVID-19 cases and deaths recorded in San Francisco, USA during the study period	Mean daily cases: 57.59; cumulative cases: 3939.27; deaths: 0.45; cumulative deaths: 42.83	CO and $PM_{2.5}$	COVID-19 infections and deaths from COVID-19 testing	N/A	Pearson correlation to assess relationship, Poisson regression analysis to predict cases and deaths
Howard et al., 2021[46]	Canada	Retrospective cohort	2.5 months for 4 years (June 15–August 31, 2012–2015)	Individuals from Yellowknife and surrounding communities presenting for care between 2012 and 2015	N/A	$PM_{2.5}$	ED visits and hospital admissions for Pneumonia, Respiratory Diseases (ICD-10: J00-99)	Humidity and temperature, age, sex, ethnicity, day of week	Generalized linear Poisson regression models
Kiser et al., 2021[47]	United States	Time-series study	5 months (May 15–October 20, 2020)	SARS-CoV-2 nucleic acid amplification (NAA) test results and COVID-19 cases in Reno	Patients tested: 35,955; Tested positive: 2881	$PM_{2.5}$	COVID-19 infections and SARS-CoV-2-test results from COVID-19 testing	7-day average of mean temperature, count of the positive cases on the previous day, day of the week, total number of tests administered	Generalized Additive model from the Negative Binomial Distribution
Malig et al., 2021[48]	United States	Time-series study with comparison group	3 months (October 2015, 2016, and 2017) Fire period 9 days in October 2017 Nonfire periods: 18 days (in October 2015, 2016, 2017)	Diagnosis-specific counts of ED visits and hospitalizations for nine San Francisco Bay area counties	N/A; Mean daily ED visits for respiratory: 674.5 for fire period; mean daily hospitalization for respiratory: 84.4 for fire period	$PM_{2.5}$	ED visits and hospital admissions for Pneumonia, Acute Upper Respiratory Infection	Temperature, weekend days, county population, wildfire period	Quasi-Poisson regression model
Meo et al., 2021[49]	United States	Time-series study	6 months (March 2, 2019 to August 15, 2020 (Group A before wildfire) and February 8, 2015 to September, 22, 2020 (Group B during wildfire))	Daily new COVID-19 cases and deaths in 10 different counties in California region during the study period	Total cases: 390; mean cases: 100.62; mean deaths: 2.21	$PM_{2.5}$, CO and O_3	COVID-19 infections and deaths from COVID-19 testing	Temperature, humidity, date, day of the week, weekends	Spearman Rho correlation to assess the relationship, Poisson regression to predict cases and deaths
Zhou et al., 2021[50]	United States	Time-series study	9 months (March 15 to December 16, 2020)	COVID-19 cases and deaths in 92 counties located in three USA states that were affected by 2020 wildfire (Washington, Oregon, California)	Median daily COVID-19 (per 100,000) case rate: 8.5; deaths rate (per 1,000,000): 1.23	$PM_{2.5}$	COVID-19 infections and deaths from COVID-19 testing	The day of the week, nonlinear confounding effect of temperature and humidity, seasonality, county-specific population size, long-term trends, mobility	BH-ZINB-DL model, a distributed lag model
Ademu et al., 2022[51]	United States	Time-series study	11 months (Feb 1 to Dec 31, 2020)	COVID-19 cases in 20 counties affected by the 2020 California Wildfires	N/A, mean daily confirmed COVID-19 cases: 553 (peak), 142 (off-peak)	Air Quality Index, CO, NO_2 and $PM_{2.5}$	COVID-19 infections from COVID-19 testing	Daily mean temperature, mean precipitation, average wind speed, day-fixed effects, county fixed effects, autocorrelation from previous COVID-19 cases, log of the daily number of SARS-CoV-2 tests for every county	Time-series methods: Generalized Additive Models
Cortes-Ramirez et al., 2022[52]	Australia	Ecological design/cross-sectional analyses	7 months (September 2019 to March 2020)	Locally acquired COVID-19 cases	N/A, mean COVID-19 incidence rate: 13.4	PM_{10} and Wildfire burned area	COVID-19 incidence rate from COVID-19 testing	Age, sex, population density, index of relative socioeconomic disadvantage	Mixed effects Bayesian regression model

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Table 1. continued

Author	Study location	Study design	Study period	Study population	Sample size	Wildfire-related air pollution exposure	Outcome	Confounders	Statistical analysis
Mahsin et al., 2022[53]	Canada	Time-series study with comparison group	6 months (from August 1 to September 30 2015 and September 1−30 2014 and 2016 are control years)	Patients who visited outpatient physician visits for respiratory and cardiovascular conditions in the city of Calgary during the study period and recorded in Practitioner Billing Claims database	Total physician visits: 150,411 by 68,701 patients	$PM_{2.5}$	Outpatient visits for Acute Bronchitis, Acute Upper Respiratory Infection	Weekend or public holiday vs weekday, wildfire period, natural splines of CH_4 with two degrees, wind direction, relative humidity	Generalized Additive Quasi-Poisson Regression model with a log-link function
Schwarz et al., 2022[54]	United States	Quasi experimental method/natural experiment	5.5 months (June 1 to November 10, 2020)	COVID-19 cases and deaths in San Francisco Bay Area (SFBA) counties with more than 10 cases in the study period	Total COVID-19 cases: 91,985 deaths: 1255	Smoke (Smoke cover)	COVID-19 case-fatality ratio from COVID-19 testing	Synthetic Control Method (SCM) adjusts for any unmeasured confounding by design	SCM, a quasi-experimental method
Shirangi et al., 2022[55]	Australia	Time-series study	1.5 years (July 1, 2015 to December 31, 2017)	All residents who attended Western Australia ED over the study period	Total ED visits: 1,543,222; Acute Lower Respiratory Tract Infection: 36,859	$PM_{2.5}$	ED visits for Acute Lower Respiratory Tract Infection	Age, sex, socio-economic indexed for areas (SEIFA), dewpoint temperature, humidity, woodfire, holiday, weekend and season	Univariate statistical analysis and multivariate Poisson regressions
Mulliken et al., 2023[29]	United States	Population-based retrospective longitudinal study	3.5 months (between October 1, 2014 and May, 31, 2018)	Hospital administrative data for systemic fungal infection from 22 hospitals in California, USA who were members of Vizient during the study period	Median annual admission per hospital: 1638	Smoke (Smoke plume density)	Hospital admissions for Aspergillosis and Coccidioidomycosis	Monthly differences in hospital admission rates across hospitals, average monthly temperature, average monthly humidity, month of the year, and whether the hospital provided care to cancer patients	Generalized linear mixed-effects models
Naqvi et al., 2023[56]	United States	Time-series study with comparison group	3 months (July 15 to October 16 2020)	COVID-19 cases and deaths in five cities (Fresno, Los Angeles, Sacramento, San Diego, and San Francisco) severely affected by wildfires	N/A	$PM_{2.5}$, CO and NO_2	COVID-19 infections and deaths from COVID-19 testing	N/A	N/A, time-series trend study
Yu & Hsueh, 2023[57]	United States	Time-series study	8 months (April 1, 2020 to November 30, 2020)	COVID-19 cases and deaths reported in California	COVID-19 infections: 431,241; deaths: 7977	$PM_{2.5}$	COVID-19 infections and deaths from COVID-19 testing	Hospital resource availability, available public shelters and housing units, social vulnerability, fire indicator, testing, hospitalization	County-by-day panel fixed effects model

[a]N/A − Not available; ED visit − Emergency Department visit; df − Degree of freedom; PM_{10} - particulate mattter with a diameter 10 micrometers or less).

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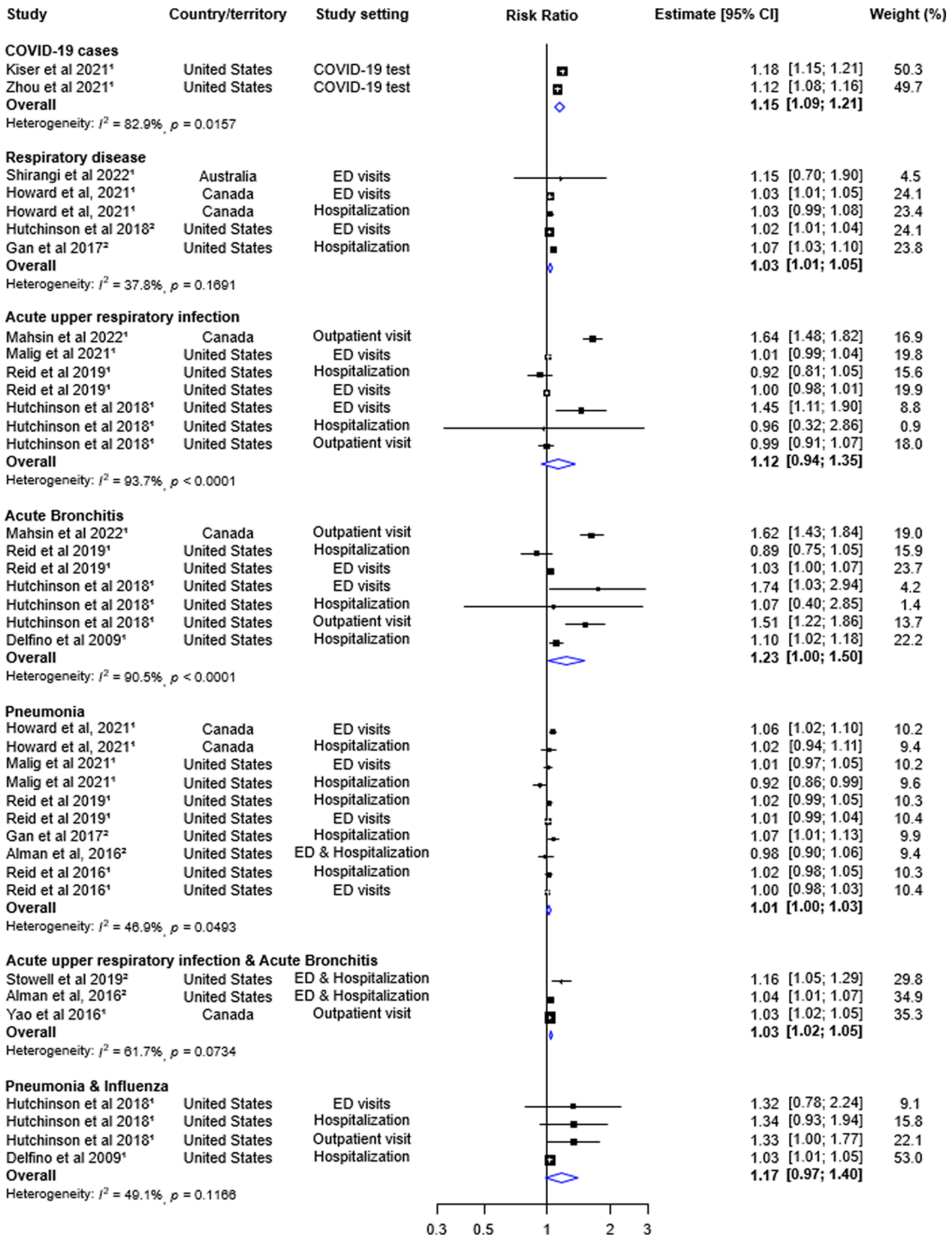

Figure 2. Pooled effect estimates for cause-specific respiratory infectious diseases with 95% CI for an increase of 10 $\mu g/m^3$ wildfire $PM_{2.5}$. [1]Relative risk, [2]Odds ratio.

quality; and one study was classified as low quality with 9 out of 24 points. Only one study scored the highest score of 20 out of 24 among the time-series studies.

Based on the OHAT risk of bias rating tool, one-third of the included studies (9/30 included studies) exhibited bias in at least one of the six domains assessed, and the remaining 21 studies were identified as low risk of bias (Table S6). However, some included studies had limitations related to selection bias, confounding bias, and biases in the assessment of both exposure and outcomes.

3.3. Characteristics of Included Studies

A summary of characteristics of studies included in quantitative and descriptive analyses were shown in Table 1. Most of the studies were conducted in developed countries such as Australia (16.67%, n = 5), USA (66.67%, n = 20), Canada (10%, n = 3) and Singapore (3.33%, n = 1) with only one study originating from a developing country (Brunei Darussalam).

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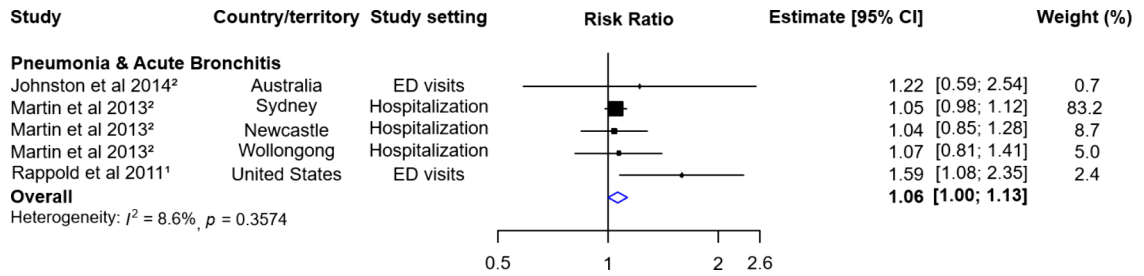

Figure 3. Pooled effect estimates for pneumonia combined with acute bronchitis with 95% CI for short-term exposure to wildfire smoke. [1]Relative risk, [2]Odds ratio.

Included studies estimated the effects for hospital admissions (16.67%, n = 5), ED visits (13.33%, n = 4), outpatient visits (13.33%, n = 4) or a combination thereof (26.67%, n = 8), and 30% of studies used COVID-19 testing results (n = 9).

Most of the studies used either time-series (n = 20) or case-crossover (n = 6) design. Of the 30 included studies, 80% (n = 24) were published during or after 2016, while one was published in 2003[28] and the most recent one in 2023.[29] Only nine studies were conducted for one year or longer, while the remaining studies (n = 21) were conducted for a couple of months around the wildfire events. Statistical methods used by included studies were mainly Poisson regression models (46.67%, n = 14) and conditional logistic regression models (20%, n = 6). Ninety percent of studies (n = 27) considered for potential confounders including socio-demographic and socio-economic factors (age, sex, ethnicity, income, socioeconomic status), environmental factors (temperature, relative humidity, flooding, surface pressure, rainfall, ozone, wind speed), spatiotemporal factors (season, year, public and school holidays, day of the week) and the influenza epidemic.

Included studies assessed a range of wildfire-related air pollution exposures. Out of 30 studies, 16 studies focused primarily on wildfire $PM_{2.5}$, five studies on wildfire smoke, two studies on wildfire PM_{10} (particulate matter with a diameter of 10 μm or less) and only one study on pollution standard index, while six studies investigated a combination of wildfire-related air pollutants such as CO, O_3, NO_2, with $PM_{2.5}$ or PM_{10}.

All the included studies focused on respiratory infectious diseases, except for one that investigated systemic fungal infections such as Aspergillosis and Coccidioidomycosis, which are often acquired through inhalation and can also be considered respiratory infections. Since 2020, there has been a rise in the number of studies focusing on impacts of wildfire air pollution on COVID-19 infections and deaths. Among 29 respiratory infectious disease related studies, 9 studies were about COVID-19 infections and deaths. In our included studies, respiratory infectious diseases encompassed acute bronchitis (ICD-9:466), pneumonia (ICD-9:480−486, ICD-10: J12−J18), acute upper respiratory infections (ICD-9:460−465, ICD-10:J00−J06) and influenza (ICD-9:487). Three studies focused on respiratory diseases (ICD-9:460−519, ICD-10:J00−J99) where no separate effect estimates were reported for specific respiratory infectious diseases. Another three studies did not classify respiratory infectious diseases according to the ICD classification system, instead reported as acute respiratory infections which were subsequently categorized as respiratory diseases for our quantitative analyses.

3.4. Association between Short-Term Exposure to Wildfire-Related Air Pollution and Infectious Diseases

Out of 27 studies on short-term wildfire-related air pollution exposure, 20 studies reported a quantitative effect estimate for the association between wildfire-related air pollution and respiratory infectious diseases. Of them, 15 and 3 studies focused on wildfire $PM_{2.5}$ and smoke, respectively, and were eligible for the meta-analysis.

Figure 2 shows the pooled effect estimates of cause-specific respiratory infectious diseases with 95% CI for an increase of 10 $\mu g/m^3$ wildfire $PM_{2.5}$. A 10 $\mu g/m^3$ increase in wildfire $PM_{2.5}$ was significantly associated with a 15% increase in COVID-19 infection (RR: 1.15; 95% CI: 1.09−1.21) and a 3% increase in respiratory diseases (RR: 1.03; 95% CI: 1.01−1.05) and a combination of acute upper respiratory infections and acute bronchitis (RR: 1.03; 95% CI: 1.02−1.05). Increasing wildfire $PM_{2.5}$ was marginally associated with acute bronchitis (RR: 1.23; 95% CI: 1.00−1.50) and pneumonia (RR: 1.01; 95% CI: 1.00−1.03). While increasing numbers of acute upper respiratory infection, a combination of pneumonia and influenza cases were observed with rising $PM_{2.5}$ concentration; however, the associations were not statistically significant. A substantial degree of heterogeneity was seen in studies investigated following respiratory infectious diseases: COVID-19 cases (I^2 = 83%, p = 0.02), acute upper respiratory infection (I^2 = 94%, p < 0.01), acute bronchitis (I^2 = 90%, p < 0.01), and a combination of acute upper respiratory infection and acute bronchitis (I^2 = 62%, p = 0.07).

A few studies on wildfire $PM_{2.5}$ and COVID-19 infections and deaths with no effect estimates showed similar findings as our meta-analysis in which elevated wildfire $PM_{2.5}$ concentration was positively associated with COVID-19 infections[45,51,57] and also showed mixed association with COVID-19 deaths.[45,57]

Of the studies examining short-term wildfire-related air pollution, three studies provided quantitative effect estimates for wildfire smoke and were included in the meta-analysis. A pooled effect estimate for pneumonia and acute bronchitis with 95% CI for the increase of wildfire smoke was presented in Figure 3. Short-term exposure to wildfire smoke was marginally associated with a 6% increase in pneumonia and acute bronchitis hospitalization and ED visits (RR: 1.06; 95% CI: 1.00−1.13), and no heterogeneity was presented (I^2 = 9%, p = 0.36). One of the excluded studies from meta-analysis showed a 68% increase in ED visits for acute upper respiratory infections with increasing wildfire smoke (RR: 1.68; 95% CI: 0.94−3.00) but not significant.[32]

The studies that were not included in the meta-analysis for insufficient quantities for certain wildfire-related air pollution exposures showed mixed findings. A 10-$\mu g/m^3$ increase in

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wildfire PM_{10} was significantly associated with a 4.8% increase in respiratory diseases (RR: 1.048; 95% CI: 1.01−1.11).[30] In addition, increased COVID-19 infections and deaths were also significantly associated with a rising combination of wildfire-related $PM_{2.5}$, CO, and O_3.[49] However, elevated wildfire-related O_3 was not significantly associated with ED visits and hospitalization for pneumonia, acute bronchitis or acute upper respiratory infection.[42]

Studies that did not report an effect estimate also indicated a positive association or increasing trend in COVID-19 infections and deaths with increasing levels of wildfire CO,[45,51] wildfire smoke,[54] Air Quality Index,[51] and a combination of wildfire $PM_{2.5}$, CO, and NO_2.[56] However, negative associations between COVID-19 infections and wildfire NO_2,[51] as well as COVID-19 deaths and wildfire $PM_{2.5}$,[45] were also evident from recent studies. Another study from Brunei Darussalam showed a positive relationship between acute respiratory infections and influenza and wildfire generated PM_{10} and CO.[28]

3.5. Association between Medium-Term Exposure to Wildfire-Related Air Pollution and Infectious Diseases

Studies investigating the effects of medium-term wildfire-related air pollution on infectious diseases were scarce. We found that three studies examined systemic fungal infection, COVID-19 incidence rate and influenza. The only study on systemic fungal infection in our included studies reported that the number of hospital admissions for coccidioidomycosis increased by 20% (95% CI: 5%−38%) in the month following any wildfire smoke exposure.[29] There was a 32% increase of COVID-19 incidence rate (RR: 1.32; 95% CI: 1.05−1.67) for each 1.8% increase of wildfire burned area five months following the onset of wildfire in Australia.[52] Similarly, wildfire $PM_{2.5}$ was associated with rising influenza cases in USA.[44]

3.6. Publication Bias

The funnel plots and results of Egger's tests (p-values) can be found in Supporting Information (Figures S1−S7). Both funnel plots and Egger's test results showed no publication bias for our included studies.

4. DISCUSSION

4.1. Key Findings

To our knowledge, this systematic review and meta-analysis represents the first quantitative synthesis of the relationship between wildfire air pollution and infectious diseases based on studies obtained through a comprehensive search. Studies have primarily focused on wildfire air pollution and its effect on respiratory infectious diseases in the USA, Canada and Australia, with increased attention on COVID-19 since the 2020 pandemic. Interest in this topic gained significant attention after 2000, becoming particularly prominent over the past decade. We found that short-term exposure to wildfire-related $PM_{2.5}$ shows a strong and statistically significant association with COVID-19 infections (15% increase, RR: 1.15, 95% CI: 1.09−1.21), while other respiratory infections (e.g., acute bronchitis, pneumonia) show weaker or marginally significant associations. High heterogeneity across studies suggests variability in effects. However, short-term exposure to wildfire-related O_3 was not linked to elevated ED visits and hospitalization for pneumonia, acute bronchitis or acute upper respiratory infection. On the other hand, medium-term exposure appears to have a stronger impact on COVID-19 (32% increase, RR: 1.32, 95% CI: 1.05−1.67) and fungal infections (20% increase, 95% CI: 5%−38%), with significant associations observed despite fewer studies. The findings of our systematic review provide a detailed overview of current research and identified key knowledge gaps, providing valuable insights for guiding future research on this topic.

4.2. Potential Pathways/Mechanisms

The significant association between wildfire-related air pollution and respiratory infectious diseases we found in our meta-analysis was similar compared with existing evidence.[8,10,11] This is because fine particles in wildfire smoke are believed to be more toxic than coarse particles,[58] causing lung inflammation[11] and increasing the risk of respiratory infections during wildfire events. In the short term, exposure to wildfire-related $PM_{2.5}$ can trigger acute inflammation and oxidative stress which weakens the body's defense against respiratory infections, leading to a rapid increase in cases.[59] We observed that an increasing short-term wildfire-related $PM_{2.5}$ was significantly associated with a 15% increase in COVID-19 infections which was similar to recent findings.[17,18,60] Exposure to air pollution is believed to compromise the adaptive immune response against viral infections, potentially inducing virus-induced tissue inflammation and damage in the lungs[60,61] that may increase the COVID-19 severity.

For medium-term exposure, persistent inflammation and impaired adaptive immunity with decreased antibody production may contribute to prolonged susceptibility to infections[59] as seen in studies showing increased COVID-19 and fungal infections weeks or months after wildfire events. Furthermore, wildfire smoke can carry live pathogens[14] and may serve as a vector for transmitting infectious disease-causing pathogens over long distances,[4] by potentially spreading disease-causing microbes for longer periods in nonwildfire regions.

For example, COVID-19 transmission through air and $PM_{2.5}$ could extend the virus's travel over greater distances,[4] potentially increasing the spread and severity of COVID-19 beyond what is typically considered for close contact. However, one of the studies included in our systematic review reported a negative association between wildfire $PM_{2.5}$ and COVID-19 deaths, potentially influenced by the use of a face mask, COVID-19 vaccination, and other control measures.[62]

Short-term wildfire O_3 exposure was not associated with respiratory infectious diseases, likely due to its secondary formation process and distinct biological effects compared to direct wildfire pollutants like $PM_{2.5}$.[5] While $PM_{2.5}$ can directly suppress immune function[59] and carry pathogens,[14] O_3 primarily acts as an oxidant, causing airway irritation,[5,6] which may not immediately increase infection risk. In fact, some studies have suggested that low-level O_3 may have protective effects on human health.[63] Additionally, it has been explored for clinical applications.[64] These differences highlight the complex and O_3-specific impacts of wildfire-related air pollution on respiratory infectious diseases.

4.3. Potential Knowledge Gaps and Future Directions

4.3.1. Infectious Diseases. Regarding infectious diseases studied, nearly all of the included studies focused on respiratory infectious diseases, except for one study that examined fungal infection. Extensive research has been conducted on the effects of air pollution and temperature on the risk of COVID-19 morbidity,[18] dengue, infectious diarrhea, hand foot and mouth diseases[17] and tuberculosis.[19] Fur-

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thermore, the majority of research in the climate change and infectious disease literature focused on vector-borne or insect-borne diseases.[15,16] Given this evidence, exploring whether exposure to wildfire-related air pollution associated with these infectious diseases, while considering global warming, high temperatures and climate change as modifying factors, is worthwhile. Therefore, further studies are urgently needed to explore the association between exposure to wildfire-related air pollution and a wide range of infectious diseases such as vector-borne, food-borne, water-borne and air-borne diseases. Furthermore, well-designed studies reporting effect estimates with 95% CI and utilizing the ICD classification system to group infectious diseases may be beneficial for future comparisons across studies.

4.3.2. Exposures and Assessment Method. Most of the included studies used wildfire incidents to assess both the wildfire exposure and duration, but variations in wildfire extents and intensity over time could lead to potential exposure misclassifications. Employing single source exposure data to assess the exposure to wildfire-related air pollution may also result in exposure misclassification, given the spatial variability of air pollutants' levels during wildfires.[11,65] Most studies in our systematic review used exposure data from multiple sources, and some studies blend modeled and monitored wildfire-related air pollution measures which improved the accuracy of exposure assessment.[66] However, studies on long-term exposure to wildfire-related air pollution are limited, and most of the included studies assessed mainly short-term and medium-term exposures to wildfire-related air pollution.

The most widely used air pollutant to quantify the wildfire-related air pollution exposures in included studies was wildfire-related $PM_{2.5}$. However, wildfire-related air pollution that poses public health risks mainly includes particulate matter, CO and O_3.[5−7] Research has explored the health impacts of O_3 pollution[67,68] and identified inconsistent findings of O_3 pollution and influenza,[69,70] yet there remains limited existing evidence assessing the relationship between wildfire-related O_3 pollution and infectious diseases. Only one study included in our systematic review studied the effects of wildfire-related O_3 on hospitalization and ED visits for pneumonia, acute bronchitis and acute upper respiratory infection and did not find any significant association. The increased presence of O_3 during wildfire events,[7,65] along with its link to hospital admission for cardiovascular problems[67] and ED visits for respiratory conditions,[68] underscores the need for comprehensive research into the effects of wildfire-derived O_3 on types of infectious diseases.

4.3.3. Study Location and Duration. Over the 2000−2019 period, concentration of wildfire $PM_{2.5}$ in low-income countries was nearly four times higher than in high-income countries.[7] However, the majority of studies included in our systematic review were conducted in developed countries, with limited data available from developing countries, restricting the generalizability of research findings to developing nations. Furthermore, apart from studies conducted in wildfire hotspots such as Australia, USA and Canada, there is a lack of research from other countries across the world where the concentrations of wildfire-related air pollution are higher.[1,7] Amidst the existing geographical and socioeconomic disparities in population exposure to wildfire-related air pollution,[7] more studies are warranted to understand whether there are any modifying effects of these disparities on wildfire air pollution and infectious disease outcomes. In addition, high-quality study designs investigating longer periods such as one year or multiple years are scarce but useful in understanding the temporal trends and causal pathways of infectious diseases associated with wildfire-related air pollution. Therefore, studying the effects of wildfire-related air pollution on infectious diseases across wider geographical areas and over extended time periods using high-quality study designs will provide a clearer understanding of how spatiotemporally wildfire-related air pollution affects infectious diseases.

4.4. Strengths and Limitations of the Systematic Review

To our knowledge, this is the first systematic review and meta-analysis that comprehensively examined the relationship between wildfire-related air pollution and infectious diseases. The strengths of our systematic review are its focus on this novel topic, the use of a comprehensive and systematic search strategy to ensure a thorough synthesis of existing research, and the identification of critical knowledge gaps, which underscore the need for further investigation in this emerging area of public health. However, there are certain limitations in our review. All the included studies confined to respiratory infectious diseases in three developed countries may limit the generalizability of our findings to respiratory infectious diseases and developed nations. There was also significant heterogeneity across certain studies which could be from diverse methodological approaches including differences in exposure assessment, study design, handling of confounding factors, classification of infectious diseases using ICD-9 and ICD-10 classification systems, and variations in underlying population characteristics. This reported heterogeneity should be considered when interpreting the pooled estimates for those certain diseases. However, subgroup analyses to identify the source of heterogeneity was not possible due to insufficient number of studies on cause-specific respiratory infectious diseases. Additionally, while we acknowledge the importance of exploring subgroup vulnerabilities, such as age-specific effects on the elderly and children, many of the included studies did not assess these factors, limiting our ability to conduct a meaningful assessment of differential impacts across vulnerable populations. We emphasize the need for future research to evaluate the impacts of wildfire-related air pollution on high-risk groups. Furthermore, we included only peer-reviewed studies, potentially overlooking relevant "grey" literature that could have been eligible. Future studies should include a broader range of countries, particularly developing regions, and adopt standardized methodologies to reduce heterogeneity and improve comparability. Additionally, research should focus on the vulnerable population, incorporate 'grey' literature, and expand on cause-specific infectious diseases to enable meaningful subgroup analyses.

4.5. Public Health Implications

The findings from this review have important public health implications, particularly regarding the management and mitigation of health risks associated with wildfire-related air pollution. The clear association between short-term exposure to wildfire smoke and increased respiratory infections, including COVID-19, highlights the need for enhanced air quality monitoring and public health interventions during wildfire events.[6] Additionally, the association between medium-term exposure to pollutants like $PM_{2.5}$ and systemic fungal infections emphasizes the importance of monitoring vulnerable populations, particularly in regions prone to wildfires. Public health strategies should include proactive

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measures such as public advisories, health screenings and improved healthcare infrastructure to address the rise in infections during wildfire seasons.[6] These findings also underscore the need for more research to fill the identified knowledge gaps and better understand the long-term impacts of wildfire air pollution on infectious diseases.

5. CONCLUSION

We systematically summarized studies assessing association between wildfire-related air pollution and infectious diseases. Most of the current evidence primarily examined the impact of wildfire air pollution on respiratory infections in developed countries. We found significant association between increased short-term wildfire-related air pollution exposure and COVID-19 infection and deaths, respiratory diseases and acute upper respiratory infections combined with acute bronchitis infections, whereas short-term exposure to wildfire-related O_3 was not associated with elevated ED visits and hospitalization for pneumonia, acute bronchitis or acute upper respiratory infections. In addition, medium-term exposure to wildfire-related air pollution was associated with increased hospitalizations for coccidioidomycosis fungal infection and influenza cases. Given the increasing frequency and severity of wildfires in the future and ability of wildfire smoke to carry living pathogens and travel long distances, more high-quality population based scientific evidence is a timely requirement to determine the impact of wildfire-related air pollution on infectious diseases and to fill the current knowledge gap in this field.

■ ASSOCIATED CONTENT

Supporting Information

The Supporting Information is available free of charge at https://pubs.acs.org/doi/10.1021/acsenvironau.4c00087.

PRISMA checklist, detailed search strategy, quality and risk of bias assessments, funnel plots, and Egger's test results (PDF)

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Notes

The authors declare no competing financial interest.

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