

# 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.

1 **Title: Wildfire smoke exposures and adult health outcomes**

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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*

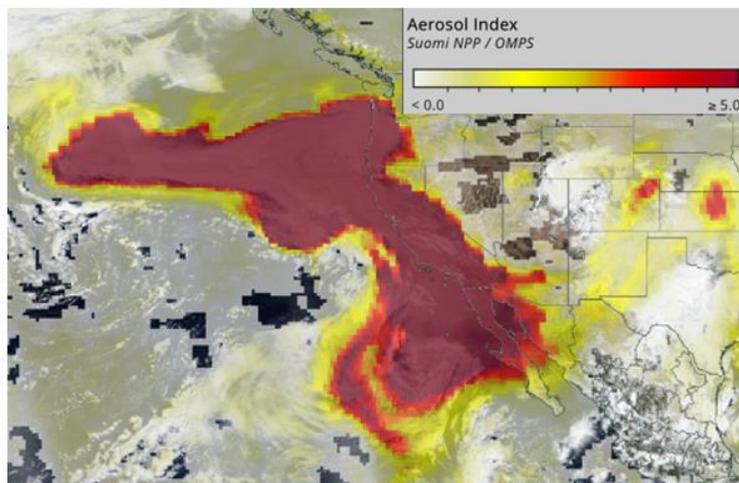
25 Landscape fires can have devastating impacts on human health through contributions to  
26 surface air pollution. Fires contribute to enhanced surface concentrations of fine particulate  
27 matter (PM<sub>2.5</sub>; particles < 2.5 microns in diameter) and trace gases such as ozone (O<sub>3</sub>), 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 decades<sup>1</sup>.

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 10<sup>th</sup>, 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<sub>2.5</sub>  
55 exposure. This provides useful information across several dimensions: (1) the relative contribution

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

59 Global exposure to smoke PM<sub>2.5</sub> 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)<sup>2,3</sup>. More than 44 million people around the world are exposed to unhealthy annual  
63 average PM<sub>2.5</sub> smoke pollution (> 55 µg/m<sup>3</sup>)<sup>3</sup>. 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.<sup>2</sup> and Roberts and Wooster  
69<sup>3</sup> highlighted sub-Saharan Africa and Southeast Asia as global hotspots in driving the attributable  
70 mortality burden of smoke PM<sub>2.5</sub> exposure. Recent estimates suggest nearly 10% of premature  
71 deaths due to PM<sub>2.5</sub> exposure in Brazil were linked to smoke pollution<sup>4</sup>. Preventing vegetation  
72 fires in the Amazon Basin could avert approximately 17,000 premature deaths due to smoke  
73 PM<sub>2.5</sub> exposure<sup>5</sup>. 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<sup>6,7</sup> and the  
75 exposure of nearly 70 million people to unhealthy smoke pollution levels<sup>8</sup>.

76 Landscape fires also contribute to local and regional pollution in non-equatorial regions.  
77 Vegetation fires were linked to short-term PM<sub>2.5</sub> increases in southern and eastern Europe and  
78 low-to-moderate increases in daily PM<sub>2.5</sub> across the continent<sup>9</sup>. 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<sub>2.5</sub>. Across the U.S., fires contribute to approximately 11% of  
81 PM<sub>2.5</sub> and 1% of O<sub>3</sub> on average but play a more important role in western states<sup>10</sup> and during  
82 extreme weather events<sup>10,11</sup>. For instance, fires can contribute up to 50% of PM<sub>2.5</sub> in some parts  
83 of the western U.S.<sup>12</sup>. 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<sub>2.5</sub>), with corresponding increases  
85 in respiratory hospital admissions<sup>13</sup>. A recent study estimated the number of asthma hospital  
86 admissions, emergency department visits, and premature deaths attributable to acute smoke  
87 PM<sub>2.5</sub> exposure across the U.S. using concentration-response functions (CRFs refer to the  
88 estimated dose-response between levels of PM<sub>2.5</sub> and the risk of observing a given health  
89 outcome of interest) specific to smoke PM<sub>2.5</sub> exposure as well as gas-phase hazardous air  
90 pollutants (HAPs)<sup>14</sup>. 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 year<sup>15</sup>. 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<sup>16,17</sup>. 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<sub>2.5</sub> or (more rarely) for smoke PM<sub>2.5</sub> 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) <sup>18-21</sup>. The perinatal and child health burden is discussed in the following chapter. We primarily document the health impacts associated with PM<sub>2.5</sub> 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<sub>2.5</sub> 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 (O<sub>3</sub>), 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)<sup>14</sup>.

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 <sup>22-29</sup>. PM<sub>2.5</sub> is one of the primary constituents of smoke pollution and is the focus of this section. Ambient PM<sub>2.5</sub> 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<sub>2.5</sub> also impacts human health through systemic inflammation and activation of the autonomic nervous system<sup>30</sup>. Evidence regarding the health effects of all-source PM<sub>2.5</sub> is vast, and several reviews have been published in the past decades <sup>31-34</sup>, 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<sub>2.5</sub> 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<sub>2.5</sub>, recent research justifies studying smoke as a separate

148 exposure for several reasons. First, PM<sub>2.5</sub> 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<sub>2.5</sub> may be more toxic than equal doses of PM<sub>2.5</sub> from other sources due  
154 to particle composition<sup>35</sup>. Current air pollution guidelines or regulations do not distinguish by  
155 emissions source or chemical composition for PM<sub>2.5</sub>. 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 days<sup>36</sup>. 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<sup>22,23</sup>. In a recently published global analysis, Chen et al.<sup>37</sup>  
167 found an annual average of 33,510 all-cause deaths to be attributable to smoke PM<sub>2.5</sub> 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<sup>38 39</sup>, physician visits, and emergency departments  
175 (ED) visits or hospital admissions for respiratory problems<sup>25</sup>. 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 review<sup>25</sup> focusing on asthma-related outcomes found consistent evidence for  
179 this outcome.

180 Fewer studies have examined changes in lung function<sup>25</sup>. 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 results<sup>22</sup>. Finally, there is increasing evidence  
184 that wildfire smoke also exacerbates Chronic Obstructive Pulmonary Disease (COPD)<sup>40</sup>. More  
185 recent studies also investigated the role of smoke pollution on exacerbating respiratory infections  
186 or disease severity such as for seasonal influenza<sup>41</sup>; such connections between respiratory  
187 infectious diseases and wildfire are particularly relevant in the context of the COVID-19  
188 pandemic<sup>42,43</sup>.

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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 hypertension<sup>44</sup>. Most CVD studies assessed hospital admissions or ED

194 visits for CVD causes<sup>22</sup>. Fewer studies investigate specific CVD endpoints, like congestive heart  
195 failure<sup>45</sup>, ischemic heart disease<sup>46</sup>, cardiac arrest<sup>47</sup> or myocardial infarction<sup>48</sup>. 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<sup>49,50</sup>. 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)<sup>51</sup>. 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<sub>2.5</sub> in particular<sup>52</sup>.

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 interest<sup>53</sup>. Among  
212 these studies, most focused on age as a susceptibility factor<sup>48,54,55</sup>. 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 age<sup>22</sup>. Several studies assessed potential gender  
216 heterogeneity, but the results are mixed<sup>22</sup>.

217 Differential susceptibility across socioeconomic and racial/ethnic groups, including  
218 individual race or ethnicity<sup>50</sup>, neighborhood SES<sup>57</sup>, indigenous status<sup>58</sup> or proxies such as  
219 district-specific food consumption<sup>59</sup> 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 year<sup>57</sup>) but available evidence is inconclusive to date.

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

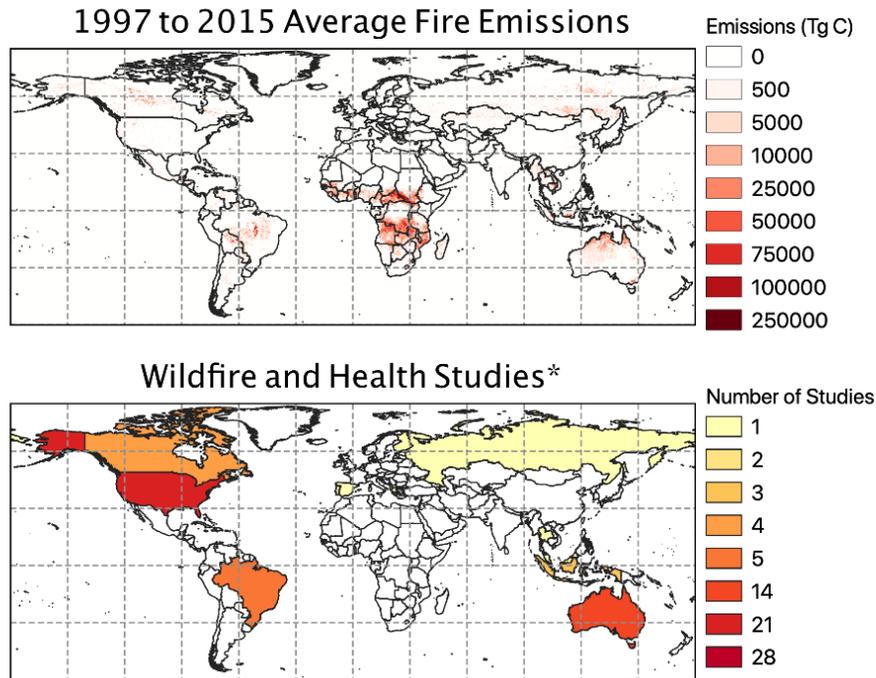
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#### 227 *3.1 Geographic disparities in health studies*

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 vulnerable<sup>60</sup>. 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<sup>61,62</sup>. The geographical distribution of existing epidemiological studies  
234 highlighted an important gap, which is not unique to smoke pollution<sup>63</sup>. 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).

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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 <sup>64,65</sup>.



\* The included studies are extracted from 4 recent systematic reviews <sup>22-25</sup>

### 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. <sup>66</sup> and Johnston et al. <sup>2</sup> 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 <sup>67</sup>. 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 <sup>12,68,69</sup>. 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<sub>2.5</sub> concentrations during fire events <sup>70,71</sup>. 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<sub>2.5</sub> during wildfire events due to their  
266 dense spatial coverage<sup>72</sup>.

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 codes<sup>73</sup>, 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<sub>2.5</sub> concentrations. For example, ground station monitors  
277 provide surface-level estimates of PM<sub>2.5</sub> 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.<sup>74</sup> 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.<sup>62</sup> 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.<sup>75</sup> also found more accurate wildfire  
285 PM<sub>2.5</sub> 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 useful<sup>15</sup>. Finally, recognizing the implications of exposure  
290 method for issuing public health guidance is critical. Fadadu et al.<sup>76</sup>, 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.

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### 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 wildfires<sup>77</sup>, summer Russian wildfires in 2010<sup>78</sup>, or Indonesia's forest fires of 1997<sup>59</sup>. In such  
303 settings, authors relied on various study designs including case crossover designs<sup>77</sup>, interrupted  
304 time series designs<sup>79</sup> or panel analyses<sup>80</sup>. 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 trends<sup>81</sup>. 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 years<sup>78</sup>. This technique has been frequently used in the context of extreme weather  
314 events, like extreme heat, hurricanes to estimate excess mortality<sup>82,83</sup>. 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<sup>84,85</sup> and individual designs based on existing cohorts, nested  
323 case-control designs or ad hoc surveys<sup>86</sup>.

324 Finally, several studies<sup>73,87</sup> 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)<sup>88</sup>, changing psychological outcomes<sup>89</sup> or mental and emotional well-  
334 being<sup>90</sup>. 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 & Afifi<sup>91</sup> 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 diabetic<sup>92</sup> or ophthalmologic<sup>93</sup> outcomes, as well as injuries<sup>94</sup> were investigated by few studies  
343 and more evidence is definitively warranted. Furthermore, given the emerging literature linking  
344 exposure to PM<sub>2.5</sub> and incident diabetes<sup>95</sup> and dementia<sup>96</sup>, 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 function<sup>97</sup>, 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 review<sup>98</sup> 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<sub>2.5</sub>. More developed is the literature addressing the protection  
368 of the built environment from biomass burning events Penman et al.<sup>99</sup> 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 2010<sup>100</sup>, increasing the likelihood of the built environment's involvement in wildfire events.  
377 Wildfire adaptation has been investigated at the individual<sup>101</sup> and community<sup>102</sup> 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<sub>2.5</sub> as the  
383 main component of biomass burning smoke exposure, other pollutants, such as O<sub>3</sub> or polycyclic  
384 aromatic hydrocarbons (PAHs) are generated in fire plumes. Recent studies have shown that  
385 wildfires generate increases in tropospheric O<sub>3</sub> levels through processes distinct from PM<sub>2.5</sub>  
386 <sup>103,104</sup>. In future studies, it will be particularly important to understand how smoke pollutants  
387 other than PM<sub>2.5</sub> impact population health and study potential synergies among these pollutants.

388 Another important area of research relates to whether PM<sub>2.5</sub> smoke pollution affects  
389 health outcomes differently from PM<sub>2.5</sub> 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<sub>2.5</sub> compared to ambient sources <sup>35,105-107</sup>, evidence at the population level is lacking.  
392 To the best of our knowledge, only two studies <sup>108,109</sup> focusing on asthma addressed this research  
393 question. Furthermore, in a recent study, it has also been shown that smoke PM<sub>2.5</sub> can cause a  
394 greater impact on respiratory health than PM<sub>2.5</sub> from other sources <sup>110</sup>. 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<sub>2.5</sub> 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 <sup>12,111</sup>. As the length of wildfire seasons increases<sup>112</sup>, 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<sub>2.5</sub> concentrations under multiple climate change scenarios  
405 suggests an increasing threat to public health, particularly for many vulnerable subpopulations  
406 <sup>111,113,114</sup>. 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) <sup>115</sup>.

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 <sup>116</sup>. 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<sub>2.5</sub> concentrations during the wildfire  
423 season led to an increase in the influenza incidence in the following winter influenza season in  
424 Montana<sup>41</sup>. In parallel, experimental studies also showed that exposure to air pollutants,  
425 including PM<sup>117</sup> 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 <sup>118</sup>. The interaction between air  
428 pollution and infectious diseases such as tuberculosis <sup>119</sup> and coronavirus infection (e.g.  
429 SARS<sup>120</sup>) constitutes a novel area of research that is judicious in the context of the COVID-19  
430 pandemic <sup>42,121,122</sup>. Notably, a recent study by Zhou et al.<sup>43</sup> found that the 2020 wildfires in  
431 Washington, Oregon and California counties amplified the effect of short-term exposure of PM<sub>2.5</sub>  
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 capacity<sup>123</sup>. 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<sup>124,125</sup>. 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 effects<sup>126</sup>. 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 impacts<sup>127</sup>.  
459 New research is needed to focus on the unique characteristics of prescribed fires as a coupled  
460 human-natural system<sup>128</sup>.

461 Schweizer et al.<sup>69</sup> 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 burning<sup>129</sup>. However, quantifying the health benefits of prescribed burning remains highly  
466 uncertain<sup>130</sup>. 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  
468<sup>131</sup>. 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 events<sup>130</sup>.

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<sup>123,132</sup>. 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<sub>2.5</sub>  
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 preparedness<sup>133</sup>. Prior studies suggest that intervention advisories  
482 about low PM<sub>2.5</sub> concentration thresholds, coupled with strong public adherence, can effectively  
483 reduce risk<sup>134</sup> 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 scale<sup>135</sup>. 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 emissions<sup>136</sup>. 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 <sup>137</sup>  
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 <sup>138,139</sup>.  
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

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