

Wildfire smoke exposures and adult health outcomes

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Abstract

Health outcomes attributable to wildfire smoke pollution exposure are an increasingly important global health issue especially as wildfires are increasing in frequency and intensity with climate change. In this chapter, we present an up-to-date overview of the literature regarding the health consequences of wildfire smoke pollution exposure experienced by adults, identify research gaps, and propose possible areas for future epidemiological studies. We also discuss existing interventions to reduce the negative health outcomes associated with wildfire smoke pollution exposure.

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10 **Summary**

- 11 • Health outcomes attributable to wildfire smoke pollution exposure are an increasingly
12 important global health issue especially as wildfires are increasing in frequency and intensity
13 with climate change.
- 14 • In this chapter, we present an up-to-date overview of the literature regarding the health
15 consequences of wildfire smoke pollution exposure experienced by adults, identify research
16 gaps, and propose possible areas for future epidemiological studies.
- 17 • We also discuss existing interventions to reduce the negative health outcomes associated with
18 wildfire smoke pollution exposure.

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22 **1 Global Background and Significance of the Problem**

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24 *1.1 Background*

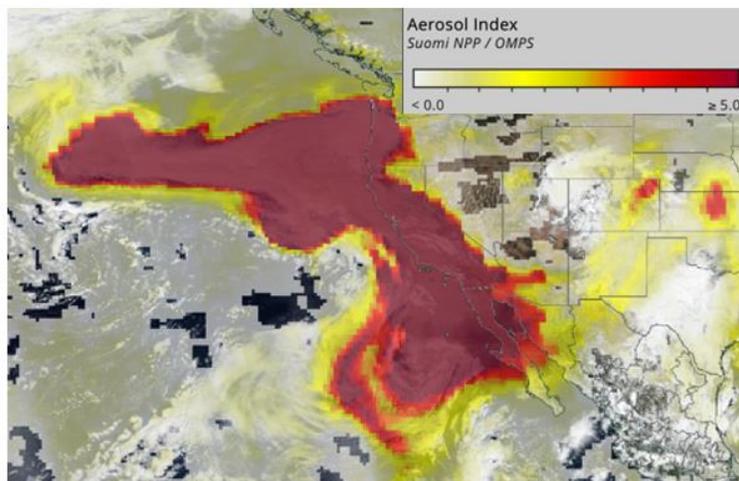
25 Landscape fires can have devastating impacts on human health through contributions to
26 surface air pollution. Fires contribute to enhanced surface concentrations of fine particulate
27 matter (PM_{2.5}; particles < 2.5 microns in diameter) and trace gases such as ozone (O₃), carbon
28 monoxide (CO), and other pollutants. Severe fire events in Australia, the western U.S.,
29 Indonesia, and the Amazon that recently captured the world’s attention have also exposed broad
30 regional populations to dangerous levels of fire-contributed air pollution, hereafter referred to as
31 smoke pollution (Figure 1). In some regions of the world, increases in smoke pollution have
32 negated other air quality improvements over past decades¹.

33 Understanding and documenting the health outcomes associated with smoke pollution
34 exposure is an important and growing public health issue. First, climate change is increasing the
35 contribution of wildfires to smoke pollution in many regions. In the same vein, while wildfires
36 are traditionally considered as acute events, their staggering increase in prevalence and intensity
37 is gradually constituting a sub-chronic environmental exposure, albeit with limited
38 epidemiological evidence. Second, documenting which communities are particularly exposed
39 and/or impacted and which underlying health conditions (e.g. diabetes, cardiovascular diseases)
40 drive a higher susceptibility to smoke pollution is crucial to inform prevention efforts. Third,
41 although evidence for smoke pollution and health outcomes has grown in the past two decades,
42 this primarily comes from high income countries. Many other regions are exposed to smoke
43 pollution, constituting a critical need for future research.

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46 **Figure 1.** *Aerosol Index from September 10th, 2020 showing the presence of absorbing particles*
47 *in the atmosphere across the western U.S. during a wildfire event. Observations from Suomi*
48 *Ozone Mapping and Profiler Suite (OMPS)/National Polar orbiting Partnership (NPP) (OMPS-*
49 *NPP); image courtesy of NASA Worldview.*



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53 *1.2 Smoke pollution health burdens around the world*

54 Several studies have estimated global or regional health burdens to smoke PM_{2.5}
55 exposure. This provides useful information across several dimensions: (1) the relative contribution

56 of smoke to the health burden of PM_{2.5} compared to other sources of ambient pollution, (2) geographic
57 and temporal variability in the health burden of smoke PM_{2.5} exposure, and (3) sources of fire
58 emissions that can inform intervention strategies.

59 Global exposure to smoke PM_{2.5} from landscape fires is responsible for an estimated
60 340,000-680,000 deaths per year, amounting to between 8 and 21% of the total outdoor air
61 pollution mortality burden (i.e. the total number of premature deaths directly attributable to this
62 exposure)^{2,3}. More than 44 million people around the world are exposed to unhealthy annual
63 average PM_{2.5} smoke pollution (> 55 µg/m³)³. However, there is significant spatial variability in
64 smoke pollution sources that contribute to landscape fires (wildfires, deforestation and forest
65 degradation fires, savanna fires, agricultural fires, etc.) and the magnitude of public health
66 burden.

67 In many tropical countries, fires associated with land use and drought conditions
68 contribute to high levels of smoke pollution exposure. Johnston et al.² and Roberts and Wooster
69³ highlighted sub-Saharan Africa and Southeast Asia as global hotspots in driving the attributable
70 mortality burden of smoke PM_{2.5} exposure. Recent estimates suggest nearly 10% of premature
71 deaths due to PM_{2.5} exposure in Brazil were linked to smoke pollution⁴. Preventing vegetation
72 fires in the Amazon Basin could avert approximately 17,000 premature deaths due to smoke
73 PM_{2.5} exposure⁵. Another tropical fire hotspot is Indonesia, where severe fires in 2015 were
74 linked to an estimated 44,000-100,000 premature deaths across Equatorial Asia^{6,7} and the
75 exposure of nearly 70 million people to unhealthy smoke pollution levels⁸.

76 Landscape fires also contribute to local and regional pollution in non-equatorial regions.
77 Vegetation fires were linked to short-term PM_{2.5} increases in southern and eastern Europe and
78 low-to-moderate increases in daily PM_{2.5} across the continent⁹. Kollanus et al. estimated that
79 1,483 and 1,080 premature deaths across 27 countries in Europe in 2005 and 2008, respectively,
80 were attributable to smoke PM_{2.5}. Across the U.S., fires contribute to approximately 11% of
81 PM_{2.5} and 1% of O₃ on average but play a more important role in western states¹⁰ and during
82 extreme weather events^{10,11}. For instance, fires can contribute up to 50% of PM_{2.5} in some parts
83 of the western U.S.¹². In this region, nearly 50 million people over 2004-2009 were exposed to a
84 ‘smokewave’ event (more than two days with high smoke PM_{2.5}), with corresponding increases
85 in respiratory hospital admissions¹³. A recent study estimated the number of asthma hospital
86 admissions, emergency department visits, and premature deaths attributable to acute smoke
87 PM_{2.5} exposure across the U.S. using concentration-response functions (CRFs refer to the
88 estimated dose-response between levels of PM_{2.5} and the risk of observing a given health
89 outcome of interest) specific to smoke PM_{2.5} exposure as well as gas-phase hazardous air
90 pollutants (HAPs)¹⁴. They estimated that 216,000 deaths were attributable to wildfire smoke and
91 that most of the burden took place outside the western U.S. as smoke typically travelled across
92 the continent impacting a very large population.

93 Agricultural fires also contribute to consistent seasonal pollution enhancements in many
94 parts of the world. For example, agricultural waste burning in Central and West Africa is the
95 dominant driver of smoke pollution across the continent, linked to 43,000 premature deaths per
96 year¹⁵. Another important example is in India, where crop residue burning contributes to
97 seasonal extreme pollution above World Health Organization guidelines in rural areas and urban
98 centers^{16,17}. In many regions, additional research is required to separate agricultural fire
99 contributions from other sources of pollution to quantify the health burden attributable to
100 agricultural fires. However, it is worth mentioning that most studies described above applied
101 CRFs developed for all-source PM_{2.5} or (more rarely) for smoke PM_{2.5} specifically.

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1.3 Scope of the chapter

The aim of this chapter is to provide a current overview of the health consequences of smoke pollution exposure experienced by adults, excluding occupational settings (e.g. among firefighters) ¹⁸⁻²¹. The perinatal and child health burden is discussed in the following chapter. We primarily document the health impacts associated with PM_{2.5} smoke pollution exposure, but also briefly address other potentially synergistic consequences associated with the trauma of fire events, such as the emergence of mental health stressors. While PM_{2.5} is the most investigated smoke constituent, it is important to note that other harmful compounds of smoke that impact human health exist such as ozone (O₃), volatile organic compounds (VOCs), carbon monoxide, lead, and other heavy metals and toxins that can be generated by burning biomass and flame contact with built structures. However, such pollutants may primarily impact populations directly impacted by the fire (not only through smoke)¹⁴.

We first present a summary of the epidemiological literature on smoke pollution and adult health outcomes by synthesizing several recent reviews and additional studies. We consider the environmental justice implications of this phenomenon and the need to address differential susceptibility and exposure to biomass burning smoke pollution. We then discuss opportunities to improve our understanding of the adult health burden of smoke exposure more holistically. This includes how smoke pollution exposure estimates are matched to health data through different study designs, settings in which evidence is still lacking, and additional areas of future research, such as repeated smoke exposures and compounded impacts. We then present an overview of existing interventions to reduce the negative health outcomes associated with smoke pollution exposure. We conclude with a summary of research gaps and future directions.

2 Overview of Epidemiologic Evidence on Adult Health Outcomes

2.1 Introduction

In the past few years, several literature reviews have been conducted with regards to the health impacts associated with landscape fire smoke pollution exposure ²²⁻²⁹. PM_{2.5} is one of the primary constituents of smoke pollution and is the focus of this section. Ambient PM_{2.5} concentrations are monitored and regulated, as such particles are small enough to penetrate the respiratory system, interact with the circulation system, and can further impact any organ in the body. Furthermore, PM_{2.5} also impacts human health through systemic inflammation and activation of the autonomic nervous system³⁰. Evidence regarding the health effects of all-source PM_{2.5} is vast, and several reviews have been published in the past decades ³¹⁻³⁴, including both acute (e.g. asthma exacerbation, myocardial infarction, etc.) and chronic (atherosclerosis, dementia, lung cancer, etc.) effects. Smoke pollution concentrations are mostly considered as acute exposures in the epidemiological literature but, as we discuss below, some long-term consequences may exist. The repeated nature of such events in the context of climate change makes such exposure more frequent and considering such exposures as sub-chronic in certain regions of the globe may be warranted in future studies.

The mechanisms through which PM_{2.5} can impact human health (such as oxidative stress, alteration of the pulmonary immune system, and chronic inflammation) may differ according to particle composition (for more details, see Chapter 11). While it may seem reasonable to initially assume that smoke pollution may have similar toxicological mechanisms and impacts on human health compared to all-source PM_{2.5}, recent research justifies studying smoke as a separate

148 exposure for several reasons. First, PM_{2.5} concentrations during an extreme biomass burning
149 event can be one order of magnitude larger or more when compared to typical exposure levels.
150 This implies that epidemiological evidence from other sources of fine particles regarding the
151 dose-response relationship, the types of symptoms, or which subgroups of the populations are
152 susceptible may not extrapolate to such exposures. Second, recent toxicological evidence
153 suggests that smoke PM_{2.5} may be more toxic than equal doses of PM_{2.5} from other sources due
154 to particle composition³⁵. Current air pollution guidelines or regulations do not distinguish by
155 emissions source or chemical composition for PM_{2.5}. With these considerations, focusing on
156 studies with specific smoke pollution exposures has led to dozens of epidemiological studies in
157 the past two decades that we summarize below. We describe the types of health outcomes that
158 have been investigated and the state of evidence is regarding these outcomes. In Section 3, we
159 will discuss health outcomes such as mental health for which evidence is still sparse.

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161 *2.2 Mortality*

162 The evidence related to the impact of smoke pollution on acute premature mortality is
163 relatively strong. Many studies have consistently found an increase in daily mortality during a
164 wildfire event or in subsequent days³⁶. However, most of these studies focused on all-cause
165 mortality or mortality for respiratory or CVD endpoints; studies about other cause-specific
166 mortality outcomes are still lacking^{22,23}. In a recently published global analysis, Chen et al.³⁷
167 found an annual average of 33,510 all-cause deaths to be attributable to smoke PM_{2.5} pollution
168 exposure using data from 749 cities in 43 countries.

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170 *2.3 Morbidity*

171 *2.3.1 Respiratory diseases*

172 Respiratory health outcomes have received the most attention in the published
173 epidemiological literature. Various respiratory morbidity outcomes have been studied, including
174 lung function, respiratory medication usage^{38 39}, physician visits, and emergency departments
175 (ED) visits or hospital admissions for respiratory problems²⁵. When considering ED or physician
176 visits for various respiratory outcomes, published studies strongly suggest a detrimental effect of
177 smoke pollution. Among the specific respiratory outcomes, asthma has been extensively studied.
178 A recent systematic review²⁵ focusing on asthma-related outcomes found consistent evidence for
179 this outcome.

180 Fewer studies have examined changes in lung function²⁵. Amid mixed results, most
181 studies were not able to identify the effect of smoke pollution. For medication usage, studies
182 focused on various endpoints, such as medication use, initiation of oral steroid use, or medication
183 for obstructive lung disease also have inconsistent results²². Finally, there is increasing evidence
184 that wildfire smoke also exacerbates Chronic Obstructive Pulmonary Disease (COPD)⁴⁰. More
185 recent studies also investigated the role of smoke pollution on exacerbating respiratory infections
186 or disease severity such as for seasonal influenza⁴¹; such connections between respiratory
187 infectious diseases and wildfire are particularly relevant in the context of the COVID-19
188 pandemic^{42,43}.

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190 *2.3.2 Cardiovascular diseases*

191 Less epidemiological evidence exists for cardiovascular outcomes than for respiratory
192 outcomes. Some studies have considered smoke pollution exposure and cardiovascular diseases
193 (CVD) outcomes such as hypertension⁴⁴. Most CVD studies assessed hospital admissions or ED

194 visits for CVD causes²². Fewer studies investigate specific CVD endpoints, like congestive heart
195 failure⁴⁵, ischemic heart disease⁴⁶, cardiac arrest⁴⁷ or myocardial infarction⁴⁸. However, the
196 results of these studies focusing on CVD outcomes are mixed, with some studies identifying an
197 increasing risk and other studies not detecting any effect.

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199 *2.4 Vulnerable populations*

200 While most studies conducted to date focused on the health impacts on the entire adult
201 population, several have investigated whether certain population subgroups are more susceptible
202 to the health impacts associated with a specific landscape fire event or smoke pollution more
203 generally^{49,50}. Such work investigating effect modification by various socio-demographic
204 characteristics is motivated by the large evidence on differential susceptibility for fine particles
205 in general (i.e. from other sources of emission)⁵¹. Indeed, the environmental justice literature has
206 found that socioeconomic and racial and ethnic minorities suffer from a disproportionate burden
207 of air pollution exposure in general, and PM_{2.5} in particular⁵².

208 However, studies assessing the extent to which certain socio-demographic characteristics
209 modify the smoke pollution-health risk remain limited. Most studies investigating such
210 differential susceptibility questions conducted stratified analyses or included an interaction term
211 between smoke pollution exposure and the socio-demographic variable of interest⁵³. Among
212 these studies, most focused on age as a susceptibility factor^{48,54,55}. Some studies have shown that
213 the risk for most health outcomes was higher among older populations (with various cutoffs
214 across the studies such as > 65 or > 75 years old), but other studies found the opposite pattern or
215 no evidence of such effect modification by age²². Several studies assessed potential gender
216 heterogeneity, but the results are mixed²².

217 Differential susceptibility across socioeconomic and racial/ethnic groups, including
218 individual race or ethnicity⁵⁰, neighborhood SES⁵⁷, indigenous status⁵⁸ or proxies such as
219 district-specific food consumption⁵⁹ have also been considered. However, results from these
220 studies were mixed, with some studies finding that low SES groups were more susceptible to
221 wildfire and other studies found no differences among groups. Finally, other vulnerability factors
222 included pre-existing health conditions (using different proxies such as number of physician
223 visits in the previous year⁵⁷) but available evidence is inconclusive to date.

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225 **3. Considerations for future epidemiological studies**

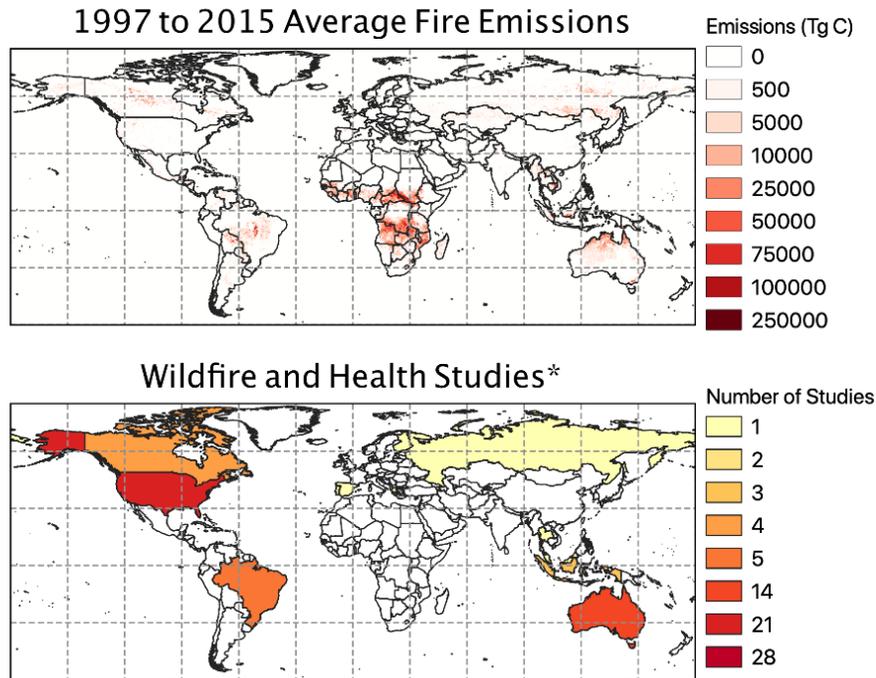
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227 *3.1 Geographic disparities in health studies*

228 More studies are needed in geographically underrepresented areas where wildfire smoke
229 pollution is common and/or projected to increase in the future, especially where the public health
230 infrastructure is more vulnerable⁶⁰. This emphasis should be placed on developing exposure and
231 health operational capacity in parts of the world with high levels of smoke pollution, particularly
232 in sub-Saharan Africa, given the majority of existing studies examined these impacts in North
233 America or Australia^{61,62}. The geographical distribution of existing epidemiological studies
234 highlighted an important gap, which is not unique to smoke pollution⁶³. In Figure 2 below, we
235 illustrate the discrepancies between where studies have been conducted thus far and where most
236 wildfire emissions take place. Epidemiological evidence is lacking in several regions where fires
237 are an important source of emissions, especially in Lower Middle Income Countries (LMICs).

238

239 **Figure 2.** Maps comparing the number of all-source smoke pollution-health studies conducted in
 240 each country with the average fire emissions from 1997 to 2015 from the Global Fire Emissions
 241 Database ^{64,65}.



* The included studies are extracted from 4 recent systematic reviews ²²⁻²⁵

3.2 Exposure estimates for epidemiological studies

Fire contributions to smoke pollution are estimated with various approaches, including atmospheric modeling, satellite-based techniques, ground station data, or blended methods that merge multiple information sources. Here, we briefly review the primary smoke exposure methods through the lens of providing recommendations for use in epidemiological studies. For an in-depth discussion of each of these methods, we refer the reader to Chapters 6-9.

The first category of exposure assessment is atmospheric models, which can be applied at global to local scales. Lelieveld et al. ⁶⁶ and Johnston et al. ² used global atmospheric models to quantify the smoke pollution health burden from multiple fires around the world compared to non-fire pollution sources. At smaller scales, atmospheric dispersion models can be used to track smoke pollution from individual fire events ⁶⁷. Second, satellite observations can monitor pollution during fire events. Satellite-based products include the National Environmental Satellite, Data, and Information Service (NESDIS) Hazard Mapping System (HMS) smoke plume data in North America connects observed smoke plumes to active fires ^{12,68,69}. Aerosol Optical Depth (AOD) from the Moderate Resolution Imaging Spectroradiometer (MODIS) instrument on the Terra and Aqua satellites and the Tropospheric Monitoring Instrument (TROPOMI) have been used to map atmospheric aerosol loading and infer surface PM_{2.5} concentrations during fire events ^{70,71}. Third, ground station observations have been used as an input into blended models to replicate the spatial and temporal variability of smoke pollution. Low-cost sensor networks also show promise for informing the statistical relationship between

265 satellite column aerosol optical depth and surface-level PM_{2.5} during wildfire events due to their
266 dense spatial coverage⁷².

267 Each of these exposure methods have their own strengths and weaknesses to consider for
268 use in epidemiological studies. The first consideration is the spatial scale of exposure datasets.
269 With coarser models or sparse ground station data, the ability to resolve peak smoke pollution
270 concentrations may be reduced, which could result in an underestimate of health outcomes. An
271 additional consideration is the assignment of a single exposure variable to an entire population,
272 despite significant individual-level differences in exposure, such as across zip codes⁷³, and
273 whether an individual spends the majority of their time indoors or outdoors. Second, when
274 possible, we recommend that epidemiological studies use multiple exposure estimates to test the
275 sensitivity of the studies to exposure methods. Blended models consider multiple sources of
276 information to represent smoke PM_{2.5} concentrations. For example, ground station monitors
277 provide surface-level estimates of PM_{2.5} at specific locations and/or time points. Satellite-based
278 observations can be used to fill in some of the gaps in this spatial or temporal coverage. Cleland
279 et al.⁷⁴ recently compared the smoke pollution health burden using multiple exposure estimates
280 (ground monitor, modeled, and blended). The authors found that the choice of exposure dataset
281 drove uncertainty in the resulting health burden estimate. In a review of 28 studies around the
282 world that estimated PM associated with open burning, Johnson et al.⁶² found that blended
283 approaches tend to have the best results by at least partially compensating for limitations
284 associated with each individual approach. Lassman et al.⁷⁵ also found more accurate wildfire
285 PM_{2.5} predictions from monitors relative to satellite AOD or atmospheric modeling simulations,
286 but that blended techniques were more accurate if ground monitor density was low. Exploring
287 multiple exposure datasets may not always be possible due to data or computational limitations.
288 For example, in regions of the world that lack dense ground station networks, modeling or
289 satellite studies are particularly useful¹⁵. Finally, recognizing the implications of exposure
290 method for issuing public health guidance is critical. Fadadu et al.⁷⁶, for example, found
291 substantial variability with the magnitude and timing of peak smoke pollution derived from HMS
292 satellite-derived smoke polygons of low, medium, and high intensity and ground station
293 monitors.

294

295 *3.3 Epidemiological study designs*

296 It is first important to distinguish two approaches to evaluate the health impacts of smoke
297 pollution, including: i) single events and ii) repeated effects of long-term smoke pollution
298 exposure over a long-time span (e.g. multiple years).

299 First, several studies focused on an individual or a handful of major fire events and then
300 evaluated whether health outcomes changes were observed in affected areas (with or without
301 control groups). Examples of such events include the October 2007 Southern California
302 wildfires⁷⁷, summer Russian wildfires in 2010⁷⁸, or Indonesia's forest fires of 1997⁵⁹. In such
303 settings, authors relied on various study designs including case crossover designs⁷⁷, interrupted
304 time series designs⁷⁹ or panel analyses⁸⁰. Such designs capitalize on the specific location and
305 timing of the event of interest and formulate an identification strategy to compare observed
306 outcome in the exposed group to a substitute for the counterfactual population (that was not
307 exposed to the smoke pollution). Quasi-experimental designs, such as difference-in-differences,
308 can also be employed for such research questions but remain underused for such type of events.
309 Yet, they are a powerful alternative strategy to address various confounders that may or may not
310 be measured while checking identification assumptions, such as parallel trends⁸¹. In addition,

311 simpler approaches have been implemented where excess events were estimated by comparing
312 observed outcomes during the event of interest to outcomes rates on a given calendar dates from
313 previous years⁷⁸. This technique has been frequently used in the context of extreme weather
314 events, like extreme heat, hurricanes to estimate excess mortality^{82,83}. However, such approaches
315 do not typically control for any time-varying confounders, such as temperature or other sources
316 of air pollution, which may lead to biased estimates of exposure.

317 Second, other studies focus instead on estimating the overall impact of smoke pollution
318 by considering multiple years and focusing on various spatial scales (from single cities to an
319 entire country). In this setting, studies rely on various techniques to estimate exposure to smoke
320 pollution, such as atmospheric models or statistical techniques (for more details, see previous
321 chapters). Accordingly, various study designs have employed, including ecological time series
322 models or case crossover designs^{84,85} and individual designs based on existing cohorts, nested
323 case-control designs or ad hoc surveys⁸⁶.

324 Finally, several studies^{73,87} have investigated the spatial variability in the health impacts
325 associated with wildfires events and found important heterogeneity of the geographical
326 distribution of the impacts. Such studies remain rare as compared to studies that aggregate the
327 estimates spatially but can provide estimates that can be particularly useful to identify vulnerable
328 communities.

329

330 *3.4 Understudied health outcomes*

331 Certain health outcomes have been understudied. These include mental health outcomes
332 in the adult population, such as psychological distress, solastalgia (i.e. the distress caused by
333 environmental change)⁸⁸, changing psychological outcomes⁸⁹ or mental and emotional well-
334 being⁹⁰. Investigating the short- and long-term impacts of landscape fires on mental health is
335 particularly important to design interventions following such events and improve the resilience
336 of affected communities. In a random digit dial survey of an area affected by multiple wildfires,
337 Felix & Afifi⁹¹ found that those who were exposed to wildfire and were evacuated had poorer
338 measured mental health and greater total fire stress than those who were not evacuated; relative
339 to men, women had poorer mental health and greater total fire stress. More evidence regarding
340 these links is currently needed and future studies capitalizing on self-reported mental health
341 symptoms or medical claims are critical to the development of this field. Other issues such as
342 diabetic⁹² or ophthalmologic⁹³ outcomes, as well as injuries⁹⁴ were investigated by few studies
343 and more evidence is definitively warranted. Furthermore, given the emerging literature linking
344 exposure to PM_{2.5} and incident diabetes⁹⁵ and dementia⁹⁶, there is a need to further investigate
345 the impact of smoke pollution and these outcomes.

346 It is also particularly important to better understand which pre-existing medical
347 conditions constitute susceptibility factors for smoke pollution exposure. Apart from respiratory,
348 CVD, or other chronic conditions, such as diabetes, it is necessary to investigate the extent to
349 which individuals with dementia, Alzheimer's Disease Related Dementias (ADRD) or cancer
350 survivors, for example, are more susceptible to poor health outcomes as compared with the
351 general population. Documenting such pre-existing susceptibility factors will inform existing
352 preventive policies such as early warning systems by identifying which priority populations for
353 interventions. Other plausible susceptibility factors have also received little attention to date and
354 future epidemiological studies are critically needed. Such factors include metrics of health care
355 access, background exposure to other sources of pollution, occupation (e.g. outdoor workers),
356 and populations with physical disabilities.

357

358 *3.5 Fires and the built environment*

359 Landscape fires have the capacity to not only burn vegetation but the built environments
360 in which people live, work and function⁹⁷, as well. As a variety of substances are used in the
361 construction and maintenance of these structures, these materials have varying capacity as fuel
362 and have differing toxic potential when burned. Carratt et al.'s review⁹⁸ noted that there was a
363 spatial overlap of burned area and the prior application of pesticides in California—fire
364 combatting chemical also can be found in these locations. Epidemiological data regarding the
365 health effects of these ignited, potentially-combined chemical exposures is lacking. Studies that
366 address the acute and chronic health outcomes after landscape fire events generally do not
367 address the chemical species of PM_{2.5}. More developed is the literature addressing the protection
368 of the built environment from biomass burning events Penman et al.⁹⁹ used a Bayesian Network
369 model to analyze the strategic use of “fuel breaks”, among other factors, in San Diego County,
370 California. Found to be an effective strategy, the use of this model determined that high density
371 communities, which tend to be at low elevations, were more susceptible to burning than were
372 those at higher elevations, which tended to be less densely populated. Weather, too, contributed
373 substantially to the wildfire's size and ability to travel and affect communities; the treatment of
374 potential fuel had a minimal effect on the fire's ability to spread and endanger property. Housing
375 density in wildfire vulnerable areas in the United States increased 1350% between 1940 and
376 2010¹⁰⁰, increasing the likelihood of the built environment's involvement in wildfire events.
377 Wildfire adaptation has been investigated at the individual¹⁰¹ and community¹⁰² level, both of
378 which address the necessity of managing vulnerability at the wildland-urban interface.

379

380 *3.6 Future research needs*

381 Apart from knowledge gaps in relation to health outcomes and susceptibility factors,
382 there are multiple avenues for future research. First, while most studies focused on PM_{2.5} as the
383 main component of biomass burning smoke exposure, other pollutants, such as O₃ or polycyclic
384 aromatic hydrocarbons (PAHs) are generated in fire plumes. Recent studies have shown that
385 wildfires generate increases in tropospheric O₃ levels through processes distinct from PM_{2.5}
386 ^{103,104}. In future studies, it will be particularly important to understand how smoke pollutants
387 other than PM_{2.5} impact population health and study potential synergies among these pollutants.

388 Another important area of research relates to whether PM_{2.5} smoke pollution affects
389 health outcomes differently from PM_{2.5} from other sources. While this pattern has been
390 suggested by toxicological studies where differences in the composition led to higher effects of
391 smoke PM_{2.5} compared to ambient sources ^{35,105-107}, evidence at the population level is lacking.
392 To the best of our knowledge, only two studies ^{108,109} focusing on asthma addressed this research
393 question. Furthermore, in a recent study, it has also been shown that smoke PM_{2.5} can cause a
394 greater impact on respiratory health than PM_{2.5} from other sources ¹¹⁰. While further studies are
395 needed to confirm these emerging findings, such patterns point to the need for air quality policies
396 to consider the variability in PM_{2.5} impacts on human health according to the sources of
397 emissions.

398 Another area of research that deserves more attention is related to improvement in the
399 understanding the long-term impacts of smoke pollution on various outcomes (besides mental
400 health as described above). Indeed, while wildfire has been considered traditionally as an acute
401 environmental exposure, such instances are rapidly evolving in the context of climate change and
402 variability ^{12,111}. As the length of wildfire seasons increases¹¹², the duration of exposure to

403 extreme smoke pollution and corresponding health outcomes will likely increase. For example,
404 in the western U.S., future smoke PM_{2.5} concentrations under multiple climate change scenarios
405 suggests an increasing threat to public health, particularly for many vulnerable subpopulations
406 ^{111,113,114}. This motivates the need to better understand individual actions to reduce exposure as
407 well as larger scale interventions to reduce wildfire emissions in order to reduce negative human
408 health outcomes (see Section 4 of this chapter) ¹¹⁵.

409 It is also important to better understand the compounded impacts of smoke pollution with
410 other contemporaneous risks. For example, smoke pollution and extreme heat events may co-
411 occur as recently illustrated by Australian fires and the western U.S. in 2020. Smoke pollution
412 and extreme heat share similar mechanisms through which they impact human health and several
413 studies have demonstrated the synergistic effects of air pollution (not specific to smoke) and
414 extreme heat ¹¹⁶. In addition, evidence about heat-related vulnerability identified similar
415 population subgroups as for smoke pollution. Characterizing joint exposures to extreme heat and
416 smoke pollution, as well as associated impacts, constitutes an important area for future research,
417 especially while both events are expected to increase in intensity and frequency in our changing
418 climate.

419 Finally, an important (and timely) area of investigation is related to the interactions
420 between smoke pollution and infectious diseases. As discussed above, some recent studies have
421 shown that biomass burning smoke may influence seasonal influenza incidence rates. For
422 example, Landguth et al. concluded that increases in PM_{2.5} concentrations during the wildfire
423 season led to an increase in the influenza incidence in the following winter influenza season in
424 Montana⁴¹. In parallel, experimental studies also showed that exposure to air pollutants,
425 including PM¹¹⁷ increased susceptibility to viral lung infections by affecting the immune system.
426 A recent study suggested that other infectious agents, such as pathogenic fungus (e.g. causing
427 coccidioidomycosis), may be transported by smoke pollution ¹¹⁸. The interaction between air
428 pollution and infectious diseases such as tuberculosis ¹¹⁹ and coronavirus infection (e.g.
429 SARS¹²⁰) constitutes a novel area of research that is judicious in the context of the COVID-19
430 pandemic ^{42,121,122}. Notably, a recent study by Zhou et al.⁴³ found that the 2020 wildfires in
431 Washington, Oregon and California counties amplified the effect of short-term exposure of PM_{2.5}
432 on COVID-19 cases and deaths.

433

434 **4. Interventions to reduce the wildfire's impact on public health**

435 Our current understanding of the health outcomes associated with smoke pollution can
436 help inform potential preventive strategies to protect public health. In this section, we provide a
437 brief overview of several types of actions that exist to mitigate this health exposure.

438

439 *4.1 Pre-emptive power outages*

440 Certain strategies to reduce fire risk can also bring second-order health effects. For example,
441 pre-emptive de-energization policies in California to reduce ignition sources associated with
442 power lines during extreme fire weather conditions can disproportionately impact the health of
443 communities with lower adaptive capacity¹²³. Power outages may lead to unintended health
444 consequences, such as mental health outcomes, injuries, or heat-related illnesses (through air
445 conditioning interruption, for example). However, to the best of our knowledge, there is no
446 empirical evidence regarding these issues, and we encourage future studies to address such
447 connections.

448

449 *4.2 Land management*

450 Another important area of future research is how land management interventions could
451 reduce the public health burden of wildfire smoke pollution exposure through fuel and prescribed
452 burning management^{124,125}. These land management strategies will likely change health
453 outcomes by altering the magnitude, frequency, timing, and duration of smoke exposure. For
454 example, prescribed fires typically take place during lower atmospheric ventilation conditions to
455 help control fire behavior, which can increase local exposure, whereas intense wildfires may be
456 more likely to be injected higher into the atmosphere, with broader regional effects¹²⁶. Across
457 broader spatial and temporal scales, implementing low level prescribed burning strategies could
458 reduce the risk of extreme wildfire events and minimize large-scale smoke pollution impacts¹²⁷.
459 New research is needed to focus on the unique characteristics of prescribed fires as a coupled
460 human-natural system¹²⁸.

461 Schweizer et al.⁶⁹ compared wildfire and prescribed burning smoke plumes in California
462 using HMS observations and found that larger and more intense fires exposed more people per
463 area burned because the smoke was transported over larger distances. Preliminary studies find
464 worse health outcomes in children exposed to smoke from wildfires compared to prescribed
465 burning¹²⁹. However, quantifying the health benefits of prescribed burning remains highly
466 uncertain¹³⁰. One primary source of this uncertainty is accurately estimating how low level
467 prescribed burns could offset the risk of future, higher emissions from extreme wildfire events
468¹³¹. Another is due to measurement differences between wildfire and prescribed burning
469 exposure estimates, with the proximity of sensors to fires often closer to prescribed burns than
470 extreme wildfire events¹³⁰.

471
472 *4.3 Public health interventions*

473 Limiting the number of people directly exposed to smoke pollution through evacuations
474 is perhaps the most obvious intervention to protect public health^{123,132}. In addition to populations
475 directly exposed to wildfire threats, most of the smoke pollution health burden will be driven by
476 regional exposures due to smoke transport. In this context, early warning systems (EWS) that
477 aim to reduce a population's exposure to smoke pollution by collective or individual behavioral
478 changes are crucial. Several models provide near-real time or forecasted smoke PM_{2.5}
479 concentrations in the U.S. For example, the CDC's National Environmental Public Health
480 Tracking Network provides short-term smoke pollution forecasts to identify at-risk populations
481 and strengthen public health preparedness¹³³. Prior studies suggest that intervention advisories
482 about low PM_{2.5} concentration thresholds, coupled with strong public adherence, can effectively
483 reduce risk¹³⁴ in susceptible populations. In southern Australia, the Air Quality Visualization
484 (AQVx) combines data to assess landscape fire-health effects from smoke exposure and to
485 evaluate dispersion models, allowing targeted warning messages at a local scale¹³⁵. For a
486 discussion of other real-time and operational smoke forecasting systems, we refer the reader to
487 Chapter 9.

488 Actions that take place during such EWS include modifications to work plans, school or
489 business closures and event cancelations. Individual behavioral changes are also urged when
490 EWS are activated such as usage of individual protections (N95 masks or respirators that filter
491 particles), recommendations to stay indoors, limit physical activity, and reduce other activities
492 that impact air quality, such as smoking, wood burning, or traffic emissions¹³⁶. Other long-term
493 structural actions also exist to improve building resilience by improving mechanical ventilation
494 systems to filter incoming air or providing air purifiers with a high efficiency (HEPA) filter.

495 Studies that have evaluated the potential effectiveness of some of these interventions are rare ¹³⁷
496 and more evidence is urgently needed. We strongly encourage future experimental studies that
497 would compare the effectiveness of different actions on various populations as well as quasi-
498 experimental studies as done in the context of other EWS ^{138,139}.

499

500 **5. Conclusions**

501

502 In the context of climate change and variability, health outcomes associated with exposure to
503 smoke pollution are an increasingly important global health issue. Understanding such outcomes
504 in various locations, populations, and for different multiple health endpoints is an urgent priority.
505 In this chapter, we provide a contemporary overview of the epidemiological evidence for adult
506 health outcomes related to smoke pollution exposure. While stronger evidence exists for
507 associations between short-term exposures and all-cause mortality or respiratory morbidity, for
508 example, additional studies are needed to address cardiovascular outcomes, the mental health
509 burden, and vulnerable populations. Geographic disparities exist in existing adult
510 epidemiological studies, which requires additional information to better understand potential
511 regional differences in health outcomes. We discuss how exposure to smoke pollution has been
512 estimated, various methodological considerations for epidemiological study designs, and
513 emerging evidence for several understudied health outcomes. Several opportunities exist to
514 reduce smoke pollution exposure through land use interventions, early warning systems, and
515 behavioral modifications. Taken together, while strong evidence exists for certain health
516 outcomes and regions of the world, future studies will allow us to comprehensively understand
517 the adult health burden of smoke pollution exposure by considering additional health outcomes,
518 interactions among exposures, and additional opportunities to protect health.

519

520

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529

530 **References**

- 531 1. McClure CD, Jaffe DA. US particulate matter air quality improves except in wildfire-prone
532 areas. *Proceedings of the National Academy of Sciences*. 2018;115(31):7901-7906.
- 533 2. Johnston FH, Henderson SB, Chen Y, et al. Estimated global mortality attributable to
534 smoke from landscape fires. *Environmental health perspectives*. 2012;120(5):695-701.
- 535 3. Roberts G, Wooster M. Global impact of landscape fire emissions on surface level PM_{2.5}
536 concentrations, air quality exposure and population mortality. *Atmospheric
537 Environment*. 2021:118210.
- 538 4. Nawaz M, Henze D. Premature deaths in Brazil associated with long-term exposure to
539 PM_{2.5} from Amazon fires between 2016 and 2019. *GeoHealth*.
540 2020;4(8):e2020GH000268.
- 541 5. Butt EW, Conibear L, Reddington CL, et al. Large air quality and human health impacts
542 due to Amazon forest and vegetation fires. *Environmental Research Communications*.
543 2020;2(9):095001.
- 544 6. Koplitz SN, Mickley LJ, Marlier ME, et al. Public health impacts of the severe haze in
545 Equatorial Asia in September–October 2015: demonstration of a new framework for
546 informing fire management strategies to reduce downwind smoke exposure.
547 *Environmental Research Letters*. 2016;11(9):094023.
- 548 7. Kiely L, Spracklen DV, Wiedinmyer C, et al. Air quality and health impacts of vegetation
549 and peat fires in Equatorial Asia during 2004–2015. *Environmental Research Letters*.
550 2020;15(9):094054.
- 551 8. Crippa P, Castruccio S, Archer-Nicholls S, et al. Population exposure to hazardous air
552 quality due to the 2015 fires in Equatorial Asia. *Scientific reports*. 2016;6(1):1-9.
- 553 9. Kollanus V, Prank M, Gens A, et al. Mortality due to vegetation fire–originated PM_{2.5}
554 exposure in Europe—assessment for the years 2005 and 2008. *Environmental health
555 perspectives*. 2017;125(1):30-37.
- 556 10. Wilkins JL, Pouliot G, Foley K, Appel W, Pierce T. The impact of US wildland fires on
557 ozone and particulate matter: a comparison of measurements and CMAQ model
558 predictions from 2008 to 2012. *International journal of wildland fire*. 2018;27(10):684-
559 698.
- 560 11. O’Dell K, Ford B, Fischer EV, Pierce JR. Contribution of wildland-fire smoke to US PM_{2.5}
561 and its influence on recent trends. *Environmental science & technology*.
562 2019;53(4):1797-1804.
- 563 12. Burke M, Driscoll A, Heft-Neal S, Xue J, Burney J, Wara M. The changing risk and
564 burden of wildfire in the United States. *Proceedings of the National Academy of
565 Sciences*. 2021;118(2).
- 566 13. Liu JC, Wilson A, Mickley LJ, et al. Wildfire-specific fine particulate matter and risk of
567 hospital admissions in urban and rural counties. *Epidemiology (Cambridge, Mass)*.
568 2017;28(1):77.
- 569 14. O’Dell K, Bilsback K, Ford B, et al. Estimated Mortality and Morbidity Attributable to
570 Smoke Plumes in the US: Not Just a Western US Problem. *GeoHealth*.
571 2021:e2021GH000457.
- 572 15. Bauer SE, Im U, Mezuman K, Gao CY. Desert dust, industrialization, and agricultural
573 fires: Health impacts of outdoor air pollution in Africa. *Journal of Geophysical Research:
574 Atmospheres*. 2019;124(7):4104-4120.
- 575 16. Cusworth DH, Mickley LJ, Sulprizio MP, et al. Quantifying the influence of agricultural
576 fires in northwest India on urban air pollution in Delhi, India. *Environmental Research
577 Letters*. 2018;13(4):044018.

- 578 17. Liu T, Marlier ME, DeFries RS, et al. Seasonal impact of regional outdoor biomass
579 burning on air pollution in three Indian cities: Delhi, Bengaluru, and Pune. *Atmospheric*
580 *environment*. 2018;172:83-92.
- 581 18. McNamara ML, Semmens EO, Gaskill S, Palmer C, Noonan CW, Ward TJ. Base camp
582 personnel exposure to particulate matter during wildland fire suppression activities.
583 *Journal of occupational and environmental hygiene*. 2012;9(3):149-156.
- 584 19. Navarro KM, Cisneros R, Noth EM, Balmes JR, Hammond SK. Occupational exposure
585 to polycyclic aromatic hydrocarbon of wildland firefighters at prescribed and wildland
586 fires. *Environmental science & technology*. 2017;51(11):6461-6469.
- 587 20. Aisbett B, Wolkow A, Sprajcer M, Ferguson SA. "Awake, smoky, and hot": providing an
588 evidence-base for managing the risks associated with occupational stressors
589 encountered by wildland firefighters. *Applied Ergonomics*. 2012;43(5):916-925.
- 590 21. Groot E, Caturay A, Khan Y, Copes R. A systematic review of the health impacts of
591 occupational exposure to wildland fires. *International journal of occupational medicine*
592 *and environmental health*. 2019;32(2):121-140.
- 593 22. Reid CE, Brauer M, Johnston FH, Jerrett M, Balmes JR, Elliott CT. Critical review of
594 health impacts of wildfire smoke exposure. *Environmental health perspectives*.
595 2016;124(9):1334-1343.
- 596 23. Liu JC, Pereira G, Uhl SA, Bravo MA, Bell ML. A systematic review of the physical health
597 impacts from non-occupational exposure to wildfire smoke. *Environmental research*.
598 2015;136:120-132.
- 599 24. Henderson SB, Johnston FH. Measures of forest fire smoke exposure and their
600 associations with respiratory health outcomes. *Current opinion in allergy and clinical*
601 *immunology*. 2012;12(3):221-227.
- 602 25. Arriagada NB, Horsley JA, Palmer AJ, Morgan GG, Tham R, Johnston FH. Association
603 between fire smoke fine particulate matter and asthma-related outcomes: systematic
604 review and meta-analysis. *Environmental research*. 2019;179:108777.
- 605 26. Youssouf H, Liousse C, Roblou L, et al. Non-accidental health impacts of wildfire smoke.
606 *International journal of environmental research and public health*. 2014;11(11):11772-
607 11804.
- 608 27. Black C, Tesfaigzi Y, Bassein JA, Miller LA. Wildfire smoke exposure and human health:
609 Significant gaps in research for a growing public health issue. *Environmental toxicology*
610 *and pharmacology*. 2017;55:186-195.
- 611 28. Kochi I, Donovan GH, Champ PA, Loomis JB. The economic cost of adverse health
612 effects from wildfire-smoke exposure: a review. *International Journal of Wildland Fire*.
613 2010;19(7):803-817.
- 614 29. Naeher LP, Brauer M, Lipsett M, et al. Woodsmoke health effects: a review. *Inhalation*
615 *toxicology*. 2007;19(1):67-106.
- 616 30. Brook RD, Rajagopalan S, Pope CA, et al. Particulate matter air pollution and
617 cardiovascular disease an update to the scientific statement from the American Heart
618 Association. *Circulation*. 2010;121(21):2331-2378.
- 619 31. Feng S, Gao D, Liao F, Zhou F, Wang X. The health effects of ambient PM_{2.5} and
620 potential mechanisms. *Ecotoxicology and environmental safety*. 2016;128:67-74.
- 621 32. Atkinson R, Kang S, Anderson H, Mills I, Walton H. Epidemiological time series studies
622 of PM_{2.5} and daily mortality and hospital admissions: a systematic review and meta-
623 analysis. *Thorax*. 2014;69(7):660-665.
- 624 33. Fan J, Li S, Fan C, Bai Z, Yang K. The impact of PM_{2.5} on asthma emergency
625 department visits: a systematic review and meta-analysis. *Environmental Science and*
626 *Pollution Research*. 2016;23(1):843-850.
- 627 34. Xing Y-F, Xu Y-H, Shi M-H, Lian Y-X. The impact of PM_{2.5} on the human respiratory
628 system. *Journal of thoracic disease*. 2016;8(1):E69.

- 629 35. Kim YH, Warren SH, Krantz QT, et al. Mutagenicity and lung toxicity of smoldering vs.
630 flaming emissions from various biomass fuels: implications for health effects from
631 wildland fires. *Environmental health perspectives*. 2018;126(1):017011.
- 632 36. Doubleday A, Schulte J, Sheppard L, et al. Mortality associated with wildfire smoke
633 exposure in Washington state, 2006–2017: a case-crossover study. *Environmental*
634 *health*. 2020;19(1):1-10.
- 635 37. Chen G, Guo Y, Yue X, et al. Mortality risk attributable to wildfire-related PM_{2.5}
636 pollution: a global time series study in 749 locations. *The Lancet Planetary Health*.
637 2021;5(9):e579-e587.
- 638 38. Caamano-Isorna F, Figueiras A, Sastre I, Montes-Martínez A, Taracido M, Piñeiro-
639 Lamas M. Respiratory and mental health effects of wildfires: an ecological study in
640 Galician municipalities (north-west Spain). *Environmental Health*. 2011;10(1):1-9.
- 641 39. Vora C, Renvall MJ, Chao P, Ferguson P, Ramsdell JW. 2007 San Diego wildfires and
642 asthmatics. *Journal of Asthma*. 2011;48(1):75-78.
- 643 40. Gayle AV, Quint JK, Fuertes EI. Understanding the relationships between environmental
644 factors and exacerbations of COPD. *Expert Review of Respiratory Medicine*.
645 2021;15(1):39-50.
- 646 41. Landguth EL, Holden ZA, Graham J, et al. The delayed effect of wildfire season
647 particulate matter on subsequent influenza season in a mountain west region of the
648 USA. *Environment international*. 2020;139:105668.
- 649 42. Henderson SB. The CoViD-19 pandemic and wildfire smoke: potentially concomitant
650 disasters. In: American Public Health Association; 2020.
- 651 43. Zhou X, Josey K, Kamareddine L, et al. Excess of COVID-19 cases and deaths due to
652 fine particulate matter exposure during the 2020 wildfires in the United States. *Science*
653 *Advances*. 2021;7(33):eabi8789.
- 654 44. Arbex MA, Saldiva PHN, Pereira LAA, Braga ALF. Impact of outdoor biomass air
655 pollution on hypertension hospital admissions. *Journal of Epidemiology & Community*
656 *Health*. 2010;64(7):573-579.
- 657 45. Rappold AG, Cascio WE, Kilaru VJ, et al. Cardio-respiratory outcomes associated with
658 exposure to wildfire smoke are modified by measures of community health.
659 *Environmental Health*. 2012;11(1):1-9.
- 660 46. Crabbe H. Risk of respiratory and cardiovascular hospitalisation with exposure to
661 bushfire particulates: new evidence from Darwin, Australia. *Environmental geochemistry*
662 *and health*. 2012;34(6):697-709.
- 663 47. Dennekamp M, Straney LD, Erbas B, et al. Forest fire smoke exposures and out-of-
664 hospital cardiac arrests in Melbourne, Australia: a case-crossover study. *Environmental*
665 *health perspectives*. 2015;123(10):959-964.
- 666 48. Haikerwal A, Akram M, Del Monaco A, et al. Impact of fine particulate matter (PM_{2.5})
667 exposure during wildfires on cardiovascular health outcomes. *Journal of the American*
668 *Heart Association*. 2015;4(7):e001653.
- 669 49. Rappold AG, Reyes J, Pouliot G, Cascio WE, Diaz-Sanchez D. Community vulnerability
670 to health impacts of wildland fire smoke exposure. *Environmental Science &*
671 *Technology*. 2017;51(12):6674-6682.
- 672 50. Liu JC, Wilson A, Mickley LJ, et al. Who among the elderly is most vulnerable to
673 exposure to and health risks of fine particulate matter from wildfire smoke? *American*
674 *journal of epidemiology*. 2017;186(6):730-735.
- 675 51. Forastiere F, Stafoggia M, Tasco C, et al. Socioeconomic status, particulate air pollution,
676 and daily mortality: differential exposure or differential susceptibility. *American journal of*
677 *industrial medicine*. 2007;50(3):208-216.
- 678 52. Hajat A, Hsia C, O'Neill MS. Socioeconomic Disparities and Air Pollution Exposure: a
679 Global Review. *Current environmental health reports*. 2015;2(4):440-450.

- 680 53. Benmarhnia T, Hajat A, Kaufman JS. Inferential challenges when assessing racial/ethnic
681 health disparities in environmental research. *Environmental Health*. 2021;20(1):1-10.
- 682 54. Ignotti E, Valente JG, Longo KM, Freitas SR, Hacon SdS, Artaxo Netto P. Impact on
683 human health of particulate matter emitted from burnings in the Brazilian Amazon region.
684 *Revista de saude publica*. 2010;44:121-130.
- 685 55. Morgan G, Sheppard V, Khalaj B, et al. Effects of bushfire smoke on daily mortality and
686 hospital admissions in Sydney, Australia. *Epidemiology*. 2010:47-55.
- 687 56. de Oliveira G, Chen JM, Stark SC, et al. Smoke pollution's impacts in Amazonia.
688 *Science (New York, NY)*. 2020;369(6504):634-635.
- 689 57. Henderson SB, Brauer M, MacNab YC, Kennedy SM. Three measures of forest fire
690 smoke exposure and their associations with respiratory and cardiovascular health
691 outcomes in a population-based cohort. *Environmental health perspectives*.
692 2011;119(9):1266-1271.
- 693 58. Hanigan IC, Johnston FH, Morgan GG. Vegetation fire smoke, indigenous status and
694 cardio-respiratory hospital admissions in Darwin, Australia, 1996–2005: a time-series
695 study. *Environmental Health*. 2008;7(1):1-12.
- 696 59. Jayachandran S. Air quality and early-life mortality evidence from Indonesia's wildfires.
697 *Journal of Human resources*. 2009;44(4):916-954.
- 698 60. Chen H, Samet JM, Bromberg PA, Tong H. Cardiovascular health impacts of wildfire
699 smoke exposure. *Particle and Fibre Toxicology*. 2021;18(1):1-22.
- 700 61. Voulgarakis A, Field RD. Fire influences on atmospheric composition, air quality and
701 climate. *Current Pollution Reports*. 2015;1(2):70-81.
- 702 62. Johnson AL, Abramson MJ, Dennekamp M, Williamson GJ, Guo Y. Particulate matter
703 modelling techniques for epidemiological studies of open biomass fire smoke exposure:
704 a review. *Air Quality, Atmosphere & Health*. 2020;13(1):35-75.
- 705 63. Green H, Bailey J, Schwarz L, Vanos J, Ebi K, Benmarhnia T. Impact of heat on
706 mortality and morbidity in low and middle income countries: a review of the
707 epidemiological evidence and considerations for future research. *Environmental
708 research*. 2019;171:80-91.
- 709 64. Giglio L, Randerson JT, van der Werf GR. Global Fire Emissions Indicators, Grids: 1997-
710 2015. In. Palisades, NY: NASA Socioeconomic Data and Applications Center (SEDAC);
711 2018.
- 712 65. Giglio L, Randerson JT, van der Werf GR. Analysis of Daily, Monthly, and Annual
713 Burned Area Using the Fourth-Generation Global Fire Emissions Database (GFED4).
714 *Journal of Geophysical Research*. 2013;118(1):317-328.
- 715 66. Lelieveld J, Evans JS, Fnais M, Giannadaki D, Pozzer A. The contribution of outdoor air
716 pollution sources to premature mortality on a global scale. *Nature*. 2015;525(7569):367-
717 371.
- 718 67. Li Y, Tong D, Ngan F, et al. Ensemble PM_{2.5} Forecasting During the 2018 Camp Fire
719 Event Using the HYSPLIT Transport and Dispersion Model. *Journal of Geophysical
720 Research: Atmospheres*. 2020;125(15):e2020JD032768.
- 721 68. Brey SJ, Ruminski M, Atwood SA, Fischer EV. Connecting smoke plumes to sources
722 using Hazard Mapping System (HMS) smoke and fire location data over North America.
723 *Atmospheric Chemistry and Physics*. 2018;18(3):1745-1761.
- 724 69. Schweizer D, Preisler HK, Cisneros R. Assessing relative differences in smoke exposure
725 from prescribed, managed, and full suppression wildland fire. *Air Quality, Atmosphere &
726 Health*. 2019;12(1):87-95.
- 727 70. Raffuse SM, McCarthy MC, Craig KJ, et al. High-resolution MODIS aerosol retrieval
728 during wildfire events in California for use in exposure assessment. *Journal of
729 Geophysical Research: Atmospheres*. 2013;118(19):11,242-211,255.

- 730 71. Torres O, Jethva H, Ahn C, Jaross G, Loyola DG. TROPOMI aerosol products:
731 evaluation and observations of synoptic-scale carbonaceous aerosol plumes during
732 2018–2020. *Atmospheric Measurement Techniques*. 2020;13(12):6789-6806.
- 733 72. Gupta P, Doraiswamy P, Levy R, et al. Impact of California fires on local and regional air
734 quality: The role of a low-cost sensor network and satellite observations. *GeoHealth*.
735 2018;2(6):172-181.
- 736 73. Aguilera R, Hansen K, Gershunov A, Ilango SD, Sheridan P, Benmarhnia T. Respiratory
737 hospitalizations and wildfire smoke: a spatiotemporal analysis of an extreme firestorm in
738 San Diego County, California. *Environmental Epidemiology*. 2020;4(5):e114.
- 739 74. Cleland SE, Serre ML, Rappold AG, West JJ. Estimating the acute health impacts of fire-
740 originated PM_{2.5} exposure during the 2017 California Wildfires: Sensitivity to choices of
741 inputs. *GeoHealth*. 2021;5(7):e2021GH000414.
- 742 75. Lassman W, Ford B, Gan RW, et al. Spatial and temporal estimates of population
743 exposure to wildfire smoke during the Washington state 2012 wildfire season using
744 blended model, satellite, and in situ data. *GeoHealth*. 2017;1(3):106-121.
- 745 76. Fadadu RP, Balmes JR, Holm SM. Differences in the Estimation of Wildfire-Associated
746 Air Pollution by Satellite Mapping of Smoke Plumes and Ground-Level Monitoring.
747 *International Journal of Environmental Research and Public Health*. 2020;17(21):8164.
- 748 77. Hutchinson JA, Vargo J, Milet M, et al. The San Diego 2007 wildfires and Medi-Cal
749 emergency department presentations, inpatient hospitalizations, and outpatient visits: An
750 observational study of smoke exposure periods and a bidirectional case-crossover
751 analysis. *PLoS medicine*. 2018;15(7):e1002601.
- 752 78. Shaposhnikov D, Revich B, Bellander T, et al. Long-term impact of moscow heat wave
753 and wildfires on mortality. *Epidemiology*. 2015;26(2):e21-e22.
- 754 79. Leibel S, Nguyen M, Brick W, et al. Increase in pediatric respiratory visits associated
755 with Santa Ana wind-driven wildfire smoke and PM_{2.5} levels in San Diego County.
756 *Annals of the American Thoracic Society*. 2020;17(3):313-320.
- 757 80. Kim Y, Knowles S, Manley J, Radoias V. Long-run health consequences of air pollution:
758 Evidence from Indonesia's forest fires of 1997. *Economics & Human Biology*.
759 2017;26:186-198.
- 760 81. Angrist JD, Pischke J-S. *Mostly harmless econometrics: An empiricist's companion*.
761 Princeton university press; 2008.
- 762 82. Fouillet A, Rey G, Laurent F, et al. Excess mortality related to the August 2003 heat
763 wave in France. *International archives of occupational and environmental health*.
764 2006;80(1):16-24.
- 765 83. Sandberg J, Santos-Burgoa C, Roess A, et al. All over the place?: differences in and
766 consistency of excess mortality estimates in Puerto Rico after Hurricane Maria.
767 *Epidemiology*. 2019;30(4):549-552.
- 768 84. Liu JC, Mickley LJ, Sulprizio MP, et al. Particulate air pollution from wildfires in the
769 Western US under climate change. *Climatic change*. 2016;138(3-4):655-666.
- 770 85. Liu JC, Mickley LJ, Sulprizio MP, et al. Future respiratory hospital admissions from
771 wildfire smoke under climate change in the Western US. *Environmental Research
772 Letters*. 2016;11(12):124018.
- 773 86. Kolbe A, Gilchrist KL. An extreme bushfire smoke pollution event: health impacts and
774 public health challenges. *New South Wales public health bulletin*. 2009;20(2):19-23.
- 775 87. Reid CE, Jerrett M, Tager IB, Petersen ML, Mann JK, Balmes JR. Differential respiratory
776 health effects from the 2008 northern California wildfires: a spatiotemporal approach.
777 *Environmental research*. 2016;150:227-235.
- 778 88. Eisenman D, McCaffrey S, Donatello I, Marshal G. An ecosystems and vulnerable
779 populations perspective on solastalgia and psychological distress after a wildfire.
780 *EcoHealth*. 2015;12(4):602-610.

- 781 89. Bryant RA, Waters E, Gibbs L, et al. Psychological outcomes following the Victorian
782 Black Saturday bushfires. *Australian & New Zealand Journal of Psychiatry*.
783 2014;48(7):634-643.
- 784 90. Dodd W, Scott P, Howard C, et al. Lived experience of a record wildfire season in the
785 Northwest Territories, Canada. *Canadian Journal of Public Health*. 2018;109(3):327-337.
- 786 91. Felix ED, Afifi W. The role of social support on mental health after multiple wildfire
787 disasters. *Journal of Community Psychology*. 2015;43(2):156-170.
- 788 92. Xi Y, Kshirsagar AV, Wade TJ, et al. Mortality in US hemodialysis patients following
789 exposure to Wildfire smoke. *Journal of the American Society of Nephrology*.
790 2020;31(8):1824-1835.
- 791 93. Versura P, Profazio V, Cellini M, Torreggiani A, Caramazza R. Eye discomfort and air
792 pollution. *Ophthalmologica*. 1999;213(2):103-109.
- 793 94. Cleland HJ, Proud D, Spinks A, Wasiak J. Multidisciplinary team response to a mass
794 burn casualty event: outcomes and implications. *Medical journal of Australia*.
795 2011;194(11):589-593.
- 796 95. Yang B-Y, Fan S, Thiering E, et al. Ambient air pollution and diabetes: a systematic
797 review and meta-analysis. *Environmental research*. 2020;180:108817.
- 798 96. Weuve J, Bennett EE, Ranker L, et al. Exposure to Air Pollution in Relation to Risk of
799 Dementia and Related Outcomes: An Updated Systematic Review of the
800 Epidemiological Literature. *Environmental health perspectives*. 2021;129(9):096001.
- 801 97. Gill AM, Stephens SL. Scientific and social challenges for the management of fire-prone
802 wildland–urban interfaces. *Environmental Research Letters*. 2009;4(3):034014.
- 803 98. Carratt SA, Flayer CH, Kossack ME, Last JA. Pesticides, wildfire suppression chemicals,
804 and California wildfires: A human health perspective. *Current topics in Toxicology*.
805 2017;13:1-12.
- 806 99. Penman TD, Collins L, Syphard AD, Keeley JE, Bradstock RA. Influence of fuels,
807 weather and the built environment on the exposure of property to wildfire. *PLoS One*.
808 2014;9(10):e111414.
- 809 100. Strader SM. Spatiotemporal changes in conterminous US wildfire exposure from 1940 to
810 2010. *Natural hazards*. 2018;92(1):543-565.
- 811 101. McFarlane BL, McGee TK, Faulkner H. Complexity of homeowner wildfire risk mitigation:
812 an integration of hazard theories. *International Journal of Wildland Fire*. 2011;20(8):921-
813 931.
- 814 102. Schumann III RL, Mockrin M, Syphard AD, et al. Wildfire recovery as a “hot moment” for
815 creating fire-adapted communities. *International journal of disaster risk reduction*.
816 2020;42:101354.
- 817 103. Reid CE, Considine EM, Watson GL, Telesca D, Pfister GG, Jerrett M. Associations
818 between respiratory health and ozone and fine particulate matter during a wildfire event.
819 *Environment international*. 2019;129:291-298.
- 820 104. Watson GL, Telesca D, Reid CE, Pfister GG, Jerrett M. Machine learning models
821 accurately predict ozone exposure during wildfire events. *Environmental Pollution*.
822 2019;254:112792.
- 823 105. Wegesser TC, Pinkerton KE, Last JA. California wildfires of 2008: coarse and fine
824 particulate matter toxicity. *Environmental health perspectives*. 2009;117(6):893-897.
- 825 106. Wegesser TC, Franzi LM, Mitloehner FM, Eiguren-Fernandez A, Last JA. Lung
826 antioxidant and cytokine responses to coarse and fine particulate matter from the great
827 California wildfires of 2008. *Inhalation toxicology*. 2010;22(7):561-570.
- 828 107. Franzi LM, Bratt JM, Williams KM, Last JA. Why is particulate matter produced by
829 wildfires toxic to lung macrophages? *Toxicology and applied pharmacology*.
830 2011;257(2):182-188.

- 831 108. DeFlorio-Barker S, Crooks J, Reyes J, Rappold AG. Cardiopulmonary effects of fine
832 particulate matter exposure among older adults, during wildfire and non-wildfire periods,
833 in the United States 2008–2010. *Environmental health perspectives*.
834 2019;127(3):037006.
- 835 109. Kiser D, Metcalf WJ, Elhanan G, et al. Particulate matter and emergency visits for
836 asthma: a time-series study of their association in the presence and absence of wildfire
837 smoke in Reno, Nevada, 2013–2018. *Environmental Health*. 2020;19(1):1-12.
- 838 110. Aguilera R, Gershunov A, Corringham TW, Benmarhnia T. Wildfire smoke impacts
839 respiratory health more than fine particles from other sources: observational evidence
840 from Southern California. *Nature communications*. 2021;In Press.
- 841 111. Ford B, Val Martin M, Zelasky S, et al. Future fire impacts on smoke concentrations,
842 visibility, and health in the contiguous United States. *GeoHealth*. 2018;2(8):229-247.
- 843 112. Jolly WM, Cochrane MA, Freeborn PH, et al. Climate-induced variations in global wildfire
844 danger from 1979 to 2013. *Nature communications*. 2015;6(1):1-11.
- 845 113. Spracklen DV, Mickley LJ, Logan JA, et al. Impacts of climate change from 2000 to 2050
846 on wildfire activity and carbonaceous aerosol concentrations in the western United
847 States. *Journal of Geophysical Research: Atmospheres*. 2009;114(D20).
- 848 114. Liu JC, Mickley LJ, Sulprizio MP, et al. Particulate air pollution from wildfires in the
849 Western US under climate change. *Climatic change*. 2016;138(3):655-666.
- 850 115. Xu R, Yu P, Abramson MJ, et al. Wildfires, global climate change, and human health.
851 *New England Journal of Medicine*. 2020;383(22):2173-2181.
- 852 116. Anenberg SC, Haines S, Wang E, Nassikas N, Kinney PL. Synergistic health effects of
853 air pollution, temperature, and pollen exposure: a systematic review of epidemiological
854 evidence. *Environmental Health*. 2020;19(1):1-19.
- 855 117. Zelikoff JT, Chen LC, Cohen MD, et al. Effects of inhaled ambient particulate matter on
856 pulmonary antimicrobial immune defense. *Inhalation Toxicology*. 2003;15(2):131-150.
- 857 118. Kobziar LN, Thompson GR. Wildfire smoke, a potential infectious agent. *Science*.
858 2020;370(6523):1408-1410.
- 859 119. Popovic I, Magalhaes RJS, Ge E, et al. A systematic literature review and critical
860 appraisal of epidemiological studies on outdoor air pollution and tuberculosis outcomes.
861 *Environmental research*. 2019;170:33-45.
- 862 120. Cui Y, Zhang Z-F, Froines J, et al. Air pollution and case fatality of SARS in the People's
863 Republic of China: an ecologic study. *Environmental Health*. 2003;2(1):1-5.
- 864 121. Benmarhnia T. Linkages between air pollution and the health burden from COVID-19:
865 methodological challenges and opportunities. *American journal of epidemiology*.
866 2020;189(11):1238-1243.
- 867 122. Villeneuve PJ, Goldberg MS. Methodological considerations for epidemiological studies
868 of air pollution and the SARS and COVID-19 coronavirus outbreaks. *Environmental
869 health perspectives*. 2020;128(9):095001.
- 870 123. Abatzoglou JT, Smith CM, Swain DL, Ptak T, Kolden CA. Population exposure to pre-
871 emptive de-energization aimed at averting wildfires in Northern California. *Environmental
872 Research Letters*. 2020.
- 873 124. Schoennagel T, Balch JK, Brenkert-Smith H, et al. Adapt to more wildfire in western
874 North American forests as climate changes. *Proceedings of the National Academy of
875 Sciences*. 2017;114(18):4582-4590.
- 876 125. Jaffe DA, O'Neill SM, Larkin NK, et al. Wildfire and prescribed burning impacts on air
877 quality in the United States. *Journal of the Air & Waste Management Association*.
878 2020;70(6):583-615.
- 879 126. Williamson GJ, Bowman DMS, Price OF, Henderson S, Johnston F. A transdisciplinary
880 approach to understanding the health effects of wildfire and prescribed fire smoke
881 regimes. *Environmental Research Letters*. 2016;11(12):125009.

- 882 127. Long JW, Tarnay LW, North MP. Aligning smoke management with ecological and public
883 health goals. *Journal of Forestry*. 2018;116(1):76-86.
- 884 128. Hiers JK, O'Brien JJ, Varner JM, et al. Prescribed fire science: the case for a refined
885 research agenda. *Fire Ecology*. 2020;16(1):1-15.
- 886 129. Prunicki M, Rodd Kelsey JL, Zhou X, et al. The impact of prescribed fire versus wildfire
887 on the immune and cardiovascular systems of children. *Allergy*. 2019;74(10):1989.
- 888 130. Navarro KM, Schweizer D, Balmes JR, Cisneros R. A review of community smoke
889 exposure from wildfire compared to prescribed fire in the United States. *Atmosphere*.
890 2018;9(5):185.
- 891 131. Schweizer D, Cisneros R. Forest fire policy: change conventional thinking of smoke
892 management to prioritize long-term air quality and public health. *Air Quality, Atmosphere
893 & Health*. 2017;10(1):33-36.
- 894 132. McCaffrey S, Wilson R, Konar A. Should I stay or should I go now? Or should I wait and
895 see? Influences on wildfire evacuation decisions. *Risk analysis*. 2018;38(7):1390-1404.
- 896 133. Vaidyanathan A, Yip F, Garbe P. Developing an online tool for identifying at-risk
897 populations to wildfire smoke hazards. *Science of the total environment*. 2018;619:376-
898 383.
- 899 134. Rappold AG, Fann NL, Crooks J, et al. Forecast-based interventions can reduce the
900 health and economic burden of wildfires. *Environmental science & technology*.
901 2014;48(18):10571-10579.
- 902 135. Williamson GJ, Lucani C. AQVx—An Interactive Visual Display System for Air Pollution
903 and Public Health. *Frontiers in public health*. 2020;8:85.
- 904 136. Holm SM, Miller MD, Balmes JR. Health effects of wildfire smoke in children and public
905 health tools: a narrative review. *Journal of exposure science & environmental
906 epidemiology*. 2021;31(1):1-20.
- 907 137. Yao J, Brauer M, Henderson SB. Evaluation of a wildfire smoke forecasting system as a
908 tool for public health protection. *Environmental health perspectives*. 2013;121(10):1142.
- 909 138. Benmarhnia T, Bailey Z, Kaiser D, Auger N, King N, Kaufman JS. A difference-in-
910 differences approach to assess the effect of a heat action plan on heat-related mortality,
911 and differences in effectiveness according to sex, age, and socioeconomic status
912 (Montreal, Quebec). *Environmental health perspectives*. 2016;124(11):1694.
- 913 139. Chen H, Li Q, Kaufman JS, et al. Effect of air quality alerts on human health: a
914 regression discontinuity analysis in Toronto, Canada. *The Lancet Planetary Health*.
915 2018;2(1):e19-e26.
- 916