

EXTERNAL REVIEW DRAFT

NIOSH Hazard Review

Wildland Fire Smoke Exposure Among Farmworkers and Other Outdoor Workers



**U.S. Centers for Disease
Control and Prevention**
National Institute for
Occupational Safety and Health

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2 **Cover Images**

3 Migrant farmworkers harvest crops in a thick cloud of wildland fire smoke.
4 Photo by United Farm Workers.

5 Emergency response workers stand and wait as traffic is forced to stop because
6 smoke from a wildfire has enveloped the highway. Photo by RapidEye/Getty
7 Images.

8 A forest ranger walks toward wildland fire smoke. Photo by Chuck Schug
9 Photography/Getty Images.

10 A tractor-trailer is driven through the smoke from a wildland fire. Photo by
11 Vitpho.

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NIOSH HAZARD REVIEW

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**Wildland Fire Smoke
Exposure Among
Farmworkers and Other
Outdoor Workers**

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DEPARTMENT OF HEALTH AND HUMAN SERVICES
Centers for Disease Control and Prevention
National Institute for Occupational Safety and Health

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16

Foreword

Smoke from wildland fires has become an increasingly widespread and potentially serious threat to public health in recent years. As these fires become more frequent and intense, the impact on workers is a growing concern for the National Institute for Occupational Safety and Health (NIOSH) and its partners. Government agencies, academia, industry, labor organizations, and worker advocates partner with NIOSH to research and promote occupational safety and health.

Wildland fire smoke contains several potentially harmful components. This hazard review focuses on particulate matter as a major cause of adverse health among exposed workers and the primary indicator of occupational exposure to the complex mixture comprising wildland fire smoke. Research has provided evidence of a causal relationship between an exposure to particulate matter and adverse health effects, including cardiorespiratory effects and cancer.

Farmworkers and other outdoor workers are likely to be exposed to wildland fire smoke. They often spend long hours in the fields or on job sites when wildland fire smoke is present, increasing their risk of adverse health effects from exposure to harmful particulate matter and chemicals.

This NIOSH Hazard Review, *Wildland Fire Smoke Exposure Among Farmworkers and Other Outdoor Workers*, is a valuable resource for anyone concerned with the occupational health effects of wildland fire smoke. It provides an extensive review of the scientific literature, assesses potential health effects, and offers controls for reducing occupational exposures and their effects. This hazard review document aims to mitigate risks and protect the health and well-being of these essential outdoor workers.

John Howard, M.D.
Director,
National Institute for Occupational Safety and Health

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Preface

Background

When the U.S. Congress passed the Occupational Safety and Health Act (OSH Act) of 1970 (Public Law 91-596), it established the National Institute for Occupational Safety and Health (NIOSH). The OSH Act was created to assure safe and healthful working conditions for working individuals, and to this end it assigned NIOSH responsibilities for assisting and encouraging states in their efforts to assure safe and healthful working conditions; and providing for research, information, education, and training in the field of occupational safety (Public Law 91-596).

Purpose

The purpose of a NIOSH Hazard Review document is to provide a comprehensive review of the available scientific information on the potential hazards associated with a specific chemical or group of chemicals, substance, or process in the workplace. These documents are authoritative recommendations and are considered occupational safety and health guidance. They are intended to help other government agencies, partners, employers, safety professionals, labor, and other interested parties understand the risks involved and how to implement appropriate measures to protect workers' health and safety.

A NIOSH Hazard Review includes information on recommended exposure control measures following the hierarchy of controls model. These control strategies may include information and guidance on elimination, substitution, engineering control measures, administrative controls, personal protective equipment (PPE), and other relevant strategies to help mitigate occupational hazards effectively. A NIOSH Hazard Review also identifies critical research needs about a hazard, exposure assessment, and effective control strategies.

Document Development Process

NIOSH developed this hazard review using the NIOSH guidance development process, which includes these key elements:

1. Engagement with external partners, interested parties, and working partners through the following:
 - a. Request for Information (RFI) published in the *Federal Register*.
 - b. Consideration of RFI public comments in the draft document.
 - c. Public comment period of 60 days for the draft document.
 - d. Interactions with NIOSH and National Occupational Research Agenda (NORA) partners and interested parties.
2. Development of scientific content by internal NIOSH subject matter experts.
3. Peer review of the draft document by external subject matter experts without conflict of interest.

- 1 4. Consideration of peer and public comments in the final draft of the document.
- 2 5. Targeted dissemination plan to partners and interested parties, including key
- 3 employer and worker groups.

4 The guidance document development process for this hazard review followed these steps:

- 5 1. **NIOSH leadership preapproval to develop the hazard review document.** A
6 proposal for the planned document was reviewed and approved by NIOSH
7 leadership. The document proposal described the purpose and intended audience of
8 a hazard review document; the steps NIOSH will undertake to ensure public and
9 interested party engagement; and the peer review, public comment, and document
10 clearance processes.
- 11 2. **Request for Information; first external engagement.** NIOSH published an RFI in
12 the *Federal Register* on March 14, 2024.¹ The RFI invited the public to provide
13 information about approaches to assess and control the hazards of wildland fire
14 smoke to outdoor workers. NIOSH raised awareness of and disseminated the RFI by
15 reaching out to partners, other federal agencies, interested parties, NORA sector
16 councils, and NIOSH-funded centers, including Education and Research Centers,
17 Centers for Agricultural Safety and Health, and Centers of Excellence for Total
18 Worker Health. The 60-day public comment period ended May 13, 2024. NIOSH
19 received 10 sets of comments from individuals, industry and worker associations,
20 and a state government agency. These public comments are available for review at
21 [regulations.gov](https://www.regulations.gov), through the *Federal Register*.¹ The information received during the
22 public comment period was considered by NIOSH in the development of the draft
23 document.
- 24 3. **Development of the Population, Exposure, Comparator, and Outcomes (PECO)**
25 **statement.** A team of NIOSH subject matter experts created an organizing
26 statement, the PECO statement, which helped guide the assessment of human health
27 effects.² This statement focused on the impacted population, the exposure of
28 interest, the appropriate comparators, and the health outcomes most relevant to the
29 hazard. The PECO statement was then used to develop a literature search of relevant
30 evidence streams, conduct hazard identification, and construct the document
31 outline and table of contents.

1 NIOSH [2024]. National Institute for Occupational Safety and Health; Outdoor workers exposed to wildland fire smoke; Request for information. Fed Regist 89(51):18638. <https://www.federalregister.gov/documents/2024/03/14/2024-05403/national-institute-for-occupational-safety-and-health-outdoor-workers-exposed-to-wildland-fire-smoke>.

2 Morgan RL, Whaley P, Thayer KA, Schünemann HJ [2018]. Identifying the PECO: a framework for formulating good questions to explore the association of environmental and other exposures with health outcomes. Environ Int 121:1027–1031, <https://doi.org/10.1016/j.envint.2018.07.015>.

- 1 4. **Identification of existing, relevant authoritative reviews.** NIOSH subject matter
2 experts and technical information specialists searched the available scientific
3 literature for published systematic reviews and authoritative guidance that might be
4 considered in this hazard review document. See Chapter 3 for more detailed
5 information.
- 6 5. **NIOSH scoping literature review.** NIOSH conducted a scoping review of the
7 relevant epidemiologic literature on human health effects from wildland fire smoke
8 to supplement evidence presented in existing authoritative reviews of causal
9 relationships between similar or analogous exposures (e.g., particulate exposure in
10 ambient air pollution) and human health effects. See Chapter 3 for more detailed
11 information.
- 12 6. **Synthesis of evidence on health effects.** In examining the likelihood of causal
13 associations between wildland-fire smoke and adverse human health effects, NIOSH
14 relied, in part, on previous conclusions from comprehensive weight-of-evidence
15 assessments that were performed by several authoritative bodies. Given that
16 literature supporting these authoritative reviews lacked information specific to
17 wildland fires, NIOSH examined the coherence of their conclusions with findings
18 from recent literature identified in its scoping review. NIOSH used the totality of this
19 information to form its conclusions regarding the potential for exposure-related
20 health effects in working populations, as identified in the PECO statement. See
21 Chapter 3 for more detailed information.
- 22 7. **Development of recommendations to protect workers.** After NIOSH reached
23 conclusions about the association of the exposure with the health effects of interest
24 in the population, NIOSH evaluated the potential exposure pathways and made
25 recommendations and suggested considerations for mitigating exposures to reduce
26 or eliminate adverse health effects in workers. NIOSH recommended a hierarchy of
27 controls approach to eliminate or control worker exposures (see Chapter 5).^{3,4}
28 NIOSH also identified significant research gaps to encourage development of
29 scientific information to further refine worker protection recommendations (see
30 Chapter 6).
- 31 8. **External peer review and external engagement.** This external review draft of the
32 hazard review will undergo external peer review and be made available for public
33 comment. Additional details on the peer review and public comment mechanisms
34 are outlined below. This external review draft of the document is at this stage of the
35 guidance development process.

3 NIOSH [1990]. NIOSH testimony on the Occupational Safety and Health Administration’s proposed rule on health standards: methods of compliance, May 30, 1990, OSHA Docket No. H-160. NIOSH policy statements. Cincinnati, OH: U.S. Department of Health and Human Services, Centers for Disease Control, National Institute for Occupational Safety and Health.

4 NIOSH [2024]. Hierarchy of controls. In: Workplace safety and health topics.
<https://www.cdc.gov/niosh/hierarchy-of-controls/about/index.html>. Date accessed: June 6, 2024.

1 **Public Comment and Peer Review Comment Process**

2 Public comments and expert peer review are essential to strengthen NIOSH authoritative
3 recommendations.

4 **Public comment.** NIOSH announced the availability of the draft hazard review by
5 publication in the *Federal Register*. The *Federal Register* notice (FRN) invited the public and
6 interested parties to provide comments on the draft document. The charge to the peer
7 reviewers was also published in the FRN so that the public could see what NIOSH asked the
8 peer reviewers to consider in their review. The comment period is 60 days. The draft
9 document and FRN were posted on www.regulations.gov. Instructions for submitting
10 comments on the draft document are contained in the FRN. During this time, NIOSH will
11 inform other federal agencies and interested parties of the availability of the draft
12 document for comment.

13 After the public comment period closes, NIOSH will review and consider all comments
14 received. Comments and information received will be used to revise the draft document.
15 NIOSH subject matter experts will prepare a response to the public comments and make it
16 available in the public docket along with the final document at www.regulations.gov, so the
17 public will be able to see how NIOSH used the information received.

18 **Peer review.** NIOSH will also conduct an external expert peer review of its draft document.
19 The peer review will be managed by the Division of Science Integration and will be
20 documented by the NIOSH Docket Office. Independent external experts will be identified
21 with key areas of expertise for the document review. The reviews will be conducted as
22 individual independent letter reviews. Peer review consensus will not be sought since it is
23 common to have peer reviewers with diverse opinions on a hazard review document. In
24 order to participate in this review, the experts will be required to document that they are
25 free from financial conflicts of interest. NIOSH will provide a charge for the reviewers to
26 focus their review on specific scientific questions of interest.

27 Once the peer reviewers submit their reviews to the NIOSH Docket Office, NIOSH will
28 consider the comments in finalizing the draft document. NIOSH will develop a response to
29 the peer reviewers' comments. Both the peer reviews and the NIOSH responses will be
30 made available in the public docket at www.regulations.gov along with the final document.

31 The following steps will take place after the peer review and public comment period.

32 **Publication of the Final Hazard Review**

33 After NIOSH considers all comments from peer reviewers and the public and revises the
34 hazard review accordingly, the final document will be prepared for publication. Before final
35 publication, the document will undergo editorial review to ensure compliance with the
36 plain language standard and Section 508 of the Rehabilitation Act of 1973, to ensure those
37 with disabilities can access the information in the document. The revised draft will undergo
38 review and clearance at NIOSH and the Centers for Disease Control and Prevention (CDC).
39 After NIOSH and CDC approve the final document, the hazard review will be published on
40 the NIOSH website and made available on [regulations.gov](http://www.regulations.gov).

1 **Dissemination of the Hazard Review**

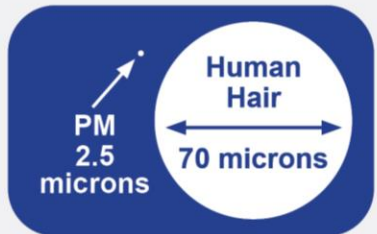
2 The hazard review will be widely disseminated through NIOSH health communication
3 channels, including partners, other federal agencies, interested parties, NORA sector
4 councils, and NIOSH-funded centers, including Education and Research Centers, Centers for
5 Agricultural Safety and Health, and Centers of Excellence for Total Worker Health®. NIOSH
6 health communicators will develop customized dissemination plans for targeted audiences
7 of impacted workers and employers, specific industrial sectors of interest, and occupational
8 safety and health professionals.
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PLAIN LANGUAGE SUMMARY

Wildland Fire Smoke Exposure Among Farmworkers and Other Outdoor Workers



PM_{2.5} stands for particulate matter 2.5 microns in diameter or smaller. A human hair is about 28 times wider.

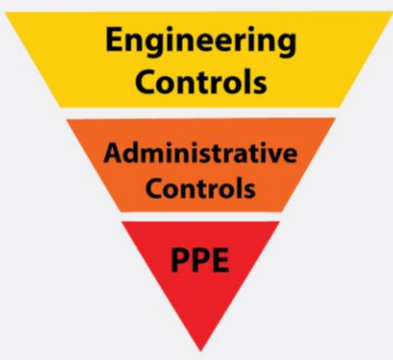
What is in wildland fire smoke that causes health concerns?

- Wildland fire smoke is a complex mixture of many parts, with some that are toxic.
- Gases and vapors from the fires spread quickly away as the smoke travels. Tiny particles, called particulate matter, stay in the air and can be breathed deep into the lungs.
- The Environmental Protection Agency (EPA) has concluded that PM_{2.5}—a major pollutant in wildland fire smoke—can cause harm to human health.



What are the health effects of PM_{2.5} in wildland fire smoke?

- Scientific studies suggest PM_{2.5} can cause heart, lung, and other health problems, as well as death.
- Studies show strong evidence for health effects from short-term (daily) exposures, but more research is needed on long-term effects and special ways workers can be affected.



Recommendations and conclusions

- One helpful scale for evaluating the level of potential health risk from PM_{2.5} is EPA's Air Quality Index (AQI).
- [Exposure controls](#) should be followed in this order: engineering controls for best protection, followed by administrative controls, then personal protective equipment (PPE).
- NIOSH recommends reducing outdoor workers' exposure based on the EPA's Air Quality Index for PM_{2.5}, with extra protection for workers who are more sensitive.

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Executive Summary

Over the past few decades, wildland fires have become more intense and severe due to factors such as natural fuel buildup, extreme weather events, and the expansion of urban areas into wildlands. Wildland fires include wildfires, prescribed fires, and fires occurring at the wildland-urban interface. These fires can produce large smoke plumes that can travel long distances and cause adverse health effects. Outdoor workers are at risk of adverse health effects from their exposures to wildland fire smoke because they spend hours in outdoor work environments where traditional exposure control measures are difficult to put in place. This hazard review evaluates the health impact of wildland fire smoke among farmworkers and other outdoor workers and provides workplace control recommendations to reduce exposures.

Outdoor Workers and Wildland Fire Smoke Exposure

Outdoor workers are people who work outside a physical structure in rural, suburban, or urban areas. People who work on farms represent approximately 800,000 to 1.5 million workers and are a large proportion of outdoor workers affected by wildland fire smoke. Many other outdoor workers are also potentially exposed, such as those working in construction, transportation, installation, maintenance and repair occupations, fishing, and forestry occupations. Chapter 2 discusses outdoor workers in more detail. NIOSH recognizes that wildland firefighters are also at risk of exposure-related adverse health effects; however, this hazard review does not focus on the hazards associated with smoke exposure during wildland firefighting. The decision to focus on other outdoor working populations was based on important differences in exposure potential, training, medical surveillance, and risk management options available to wildland firefighters compared with other working populations.

Wildland fire smoke is a complex mixture of gas and particulate chemicals that can chemically transform as it moves through the air. This complex mixture includes chemicals such as carbon monoxide, ozone, aldehydes, benzene, polycyclic aromatic hydrocarbons, other hazardous air pollutants, and particulate matter (PM). As the smoke travels downwind of the fire, exposures to these air pollutants decrease as they are diluted in clean air and the larger-sized PM are deposited onto surfaces. Smaller particulates, such as those with an aerodynamic diameter of 2.5 microns or less (PM_{2.5}), can migrate long distances with the smoke plume. PM_{2.5} has been associated with various adverse health outcomes and is often used as a surrogate for wildfire smoke exposure in research and environmental monitoring. NIOSH regards PM_{2.5} as the primary hazard of concern with respect to exposure related to health effects.

Government Resources and Efforts

The U.S. Environmental Protection Agency (EPA) conducted extensive reviews of the PM_{2.5} scientific evidence to provide the basis for its National Ambient Air Quality Standards. The EPA also established the Air Quality Index (AQI), a color-coded tool for communicating air quality to the public for common pollutants including particulate matter. Other federal

1 agencies, including the Department of the Interior, U.S. Department of Agriculture (USDA)
2 Forest Service, and the National Center for Environmental Health within the CDC, have also
3 conducted research, developed guidance, tools, and made recommendations to protect
4 workers and the public from safety and health hazards associated with wildland fire smoke.

5 NIOSH has conducted and supported wildland smoke research in farmworker populations
6 to assess the combined burden of heat and smoke and to test affordable air quality sampling
7 in rural agricultural areas. NIOSH has also conducted numerous studies investigating
8 woodsmoke and PM_{2.5} exposures, assessment methods, and health effects, with findings
9 relevant to all outdoor workers. California, Oregon, and Washington have enacted rules to
10 protect outdoor workers from wildfire smoke, with actions required of employers linked to
11 specific values of the AQI for PM_{2.5}. However, the existing federal occupational exposure
12 limit for particulates not otherwise regulated may not be sufficient to protect outdoor
13 workers exposed frequently or for extended periods of time.

14 **Hazard Review**

15 This hazard review analyzed three streams of evidence to evaluate the human health effects
16 associated with exposure to wildland fire smoke. These include (1) the evaluation of
17 previous authoritative reviews, (2) a scoping review of recent epidemiological studies, and
18 (3) a synthesis of toxicological literature.

19 Previous authoritative reviews by United States and international agencies have evaluated
20 health effects associated with exposure to particulate matter, outdoor air pollution, and
21 firefighting, which are relevant to workers exposed to wildland fire smoke. The
22 authoritative reviews provided strong evidence for causal relationships between PM_{2.5}
23 exposures and cardiorespiratory effects, cancer, and nonaccidental mortality; and lesser
24 evidence for other health effects, such as metabolic, nervous system, reproductive, and
25 developmental effects. However, these reviews focused mostly on hazards from ambient air
26 pollution and provided limited evidence from studies of working populations.

27 A scoping review of the literature published from 2017–2024 was conducted to identify and
28 evaluate recent epidemiological studies that directly examined associations between
29 wildland fire smoke exposure and adverse physical health effects. This scoping review
30 expanded the evidence base obtained from the authoritative reviews and reinforced the
31 analogy between ambient air pollution and wildland fire smoke. The scoping review
32 identified supporting evidence on previously described exposure-related health conditions,
33 as well as new evidence for an association between wildland fire emissions and increased
34 infectious disease. Although rapid growth in the scientific literature involving wildland fire
35 hazards has been observed in recent years, there remains a notable absence of health effect
36 studies examining exposed workers.

37 In vitro and in vivo toxicology studies supported the streams of evidence in the
38 authoritative document review and scoping review. These toxicological studies
39 demonstrated several mechanisms associating adverse health effects with exposure to
40 wildland fire smoke. These included cardiorespiratory effects, oxidative stress,
41 inflammation, autonomic nervous system imbalance, translocation of smoke elements into

1 the bloodstream, and a change in circulating mediators consistent with the effects of an
2 inhalation exposure.

3 **Exposure Assessment and Control Strategies**

4 An exposure assessment strategy is essential to obtain the information needed to minimize
5 exposure to wildland fire smoke and the resulting health effects. A tiered decision-making
6 approach can be used to evaluate wildland fire smoke exposure scenarios and implement
7 mitigation strategies. Tier 1 assessments use various data sources from models, satellite
8 sensors, community-based monitors, and low-cost sensors, but they can lack timeliness and
9 confidence. Tier 2 assessments can improve confidence by using direct-reading instruments
10 but require specialized equipment and personnel. Tier 3 assessments offer the most
11 confidence by using well-established sampling and analytical methods to quantify smoke
12 constituents, but they take more time to collect, which can delay the decision-making
13 process. PM_{2.5} concentration data are then defined into exposure control categories that
14 relate to health effects and inform which control measures should be considered. Quality
15 control measures should be defined and are critical considerations due to the high
16 variability of outdoor workplace conditions, as well as uncertainties from multiple sources
17 of exposure.

18 **Recommendations**

19 After the exposure assessment has been performed, workplace controls should be selected
20 to minimize worker exposure. The hierarchy of controls forms the basis of NIOSH worker
21 protection recommendations to reduce wildland fire smoke exposure. See Table 5–1 in
22 Chapter 5 for NIOSH recommendations. Overall, the use of engineering and administrative
23 solutions is more effective and preferable than personal protective equipment, according to
24 the hierarchy of controls paradigm. The hierarchy of controls should be incorporated into
25 an overall workplace safety and health program for outdoor workers, according to good
26 industrial hygiene practice:

- 27 • Engineering control solutions may include providing filtered air to reduce workers'
28 exposure. Filtered air could be provided in enclosed spaces such as temporary or
29 permanent structures and vehicles.
- 30 • Administrative controls begin with preparation for wildland fire smoke events along
31 with worker training and education. Exposures can be managed with a variety of
32 administrative control approaches, including worker relocation, reduction of shift
33 length, rotation of workers, work-rest cycles, and a reduction of work intensity.
- 34 • NIOSH Approved[®] personal protective equipment⁵, such as N95[®] respirators can be
35 effective when selected and used properly as part of a complete respiratory

5. N95 and NIOSH Approved are certification marks of the U.S. Department of Health and Human Services (HHS) registered in the United States and several international jurisdictions.

1 protection program. These respirators can protect against inhalation hazards from
2 wildland fire smoke.

3 **Health Equity**

4 Workers from economically and socially marginalized communities may face
5 disproportionate impacts from wildfires due to the location of their work and
6 characteristics of their jobs. These health inequities in the workplace can be influenced by
7 disadvantages related to occupational segregation, social hierarchies, differential statutory
8 protections, job characteristics, and other compounding factors. Populations placed at
9 increased risk include workers who are immigrants, racial and ethnic minorities, women,
10 people with low incomes, and those who are incarcerated.

11 **Medical Surveillance and Monitoring**

12 All workers should be allowed to seek medical care if they experience signs or symptoms of
13 injury or illness due to wildland fire smoke exposure. Qualified healthcare professionals
14 should understand the populations who are at greater risk of adverse health effects from
15 wildland fire smoke exposure. While not broadly recommended due to insufficient available
16 research, there are populations where medical monitoring or surveillance may best serve as
17 a tool in a comprehensive wildland fire smoke risk management plan. These situations
18 include identification of at-risk populations that may benefit from layered occupational
19 controls or as part of an outdoor worker wildland fire smoke emergency response plan.

20 **Future Research Needs**

21 Current knowledge on exposure-related health effects stems mostly from expansive
22 literature on ambient air pollution and growing literature on populations living near
23 wildland fires. However, there is a general lack of etiologic research on workers who are
24 directly exposed to wildland fire smoke, other than sparse information on small groups of
25 wildland firefighters. Research specific to the affected working populations (e.g., farm,
26 forestry, and construction workers) could improve external validity. Evidence strongly
27 supports the need to reduce worker exposures to wildland fire smoke and protect workers
28 from adverse health effects. Much more research is needed to improve the understanding of
29 occupational risk from wildland fire smoke and more effective control measures that will
30 protect farmworkers and other outdoor workers.

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Abbreviations

1		
2	AIHA	American Industrial Hygiene Association
3	ALI	air liquid interface
4	AM	arithmetic mean
5	AQI	Air Quality Index
6	AQS	air quality system
7	BenMAP-CE	Environmental Benefits Mapping and Analysis Program
8	BLS	Bureau of Labor Statistics
9	CADR	Clean Air Delivery Rate
10	CDC	U.S. Centers for Disease Control and Prevention
11	CeVD	cerebrovascular disease
12	CO	carbon monoxide
13	CO ₂	carbon dioxide
14	COPD	chronic obstructive pulmonary disease
15	CPS	Current Population Survey
16	CRF	concentration-response function
17	CVD	cardiovascular disease
18	CSV	comma separated value
19	d _{ae}	aerodynamic equivalent diameter
20	DHS	U.S. Department of Homeland Security
21	DNA	deoxyribonucleic acid
22	DOI	U.S. Department of the Interior
23	DRI	direct reading instrument
24	EC	elemental carbon
25	ECC	exposure control categories
26	EPA	U.S. Environmental Protection Agency
27	ERHMS	Emergency Responder Health Monitoring and Surveillance
28	FEM	federal equivalent method
29	FEV	forced expiratory volume
30	FEV ₁	forced expiratory volume in one second

1	FFR	filtering facepiece respirator
2	FRM	federal reference method
3	FRN	Federal Register Notice
4	FVC	forced vital capacity
5	GAO	U.S. Government Accountability Office
6	GM	geometric mean
7	GSD	geometric standard deviation
8	HAP	hazardous air pollutant
9	HCl	hydrochloric acid
10	HCN	hydrogen cyanide
11	HEPA	high efficiency particulate air
12	HHS	U.S. Department of Health and Human Services
13	HIA	health impact assessment
14	HNCO	isocyanic acid
15	HVAC	heating, ventilation, and air conditioning
16	IARC	International Agency for Research on Cancer
17	ICD	International Classification of Diseases
18	IHD	ischemic heart disease
19	ISA	Integrated Science Assessment
20	LOD	limit of detection
21	MERV	minimum efficiency reporting values
22	MI	myocardial infarction
23	µm	micrometer
24	MSHA	Mine Safety and Health Administration
25	MTBS	Monitoring Trends in Burn Severity
26	NAAQS	National Ambient Air Quality Standards
27	NASEM	National Academies of Science, Engineering, and Medicine
28	NCEH	National Center for Environmental Health
29	NIEHS	National Institute of Environmental Health Sciences
30	NIFC	National Interagency Fire Center
31	NIOSH	National Institute for Occupational Safety and Health

1	NMAM	NIOSH Manual of Analytical Methods
2	NO ₂	nitrogen dioxide
3	NOAA	National Oceanic and Atmospheric Administration
4	NORA	National Occupational Research Agenda
5	NPRM	Notice of Proposed Rulemaking
6	NWCG	National Wildfire Coordinating Group
7	O*NET	Occupational Information Network
8	O ₃	ozone
9	OC	organic carbon
10	OEL	occupational exposure limit
11	OEWS	Occupational Employment and Wage Statistics
12	OSHA	Occupational Safety and Health Administration
13	OSH	occupational safety and health
14	Ox-GS	oxidized guanine species
15	PAD	peripheral artery disease
16	PCDD	polychlorinated dibenzo-p-dioxins
17	PCDF	polychlorinated dibenzofurans
18	PAH	polycyclic aromatic hydrocarbon
19	PAN	peroxacetyl nitrate
20	PBZ	personal breathing zone
21	PCB	polychlorinated biphenyl
22	PECO	Population, Exposure, Comparator, and Outcomes
23	PEL	permissible exposures limit
24	PM	particulate matter
25	PM _{2.5}	particulate matter with a diameter of 2.5 microns or less
26	PM ₁₀	particulate matter with a diameter of 10 microns or less
27	PNOR	particulate not otherwise regulated
28	PPE	personal protective equipment
29	PPN	propionyl peroxy nitrite
30	PSI	pollutant standard index
31	PVC	polyvinyl chloride

1	REL	recommended exposure limit
2	RFI	request for information
3	RNA	ribonucleic acid
4	RPP	respiratory protection program
5	SDOH	social determinants of health
6	SEG	similar exposure group
7	SO ₂	sulfur dioxide
8	SOC	Standard Occupational Classification
9	SVOC	semi-volatile organic compounds
10	TIA	transient ischemic attack
11	TSP	total suspended particulates
12	TWA	time-weighted average
13	UFP	ultrafine particles
14	USDA	U.S. Department of Agriculture
15	VOC	volatile organic compound
16	WAC	Washington Administrative Code
17	WFLC	Wildland Fire Leadership Council
18	WUI	wildland urban interface
19	X95	95 th percentile

Glossary

Agricultural worker — An individual who performs various tasks related to cultivating, harvesting, and producing crops or livestock.

Conformity assessment — A demonstration that specified requirements relating to a product, process, system, person, or body are fulfilled.⁶

Farmer — A person who owns or manages a farm.

Farmworker — A person who is paid wages to work on a farm.

Foreign-born — An individual who is not a U.S. citizen at birth, including those who become U.S. citizens through naturalization.⁷

Health equity — The state in which everyone has a fair and just opportunity to attain their highest level of health.⁸

Migrant farmworker — An individual who is absent from a permanent place of residence for the purpose of seeking remunerated employment in agricultural work.⁹

Occupational health inequities — Avoidable differences in work-related disease incidence, mental illness, or morbidity and mortality that are closely linked with social, economic, and/or environmental disadvantage, such as work arrangements (e.g., contingent work), socio-demographic characteristics (e.g., age, sex, race, and class), and organizational factors (e.g., business size).¹⁰

Outdoor worker — A person working outside a physical structure in a rural, urban, or suburban area.

Particulate matter — A mixture of liquid and solid particles made up of a variety of substances and across a wide distribution of sizes.

6 ISO/IEC 17000 [2020]. Conformity assessment—Vocabulary and general principles. Geneva: International Organization for Standardization.

7 U.S. Census Bureau [2024]. Foreign-born. Washington, DC: U.S. Census Bureau, <https://www.census.gov/topics/population/foreign-born.html>.

8 CDC [2024]. Health equity. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, <https://www.cdc.gov/healthequity/index.html>.

9 Migrant Clinicians Network [no date]. The migrant/seasonal farmworker. Austin, TX: Migrant Clinicians Network, <https://www.migrantclinician.org/explore-migration/migrant-seasonal-farmworker.html>. Date accessed: April 5, 2024.

10 NIOSH [2019]. Occupational health equity. Cincinnati, OH: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, <https://www.cdc.gov/niosh/research-programs/portfolio/ohe.html>.

1 **Polycyclic aromatic hydrocarbons** — Volatile and semi-volatile aromatic compounds that
2 can be released from combustion of vegetative biomass or from man-made materials in
3 buildings and vehicles (e.g., insulation, upholstery, carpet, plastics).¹¹

4 **Populations at greater risk of adverse health effects from wildfire smoke exposure** —
5 Refers to people with asthma and other respiratory diseases, people with cardiovascular
6 disease, children younger than 18 years of age, pregnant people, older adults, people of low
7 socio-economic status, and outdoor workers.¹²

8 **Prescribed fire** — A planned fire intentionally ignited to meet management objectives.

9 **Seasonal farmworker** — An individual who is employed in temporary farmwork but does
10 not move from their permanent residence to seek farmwork.⁹

11 **Structural disadvantage** — The personal, interpersonal, institutional, and systemic
12 factors—such as, racism, sexism, classism, able-ism, xenophobia, and homophobia—that
13 make those identities salient to the fair distribution of health opportunities and outcomes.¹³

14 **Take-home exposures** — Exposures that occur when a worker inadvertently carries
15 contaminants from work via their clothes, skin, etc., into the home or other shared
16 environments.¹⁴

17 **Wildfire** — An unplanned fire caused by lightning or other natural causes, by human
18 ignitions (or arson), or by an escaped prescribed fire.

19 **Wildland fire** — An overarching term describing any non-structure fire that occurs in
20 vegetation and natural fuels. Wildland fire encompasses both prescribed fire and wildfire.

21 **Wildland urban interface (WUI)** — The zone where natural areas meet human-developed
22 lands, often increasing wildfire risks to homes and infrastructure.

11 NAS [2022]. The chemistry of fires at the wildland-urban interface. Washington, DC: National Academies of Sciences, Engineering, and Medicine. The National Academies Press, <https://doi.org/10.17226/26460>.

12 EPA [2024] Which populations experience greater risks of adverse health effects resulting from wildfire smoke exposure? Washington, DC: U.S. Environmental Protection Agency, <https://www.epa.gov/wildfire-smoke-course/which-populations-experience-greater-risks-adverse-health-effects-resulting#adults>.

13 National Academies of Sciences, Engineering, and Medicine [2017]. The root causes of health inequity. In: Weinstein JN, Geller A, Negussie Y, Baciu A, eds. Communities in action: pathways to health equity. Washington, DC: The National Academies Press, <https://nap.nationalacademies.org/read/24624/chapter/5>.

14 NIOSH [2024]. About take-home exposures. Cincinnati, OH: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, <https://www.cdc.gov/niosh/reproductive-health/about/take-home.html>.

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Chapter 1: Introduction

Key Chapter Takeaways

- Wildland fires have become larger and more intense, resulting in increased outdoor worker exposure to wildland fire smoke.
- This smoke contains PM_{2.5}, which has been linked to various adverse health effects as recognized by authoritative agencies and organizations.
- Current federal occupational exposure limits may not sufficiently protect outdoor workers who are frequently or extensively exposed to wildland fire smoke.
- This hazard review reports the latest scientific information and research on the health effects of exposure to wildland fire smoke and provides recommendations to protect outdoor workers.

1.1 Purpose and Scope

This National Institute for Occupational Safety and Health (NIOSH) Hazard Review, *Wildland Fire Smoke Exposure Among Farmworkers and Other Outdoor Workers*, evaluates the effects of exposure to wildland fire smoke among farmworkers and other outdoor workers. This is in response to a Department of Health and Human Services (HHS) initiative entitled “Protecting Farmworkers from Extreme Heat and Wildfire Smoke.” It provides a concise review, critical analysis, and evaluation of scientific evidence related to the impact of occupational exposure to wildland fire smoke among farmworkers and other outdoor workers. Heat exposures are not addressed within this document, as NIOSH has previously addressed exposures to heat in outdoor workers [NIOSH 2016].

This hazard review focuses on particulate matter as the primary hazard of concern with respect to exposure related to health effects. There is uncertainty in the level of worker protection from particulate hazards using current U.S. federal standards. The Occupational

1 Safety and Health Administration (OSHA) has a permissible exposure limit (PEL) for
2 particulates not otherwise regulated of 15 mg/m³ as an 8-hour time-weighted average
3 (TWA) (total dust) and 5 mg/m³ 8-hour time-weighted average (TWA) (respirable dust)
4 [OSHA, no date, b]. OSHA’s commonly acceptable definition of “respirable dust” is particles
5 with an aerodynamic diameter of 4 μm collected at 50% efficiency [OSHA 2023]. However,
6 OSHA has indicated it recognizes “that many of its PELs are outdated and inadequate for
7 ensuring protection of worker health” [OSHA, no date, b]. NIOSH does not have a
8 recommended exposure limit (REL) for particulates. In 1988, NIOSH provided comments to
9 OSHA regarding the “Proposed Rule on Air Contaminants” (29 CFR 1910, Docket No. H-020)
10 and determined that there was inadequate evidence to support OSHA’s proposed PEL of 10
11 mg/m³ (total dust) [NIOSH 1988, 2018b]. In light of this history, NIOSH evaluated the
12 scientific literature and previous authoritative guidance critical for understanding adverse
13 health effects among outdoor workers exposed to wildland fire smoke while regarding PM_{2.5}
14 as the primary hazard of concern. Research and guidance on related adverse health effects,
15 such as acute and chronic cardiorespiratory diseases and other conditions, are discussed in
16 Chapter 3.

17 The HHS initiative specified wildfire exposures to farmworkers, however, because wildfire
18 smoke exposures are likely similar for other outdoor workers, the focus of this document is
19 on wildfire smoke exposures to farmworkers and other outdoor workers. The scope of this
20 document does not focus on hazards associated with smoke exposure to wildland
21 firefighters and other first responders at the fireline. The characteristics of those exposures
22 likely differ in intensity, duration, and complexity compared with other outdoor workers
23 away from the fire line. Additionally, other important differences include training, medical
24 surveillance, and risk management options that are available to wildland firefighters that
25 may not be available to other outdoor working populations. NIOSH continues efforts that
26 focus on research related to smoke exposures to wildland and structural firefighters.
27 Though not a focus of this document, information on wildland firefighters’ exposure to and
28 health effects from wildland fire smoke is occasionally referenced for context.

29 This chapter introduces key topics related to outdoor workers’ exposure to wildland fire
30 smoke exposure. This includes background information on wildland fire smoke, the history
31 of NIOSH activities on agricultural workers, all outdoor workers, respiratory protection, and
32 the history of related activities at other agencies.

1.2 Background on Wildland Fire Smoke

Although fire helps maintain the health and resilience of forest and grassland ecosystems, the devastating impacts of wildland fires on communities and individuals, such as the loss of life and destruction of property, are undeniable [Pyne 2015; Wildland Fire Mitigation and Management Commission 2023]. Adding to these direct adverse impacts, smoke from wildland fires can also pose great risks to public health, as it is not limited to the vicinity of the fire but can spread over vast distances, traveling hundreds or even thousands of miles [EPA 2021a; Moeltner et al. 2013]. A variety of factors including changes in land management, land use, and extreme weather events (e.g., high temperatures, drought, low humidity, and strong winds) have increased the frequency of severe wildfires, resulting in greater risks to human safety and health [CBO 2022; EPA 2021b]. In addition, over the past 50 years, the recognition and understanding of the relationship between exposure to air pollution and adverse health outcomes have steadily grown—as has concern over the health risks that exposure to wildland fire smoke poses both to the public and to outdoor workers [EPA 2019a; 2021a; Navarro 2020]. The term wildland fire encompasses wildfire (i.e., unplanned non-structure fire with natural fuels), prescribed fire, and fire occurring in the wildland urban interface (WUI) [NIOSH 2024b; NPS 2022]. Outdoor workers may be exposed to smoke from all types of wildland fire, each of which pose potential health risks that should be assessed and characterized [EPA 2019b; EPA 2021b].

Charged with preventing and combating wildfires, wildland firefighters are employed by federal agencies—primarily the U.S. Department of Agriculture (USDA) Forest Service and the U.S. Department of the Interior—as well as states, counties, Tribes, and private for-profit organizations [GAO 2022; USFA 2023]. These crews confront serious health and safety hazards in the normal course of their duties that put them at risk of injury, illness, and death [Britton et al. 2013; Butler et al. 2017; Navarro 2020; NWCG 2017; NWCG 2022]. Working near fires, wildland firefighters are at risk of exposure to elevated concentrations of respirable particulate matter, carbon monoxide, aldehydes, and polycyclic aromatic hydrocarbons. A coordinated effort among the agencies and organizations listed above seeks to better characterize the health risks of wildland firefighters and implement strategies to protect their health and well-being [Navarro et al. 2022; Wildland Fire Mitigation and Management Commission 2023].

Other outdoor workers, along with wildland firefighters, may be exposed to smoke from wildfires. Chapter 2 discusses outdoor workers in more detail. The nature of this work often requires employees to spend entire shifts outdoors, increasing their health risk during periods with elevated concentrations of air pollution [Sacks et al. 2011]. Unlike wildland firefighters, who must pass stringent medical examinations and meet fitness standards as a condition of work, other outdoor workers may have a higher prevalence of preexisting health conditions that would make them more susceptible to the health effects of exposure to wildland fire smoke [NIOSH 2020; NWCG 2015].

This chapter provides information about wildland fire and smoke from wildland fires to demonstrate the increasing potential for smoke exposure to farmworkers and other

1 outdoor workers. Information is provided about the history of wildfire management in the
2 United States; the increasing size, severity, and duration of wildfires; the increasing amount
3 of wildland urban interface, and a characterization of risks from air pollution. These factors
4 play important roles in affecting outdoor workers' exposure to wildland fire smoke and in
5 our understanding of the health risks resulting from these exposures.

6 **1.2.1 Wildfire Management in the United States**

7 The USDA Forest Service was established in 1905 to safeguard the forest reserves/national
8 forests within the United States, taking over responsibility of managing wildfires from the
9 Department of the Interior (DOI) General Land Office. Five years later, a series of massive
10 wildland fires in the northwest United States burned more than 3 million acres, resulting in
11 the deaths of nearly 100 people and the destruction of entire communities [Pyne 2015;
12 USFS, no date, a]. Drawing national attention, this event was pivotal in shaping the nation's
13 strategy of wildfire management, focusing solely on suppression [Busenberg 2004; NIFC, no
14 date]. Policies and practices to prevent and rapidly extinguish wildfires were established to
15 eliminate wildfires from federal lands. A similar posture was adopted by states, resulting in
16 a de facto national policy of fire suppression that lasted for much of the 20th century
17 [Busenberg 2004; Pyne 2015].

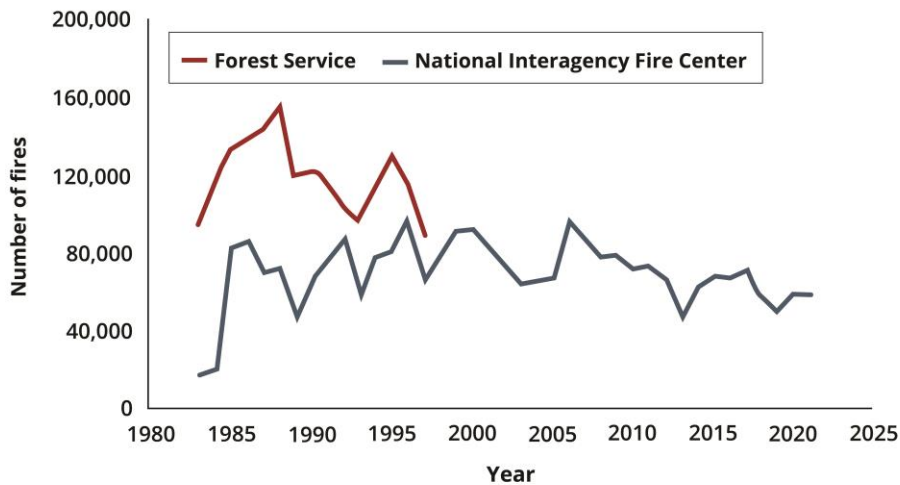
18 Informed by ecosystem studies of the beneficial effects of low-intensity fires on nutrient
19 availability and soil fertility, and in recognition that such fires have the potential to reduce
20 the risk of high-intensity fires, the USDA Forest Service and Department of Interior began in
21 the 1970s a slow transition from a policy of wildfire control to wildfire management. In
22 practice, this initiated a decades-long pivot toward balancing fuel removal, either by
23 mechanical means or through prescribed burns, with wildfire control [Busenberg 2004].
24 According to the report by the Wildland Fire Mitigation and Management Commission
25 [2023], the long-lasting policy of fire suppression resulted in a buildup of fuel that greatly
26 increased the risk of high-intensity wildfires in the United States. These risks have been
27 amplified by trends of increasing extreme weather (e.g., drought, higher global
28 temperatures, and strong surface winds) that can worsen fires [Jain et al. 2022; Zhuang et
29 al. 2021]. Additionally, the continued growth in the WUI, broadly defined as the area where
30 homes are near wildland vegetation, has resulted in an increase in wildfire ignitions,
31 increasing the risk to communities and human life and health [Radeloff et al. 2018].

32 **1.2.2 Increasing Size, Severity, and Duration of Wildfires**

33 Trends of increases in the size and severity of wildfires, along with the duration of wildfire
34 season, have increased the health risks of workers from their exposure to wildfire smoke
35 and also increased the number of workers who are at risk. The [National Interagency Fire
36 Center](#) (NIFC) has compiled reports since 1983 from local, state, and federal agencies
37 involved in firefighting to track the total area and total number of fires in the United States.
38 Burn severity, state-level acreage, and monthly totals are based on data from the
39 interagency [Monitoring Trends in Burn Severity](#) (MTBS) program, which provides the
40 location, ignition date, size, and other statistics for every individual wildfire that meets

1 certain size criteria ($\geq 1,000$ acres in the western United States or ≥ 500 acres in the eastern
2 United States). MTBS compares the “greenness” of satellite images taken before and after a
3 fire to classify how severely the land has been burned. Burn severity provides an indication
4 of the ecosystem damage and how long the effects of wildfires are likely to last.

5 Since the inception of the NIFC in 1983, an annual average of approximately 70,000
6 wildfires has resulted in a total of 5.5 million acres burned per year in the United States. The
7 Forest Service compiled data only until 1997, and its data suggests that the number of
8 wildland fires were higher for the first few years of nationwide data collection. While the
9 annual number of fires has been relatively stable since 1983 (Figure 1–1), the area burned
10 has been increasing over the same time period (Figure 1–2).



11 **Figure 1–1: Number of wildfires in the United States, 1983–2021**

12 Source: <https://www.epa.gov/climate-indicators/climate-change-indicators-wildfires>.

13 Data source: NIFC [no date]. Total wildland fires and acres (1983–2022). Boise, ID: National Interagency
14 Fire Center, www.nifc.gov/fireInfo/fireInfo_stats_totalFires.html. Date accessed May 2022.

15 Data source: USDA Forest Service [2014]. 1991–1997 Wildland fire statistics. Prepared by USDA Forest
16 Service, State and Private Forestry, Fire and Aviation Management staff, and supplemented with historical
17 records provided by Forest Service staff, April 2014.

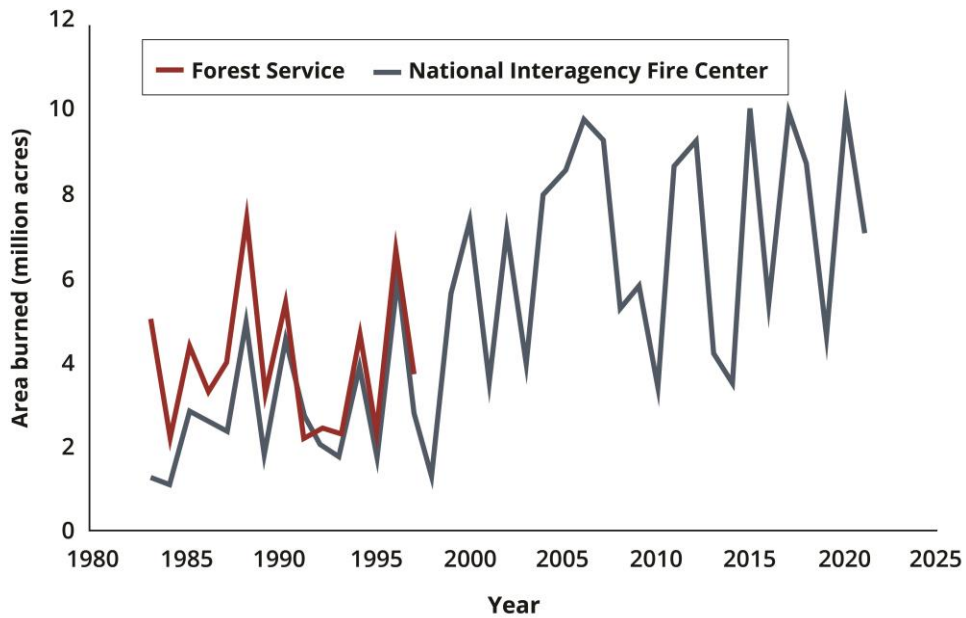


Figure 1–2: Area burned by wildfires in the United States, 1983–2021

Source: <https://www.epa.gov/climate-indicators/climate-change-indicators-wildfires>.

Data Source: NIFC [no date]. Total wildland fires and acres (1983–2022). Boise, ID: National Interagency Fire Center, www.nifc.gov/fireInfo/fireInfo_stats_totalFires.html. Date accessed May 2022.

Data Source: Short, KC [2015]. Sources and implications of bias and uncertainty in a century of U.S. wildfire activity data. *Int J Wildland Fire* 24(7):883–891.

One of the biggest changes to the U.S. wildfire season has been the shift of the start and end of the season, as well as the amount of acreage burned during each month (Figure 1–3). From 1984 to 2001, the fire season could be characterized as starting in May and ending in September, with August having the largest number of acres burned. From 2002 to 2020, the fire season appears to start in February or March and end in September or October. The number of acres burned during the spring and summer months has increased, while the months of June and July appear to have the most acres burned. Importantly, the number of acres burned by wildfires appears to have increased for every month except the winter months (November, December, and January).

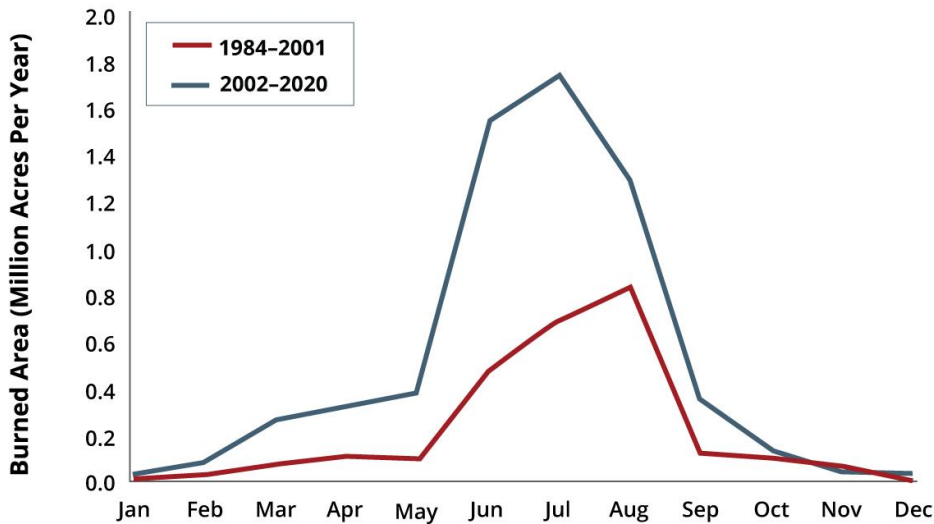


Figure 1–3: Comparison of monthly burned area due to wildfires in the United States between 1984–2001 and 2002–2020

This figure compares the annual distribution of burned area due to wildfires in the United States between the first half of the period of measurement (1984–2001) and the second half (2002–2020).

Source: <https://www.epa.gov/climate-indicators/climate-change-indicators-wildfires>.

Data Source: MTBS [2022]. Direct download. U.S. Geological Survey, USDA Forest Service, Monitoring Trends in Burn Severity, www.mtbs.gov/direct-download. Date accessed: April 2022.

The amount of damage caused by wildfires in the United States has also been increasing since the tracking of wildfires was established in 1984 (see Figure 1–4). The percentage of moderate or high damage in the total acreage burned from 1984 to 2020 fluctuated but the overall trend was upwards. The increase in damage caused by wildfires supports the idea that the number of wildfires has been consistent, but the intensity (i.e., amount of acreage burned) of wildfires is increasing. Outdoor workers, particularly those working in regions prone to wildland fires or who work in firefighting, may be exposed to more wildland fire smoke as these burning trends continue to expand the wildland fire season [Austin et al. 2021].

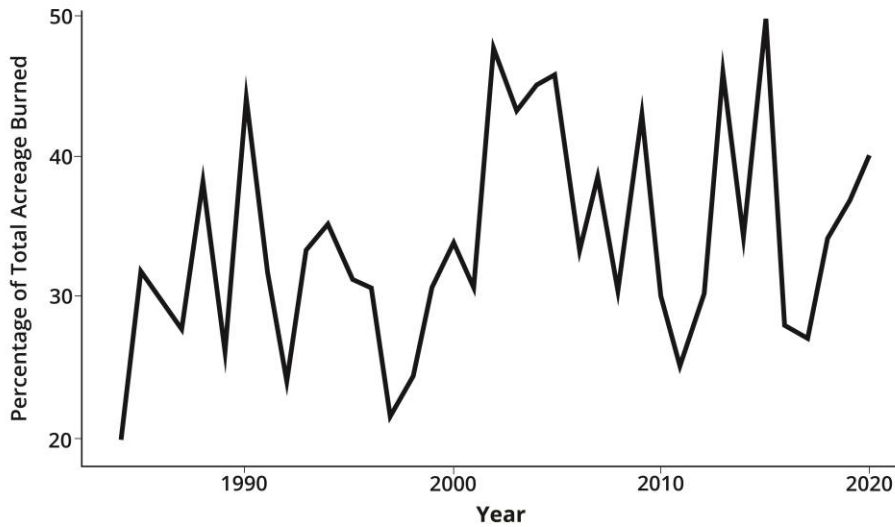


Figure 1–4: Severe* damage caused by wildfires in the United States, 1984–2020

Percentage of total acreage burned refers to the amount of acreage of moderate or high damage caused by large wildfires relative to the total acreage burned per year.

* Severe damage refers to high or moderate wildfire damage.

Created using data from: <https://www.epa.gov/climate-indicators/climate-change-indicators-wildfires>.

While the number of wildland fires has remained consistent over time, the amount of area burned and damaged by wildland fires has been increasing. Consequently, the suppression costs attributed to fighting wildland fires have been increasing as more acres burn across the United States (see Figure 1–5).

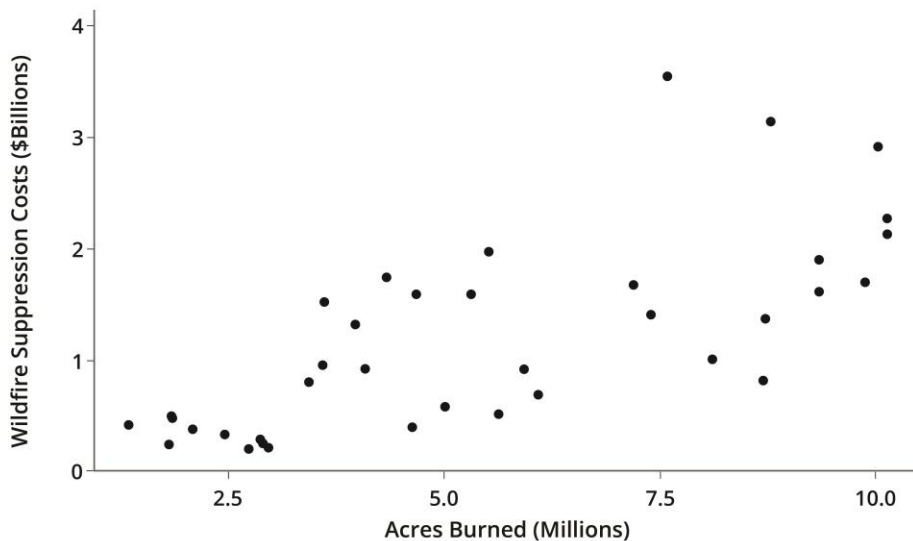
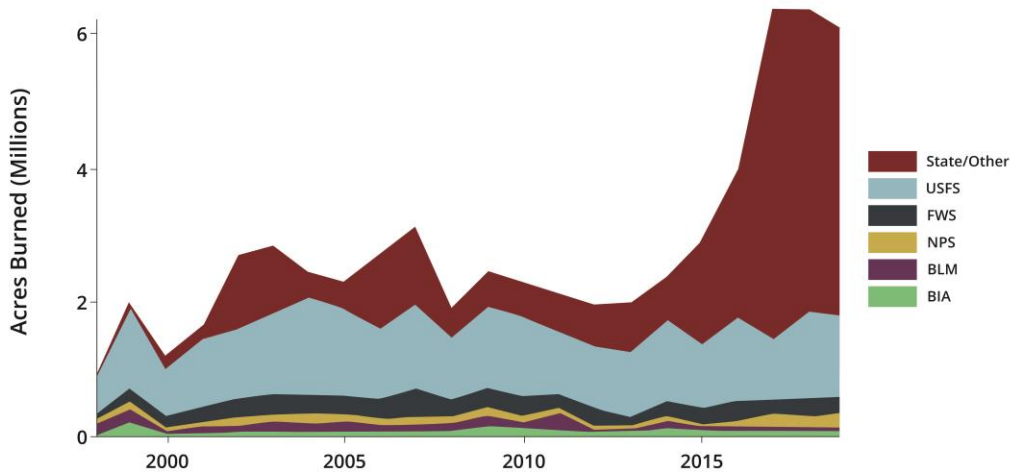


Figure 1–5: Number of acres burned from wildfires and suppression costs, 1985–2021

Created using data from: <https://www.nifc.gov/fire-information/statistics/suppression-costs>.

1 The NIFC also tracks the number of acres burned during prescribed burns by agency (see
2 Figure 1–6). The number of acres burned has been increasing since 1998, but the
3 states/other category has become the largest contributor to prescribed burns. Starting in
4 2015, states/other (states and private lands) became the largest contributor to prescribed
5 burns, and by 2016 they were burning more areas than all other agencies involved in
6 prescribed burns in the United States.



7
8

9 **Figure 1–6: Number of acres burned by agency during prescribed burns**

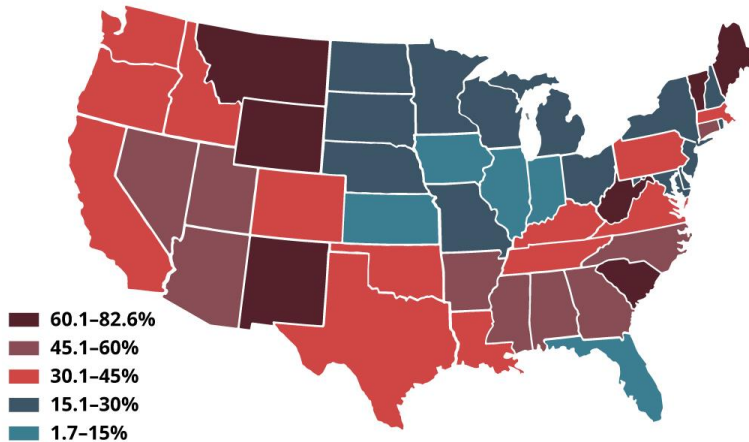
10 Created using data from: <https://www.nifc.gov/fire-information/statistics/prescribed-fire>.

11 Abbreviations: Bureau of Indian Affairs (BIA), Bureau of Land Management (BLM), U.S. Forest Service
12 (USFS), U.S. Fish and Wildlife Service (FWS), National Park Service (NPS), Other (private, Tribal, county,
13 municipalities, Department of Defense, and Bureau of Reclamation lands)

14 1.2.3 Increasing Amount of Wildland Urban Interface

15 The WUI is the zone of transition between unoccupied land and human development. It is
16 the line, area, or zone where structures and other human development meet or intermingle
17 with undeveloped wildland or vegetative fuels. The latest estimate from the U.S. Fire
18 Administration reported that more than 60,000 communities in the United States are at risk
19 from WUI fires [USFA 2023]. More than 3,000 structures per year were destroyed in WUI
20 fires in the United States from 2002 through 2016. The amount of the WUI continues to
21 grow by approximately 2 million acres per year as people and communities expand into this
22 zone. For the western states, a recent analysis of wildfires found that from 1999 through
23 2009 and 2010 through 2020, structure losses from wildfires rose 246% [Higuera et al.
24 2023]. The analysis found wildfires became significantly more destructive and wildfires
25 from unplanned, human-related ignitions (e.g., backyard burning, power lines) were
26 responsible for 76% of all structural loss.

1 WUI fires can have a significant impact on public health due to their proximity to population
2 centers and the variability in the nature of emissions from the combustion of building
3 materials and vehicles. A recent study highlighted that WUI fires can have greater emissions
4 of some hazardous air pollutants compared with natural sources in the airshed [Holder et
5 al. 2023]. Figure 1–7 shows the percentage of houses in WUI by state throughout the United
6 States in 2022. The percentage of homes in WUI is not specifically increasing in one state or
7 region, but rather is increasing across the entire United States.



8 **Figure 1–7: Number of houses in the WUI relative to the total houses in the state**
9 **(percentage)**

10 Data were not available for Alaska or Hawaii.

11 Source: <https://www.usfa.fema.gov/wui/what-is-the-wui.html>.

12 Wildland fires are one of the largest contributors of particulate matter across the United
13 States [Burke et al. 2021; Zhang et al. 2023]. One study of ground- and satellite-based air
14 pollution data from 2000 to 2022 found that since 2016, wildfire smoke had influenced
15 trends in average annual concentrations of particulate matter with an aerodynamic
16 diameter of 2.5 micrometers or less (PM_{2.5}) in nearly three-quarters of states in the
17 contiguous United States [Burke et al. 2023]. The amount of influence wildfire smoke had
18 on air quality was greatest in western and mid-west states. Those states made significant
19 progress to decrease PM_{2.5} air pollution since 2000, but some of that progress has been
20 stagnated or reversed due to PM_{2.5} from wildfire smoke.

21 **1.2.4 Characterization of Risks from Air Pollution**

22 At about the same time that the United States began easing the approach of aggressive
23 wildfire suppression toward a more holistic posture of wildfire management, a burgeoning
24 movement was gaining momentum to better understand the potentially harmful effects of
25 air pollution and address public concerns related to pollution in communities, as well as
26 health and safety risks within the workplace. In 1970, the U.S. Environmental Protection

1 Agency (EPA), OSHA, and NIOSH were created [40 CFR 1.1; Public Law 91-596] to protect
 2 human health and the environment and assure safe and health working conditions for the
 3 American workforce.

4 The Clean Air Act of 1970 required the newly established EPA to set air quality standards
 5 and work with states to implement those standards in preventing and controlling air
 6 pollution [42 USC 7401]. The National Ambient Air Quality Standards (NAAQS) have been
 7 promulgated for six air pollutants that may “reasonably be anticipated to endanger public
 8 health” [42 USC 7408]—sulfur dioxide, nitrogen dioxide, carbon monoxide, lead, ozone, and
 9 particulate matter, and are referred as “criteria” air pollutants. The Clean Air Act further
 10 requires the EPA to conduct a complete review of the science supporting the NAAQS every
 11 5 years and revise standards as appropriate [42 USC 7408, 7409]. These regulatory
 12 requirements have resulted in a significant overall investment and focus on protecting the
 13 general population from ambient air pollution in the context of the NAAQS, regardless of the
 14 source. Of note, the NAAQS are science-based standards in which the EPA cannot consider
 15 cost of implementation [EPA 2024b; Whitman v. American Trucking Associations, 531 U.S.
 16 457 (2001)]. These air quality standards, along with an evaluation and synthesis of the
 17 scientific foundation upon which the standards are based, may therefore be particularly
 18 informative and applicable in efforts to protect workers who spend extended periods of
 19 time outdoors.

20 One of the first pollutants identified by the EPA as both ubiquitous and hazardous to human
 21 populations was particulate matter, broadly defined as a mixture of liquid and solid
 22 particles made up of a variety of substances and across a wide distribution of sizes [EPA
 23 2009]. The first EPA standard for particulate matter was established in 1971 using total
 24 suspended particulates as the indicator. The standard was set a level of 260 µg/m³ for a
 25 24-hour averaging time, and a level of 75 µg/m³ for an annual averaging time [EPA 2009].

26 **Table 1–1. Timeline of particulate matter primary national ambient air quality**
 27 **standards, adapted from EPA [2024a]**

Final rule	Indicator	Averaging time	Level (µg/m ³)	Form
1971	TSP*	24 hour	260 µg/m ³	Not to be exceeded more than once a year
1971	TSP	Annual	75 µg/m ³	Annual geometric mean
1987	PM ₁₀ [†]	24 hour	150 µg/m ³	Not to be exceeded more than once a year on average over 3 years
1987	PM ₁₀	Annual	50 µg/m ³	Annual arithmetic mean, averaged over 3 years
1997	PM _{2.5} [‡]	24 hour	65 µg/m ³	98th percentile, averaged over 3 years
1997	PM _{2.5}	Annual	15.0 µg/m ³	Annual arithmetic mean, averaged over 3 years

Final rule	Indicator	Averaging time	Level ($\mu\text{g}/\text{m}^3$)	Form
1997	PM ₁₀	24 hour	150 $\mu\text{g}/\text{m}^3$	Not to be exceeded more than once a year on average over 3 years
1997	PM ₁₀	Annual	50 $\mu\text{g}/\text{m}^3$	Annual arithmetic mean, averaged over 3 years
2006	PM _{2.5}	24 hour	35 $\mu\text{g}/\text{m}^3$	98th percentile, averaged over 3 years
2006	PM _{2.5}	Annual	15.0 $\mu\text{g}/\text{m}^3$	Annual arithmetic mean, averaged over 3 years
2006	PM ₁₀	24 hour	150 $\mu\text{g}/\text{m}^3$	Not to be exceeded more than once a year on average over 3 years.
2012	PM _{2.5}	24 hour	35 $\mu\text{g}/\text{m}^3$	98th percentile, averaged over 3 years
2012	PM _{2.5}	Annual	12.0 $\mu\text{g}/\text{m}^3$	Annual arithmetic mean, averaged over 3 years
2012	PM ₁₀	24 hour	150 $\mu\text{g}/\text{m}^3$	Not to be exceeded more than once a year on average over 3 years.
2020	—	—	—	Standards retained, without revision.
2024	PM _{2.5}	Annual	9.0 $\mu\text{g}/\text{m}^3$	Annual arithmetic mean, averaged over 3 years
2024	—	—	—	24-hour PM _{2.5} and PM ₁₀ standards retained without revision.

1 * Total suspended particles (TSP).

2 † PM₁₀ is particulate matter with diameter of 10 micrometers or less.

3 ‡ PM_{2.5} is particulate matter with diameter of 2.5 micrometers or less.

4 As shown in Table 1–1, over the past 50 years the EPA has revised and lowered the
5 standard for particulate matter several times, with the indicator changing over time as a
6 result of an increased focus on smaller particles (PM_{2.5}) as the science evolved. Driving these
7 changes has been a significant increase in research on the health effects of exposure to
8 ambient particulate matter, initiated in the 1990s and continuing to the present day. In
9 evaluating the entire body of evidence from across scientific disciplines, the EPA concluded
10 in its most recent Integrated Science Assessment for Particulate Matter that a “causal” or
11 “likely to be causal” relationship exists between exposure to PM_{2.5} and several health effects
12 of interest (see Section 3.1.1). As with any toxic substance, a variety of intrinsic and
13 extrinsic factors may make particular individuals or populations more susceptible to or at
14 increased risk for health effects from exposure to particulate matter [EPA 2022; Sacks et al.
15 2011]. These factors can include age, preexisting disease, race, socioeconomic status,
16 genetic factors, and time spent outdoors.

1 Recognizing that wildland fire smoke is made up of a complex mixture of potentially
2 hazardous pollutants, this hazard review is focused largely on PM_{2.5}, both as an indicator of
3 exposure to the smoke mixture as well as in consideration of its direct health effects. In
4 addition, in terms of mass quantity of pollutants released per dry weight mass burned in a
5 wildfire, PM_{2.5} ranks third among all wildfire smoke components, only behind carbon
6 dioxide and carbon monoxide [Prichard et al. 2020]. Particulate matter, and specifically
7 PM_{2.5}, is among the most extensively studied air pollutants, with well-established adverse
8 health outcomes resulting from both acute and chronic exposures [EPA 2022, 2024c;
9 Landrigan et al. 2018]. According to the EPA, the benefits of regulations under the Clean Air
10 Act far exceed the cost of compliance and prevent 230,000 premature deaths annually, with
11 the majority of those benefits attributable to reductions in exposure to PM_{2.5} [CRS 2017;
12 OIRA 2016]. Although particulate matter comes from diverse sources and components, the
13 EPA continues to use a mass-based indicator for the PM_{2.5} standard, without taking into
14 consideration PM_{2.5} components [EPA 2024b]. In its most recent Integrated Science
15 Assessment for Particulate Matter, the EPA concluded, "... the evidence does not indicate
16 that any one source or component is more strongly related with health effects than PM_{2.5}
17 mass" [EPA 2022]. PM_{2.5} is the primary threat to public health from wildfire smoke [EPA
18 2021] and protecting outdoor workers from occupational exposure to PM_{2.5} is expected to
19 protect them from the adverse health effects of exposure to wildland fire smoke.

20 **1.3 History of NIOSH Activity**

21 Since 2005, NIOSH has committed resources and expertise to reduce exposures of outdoor
22 workers to wildland fire smoke. NIOSH activities related to wildland fire smoke exposure
23 among outdoor workers have been conducted by NIOSH staff, as well as by externally
24 funded researchers and organizations. External funding mechanisms directed to
25 researchers include research grants and cooperative agreements. NIOSH also funds
26 research through the State Occupational Safety and Health Surveillance Program [NIOSH
27 2021], Education and Research Centers [NIOSH 2023b], Centers of Excellence for Total
28 Worker Health [NIOSH 2023c], and Centers for Agricultural Safety and Health (Ag Centers)
29 [NIOSH 2023a]. The results of this research have informed the development of effective
30 controls and interventions to protect outdoor workers from exposure to wildland fire
31 smoke.

32 This section describes the major activities undertaken over the past 20 years by NIOSH staff
33 and externally funded NIOSH researchers related to wildland fire smoke exposure among all
34 outdoor workers, including farmworkers. Research directed at specifically protecting
35 wildland firefighters are not included unless they would apply to the broader category of
36 outdoor workers who typically have less direct exposure to wildland fire smoke.

1.3.1 Farmworkers

NIOSH supports research and outreach to farmworkers through its 12 Centers for Agricultural Safety and Health (see NIOSH's [Centers for Agricultural Safety and Health](#)). These centers are geographically distributed throughout the nation to be responsive to the unique characteristics of each region's agriculture, forestry, and fishing industries. NIOSH's Centers for Agricultural Safety and Health have conducted research, education, and prevention projects related to wildfire smoke exposure in farmworkers in recent years.

All farmworker-specific research described here was conducted by external researchers who were funded by NIOSH. In three agricultural regions of California, interviews with farmworkers and agricultural employers found differing perceptions of risk and responsibility for workplace safety regarding extreme weather events, including wildfire smoke exposure [Riden et al. 2020]. As a result of that study, educational materials on wildfire smoke exposure were developed for agricultural workers and employers [Pinkerton 2020]. Wan et al. [2021] analyzed ash and soil samples from wildfire-affected orchards after the 2017 Thomas Fire (Ventura County, CA) for 8 trace elements and 16 polycyclic aromatic hydrocarbons to document potential inhalation exposure for farmworkers. The study found, "Except for [mercury], the [trace element] concentrations were generally higher in ash samples than those in the soil"; however, "[t]he estimated inhalation of all the [polycyclic aromatic hydrocarbons] was lower than the tolerable limits" [Wan et al. 2021]. Marlier et al. [2022] used climate models to quantify past (2004–2009) and future (2046–2051) potential smoke PM_{2.5} exposure for California's agricultural workers, finding a 190% increase in smoke exposure days classified as "unhealthy for sensitive groups."

In Washington state, Austin et al. [2021] explored the combined burden of heat and air quality for agriculture workers, which included periods of wildfire smoke exposure, finding that concurrent high heat and PM_{2.5} exposures were highest in counties with the largest agricultural populations. Schollaert et al. [2023] tested a low-cost air quality sampling platform both before and during a wildfire smoke event as a possible solution for real-time air monitoring in rural areas, potentially aiding agricultural employers in protecting workers and complying with Washington state wildfire smoke rules. See Section 1.4.3.3 for a description of the Washington state wildfire smoke rule.

As of April 2024, NIOSH Ag Centers were funding three pilot projects and one large research project on wildfire smoke exposure in agricultural worker populations [Pacific Northwest Agricultural Safety and Health Center, no date; Western Center for Agricultural Health and Safety 2022].

1.3.2 All Outdoor Workers

1.3.2.1 NIOSH Activity Related to PM_{2.5} Exposure and Assessment Methods

Extramural and intramural NIOSH-funded research projects have conducted numerous studies investigating woodsmoke and PM_{2.5} exposures as well as exposure assessment methods that are relevant to all outdoor workers. Internally funded research has primarily focused on wildland firefighters, an occupational group not covered by this document. However, in 2020, NIOSH researchers participated in the Interagency Wildland Fire Air Quality Response Program as air resource advisors. In this role, they found that while wildland fire smoke drifted long distances (and therefore affected many downwind communities), the majority of areas with poor air quality were observed closer to the fire, such as fire camps (where firefighters and support staff might sleep, eat, and work during an event) and adjacent communities [CDC 2020].

The remaining studies in this section represent external research funded by NIOSH. A 2005 study was developed to determine biological markers of woodsmoke exposure, finding that measuring urinary methoxyphenol could be useful for gauging occupational woodsmoke exposures [Simpson 2005]. In 2007, researchers funded by NIOSH published a study comparing PM_{2.5} among outdoor and indoor workers in two Mexican cities [Tovalin-Ahumada 2007]. Though not specific to woodsmoke, the findings indicated that among the worker populations and in the conditions studied, fixed ambient monitors were not sufficient for monitoring PM_{2.5} exposure among outdoor workers. Instead, personal sampling (or personal breathing zone sampling) was found to be more appropriate [Tovalin-Ahumada 2007]. Researchers also found that outdoor workers experienced higher PM_{2.5} exposure during occupation-related activities than during nonoccupational activities.

In vivo and in vitro studies each conducted under the same cooperative agreement were published in 2009 and 2011. The 2009 study [Wegesser et al. 2009] exposed mouse bioassays (live mice) to PM_{2.5} samples collected during peak concentrations of smoke during the 2008 California wildfires and PM_{2.5} from “normal” conditions in the same region from the year prior (2007). Concentrations of PM_{2.5} in the 2008 wildfire smoke samples were greater than that of the 2007 normal air. The wildfire smoke PM_{2.5} was found to be more toxic to lungs when comparing with equal volumes of PM_{2.5} from the normal air. The 2011 in vitro study [Franzi et al. 2011] built on these findings by exposing cell cultures to coarse PM from wildland fires, as opposed to coarse PM from ambient air pollution. They found an increase in oxidative stress and a consequent increase in the death of macrophages, a type of white blood cell. Having fewer macrophages alters the body’s response to acute infections, reducing the body’s ability to fight infections.

Navarro et al. [2019] published a study that measured PM_{2.5} and polycyclic aromatic hydrocarbons (PAH) levels at a wildland fire incident command post in California. The study found that personnel assigned to the post were exposed to quantifiable PM_{2.5} and PAHs during the response. Thus, non-firefighter personnel were exposed, and researchers indicated these findings could be applied to nearby communities [Navarro et al. 2019].

1 Cleland et al. [2020] combined data from various air monitoring stations to investigate the
2 impact of the 2017 California wildland fires on air quality. Using this retrospective data, the
3 researchers estimated that approximately 65,000 people were exposed to very unhealthy
4 air, which the EPA defines as having 24-hour average PM_{2.5} concentrations exceeding 150.5
5 µg/m³. Given the widespread nature of the exposure, outdoor workers were likely among
6 those affected by the poor air quality resulting from the fires.

7 Wu et al. [2021] focused on wildland firefighter exposures and found that PM_{2.5} exposure
8 concentrations were greater in the Midwest than the West and Southwest, though the
9 duration of exposure was shorter. The investigators proposed that this difference might be
10 related to the type of vegetation serving as wildland fire fuel. These findings could be
11 extrapolated to exposures among outdoor workers in the Midwest.

12 A 2022 evaluation of the risk of PM_{2.5} exposure among construction workers in Washington
13 state used quarterly employment statistics to identify construction workers and EPA air
14 quality data to determine the number of poor air quality days [Zuidema et al. 2022].
15 Looking at a 10-year retrospective period, implementing recommendations at the
16 “encouraged” level (as defined by the emergency Washington rule and as opposed to the
17 less restrictive “required” threshold) would have led to 5.5 times more days in which the
18 wildland fire protection rule would have been in effect [Zuidema et al. 2022]. As a result, an
19 estimated 1.35 million N95® filtering facepiece respirators (FFRs) for construction workers
20 would have been recommended for use [Zuidema et al. 2022]. N95 FFRs are the minimum
21 protection required to protect workers from particulate hazards. Any NIOSH Approved®
22 respirator that offers particulate protections can be used in an occupational setting.

23 In summary, NIOSH-funded research, conducted intramurally and extramurally, has
24 identified potential practices for assessing exposure to wildland fire smoke. These include
25 using personal breathing zone monitors and measuring urinary methoxyphenol levels.
26 Studies have found that PM_{2.5} from wildland fire smoke is more harmful than PM_{2.5} from
27 ambient air, with poor air quality observed in closer proximity to the fire. Additionally,
28 wildland fire smoke PM_{2.5} concentrations were found to be higher in the Midwest compared
29 with other regions. Retrospective data analysis allows for determining the number of
30 community members, including outdoor workers, who were exposed to wildland fire smoke
31 as well as estimating the overall number of community members who might be affected by
32 proposed rules and guidance proposed by state and federal agencies.

33 **1.3.2.2 NIOSH Activity Related to Health Effects of Wildland Fire Smoke**

34 NIOSH internal research and external research funded by NIOSH has produced numerous
35 studies investigating health effects related to wildland fire smoke, as well as those related to
36 PM_{2.5} exposure, that could be extrapolated to wildland fire smoke exposures.

37 In a review conducted by staff from NIOSH as well as other federal and academic partners,
38 Navarro et al. [2021] concluded that wildland fire smoke exposure increases susceptibility
39 to respiratory disease. Although this review focused on wildland firefighters, there was
40 limited research related to wildland firefighter respiratory health and smoke exposure. For
41 this reason, the health studies in the Navarro et al. [2021] review were from the general

1 public and may be applicable to all outdoor workers, given the difference in exposure to
2 smoke and fewer fitness-for-duty requirements among most outdoor workers and wildland
3 firefighters [Navarro et al. 2021]. This review found that exposure to wildland fire smoke
4 may alter workers' susceptibility to respiratory infections because of airway inflammation,
5 cell toxicity, and oxidative stress [Navarro et al. 2021].

6 The remaining studies in this section represent external research funded by NIOSH.
7 Wettstein et al. [2018] conducted a study examining the relationship between wildland fire
8 smoke days and emergency department visits, accounting for possible lag days to consider
9 delayed health effects. The researchers found that wildland fire smoke days were associated
10 with increased rates of emergency department visits related to cardiovascular,
11 cerebrovascular (e.g., ischemic stroke), and respiratory conditions [Wettstein et al. 2018].

12 A 2021 study detailed that asthma is exacerbated by multiple environmental factors,
13 including wildland fires [Rorie and Poole 2021]. Investigators presented the case that
14 wildland fires, including large wildland fires, are likely to be the norm, and their
15 contribution to poor air quality will lead to reduced asthma control [Rorie and Poole 2021].
16 In a different 2021 study, county-level daily PM_{2.5} concentrations and mortality data were
17 used to estimate excess mortality that was attributable to wildland fire smoke exposure. An
18 estimated 4.4% increase in all-cause mortality was associated with the specific wildland fire
19 smoke event in this study [Liu et al. 2021].

20 Another 2021 study indicated that wildland fire smoke accounted for excess respiratory,
21 cardiovascular, and asthma hospital admissions [Cleland et al. 2021]. Estimates of daily
22 ground-level PM_{2.5} exposure from wildland fire smoke were combined with hospital
23 admissions data, finding an increase in respiratory and asthma admissions among adults
24 aged 20–64 years [Cleland et al. 2021]. Cardiovascular admissions were also increased but
25 were reported for all ages (0–99 years of age) [Cleland et al. 2021].

26 Regarding reproductive and developmental health, Lee et al. [2022] compared outdoor
27 PM_{2.5} exposure with the prevalence of low birthweight deliveries in 7,785 census tracts in
28 California, finding a small increase in the percentage of low birthweight deliveries. These
29 findings were amplified in areas of high poverty [Lee et al. 2022]. In 2023, outdoor
30 residential exposure to air pollution (including some level of wildland fire smoke) was
31 associated with an increased incidence of active tuberculosis diagnosis [Linde et al. 2023].

32 In summary, NIOSH-funded research, conducted intramurally and extramurally, has
33 identified that wildland fire smoke exposure was associated with increases in morbidity
34 and mortality related to the cardiovascular, cerebrovascular, and respiratory symptoms.
35 Additionally, wildland fire smoke exposure was associated with reproductive conditions
36 such as low birthweight.

37 NIOSH maintains a webpage, [Outdoor Workers Exposed to Wildfire Smoke](#), that provides
38 information on topics such as what employers and workers can do to reduce smoke
39 exposure and respiratory protection options.

1.3.3 NIOSH Activity Related to Respiratory Protection for Outdoor Workers

Respiratory protection for outdoor workers (see Section 5.1.3) to protect against wildland fire smoke is a challenging topic, given the complex nature of wildland fire smoke (see Section 2.1). Personal protective equipment (PPE), including respiratory protection, is the last resort to protect workers as part of the hierarchy of controls.

In 1995, NIOSH became the sole authority for approving respirators used in occupational settings in the United States, with the exception of mine emergency respiratory protection [Approval of respiratory protective devices, 1995]. Since 1970, NIOSH has approved over 16,000 respirators, and as of 2024, currently has nearly 10,000 active respirator approvals. The 90 manufacturers that are issued a NIOSH certificate of approval have approximately 120 manufacturing sites in 25 countries [NIOSH 2024a].

NIOSH has been responding to inquiries from the public, including workers, on respiratory protection recommendations for many years. This includes respiratory protection for wildfire smoke. Other topics include cleanup from the 2001 World Trade Center collapse and its aftermath, anthrax bioterrorism and naturally-occurring events, hazards caused by earthquakes, concerns about mold following floods, and many general exposures from a diversity of chemical inhalation hazards.

Since 2018, collaborations with the CDC National Center for Environmental Health (NCEH), the American Thoracic Society, the Department of State, the EPA, and the World Health Organization, detailed below, have further expanded NIOSH's ability to reach workers and the public.

In January 2018, NIOSH posted a [NIOSH Science Blog](#) with partners at NCEH to help those voluntarily wearing respirators at work. This blog was also for non-occupational respirator users to understand important information about wearing respirators in the absence of a respiratory protection program. The blog addressed the following topics: (1) how respirators work, (2) how to properly wear a respirator, and (3) best practices associated with respiratory protection [NIOSH 2018c].

In 2018, NIOSH participated in a workshop, "Interventions to Reduce Exposure Levels and Health Risks from Outdoor Air Pollution," which was sponsored by the American Thoracic Society and held in San Diego, California. The workshop gathered national and international experts to consolidate the current knowledge and identify knowledge gaps associated with personal interventions to reduce the risk for outdoor air pollution exposures [Laumbach et al. 2021]. During a consultation with global experts in Geneva, Switzerland, in 2019, NIOSH participated in the World Health Organization's effort to further explore personal interventions to address air pollution exposures [WHO 2020].

The growing concerns about wildfire smoke and other particulate inhalation hazards led NIOSH to initiate a collaboration with the Department of State and the EPA. NIOSH

1 partnered with EPA on its publication of “Wildfire Smoke: A Guide for Public Health
2 Officials” [EPA 2019c].

3 The partnership also produced a jointly sponsored workshop hosted by the National
4 Academies of Science, Engineering, and Medicine (NASEM) on respirators in occupational
5 and nonoccupational settings [NASEM 2021].

6 The workshop discussions focused on the following:

- 7 1. Reviewing lessons learned from the past 100 years of respiratory protection.
- 8 2. Exploring current respiratory protection needs and risks for nontraditional worker
9 groups and the public.
- 10 3. Reviewing current practices of the NIOSH respirator approval program and other
11 conformity assessment processes for respirators, exploring conformity assessment
12 processes in other countries, by third-party organizations, and in private industry.
- 13 4. Discussing the risks and benefits of these approaches in the context of respiratory
14 protective devices use by nontraditional workers and the public.

15 This workshop served as the foundation for a comprehensive consensus study sponsored
16 by the same organizations and the Centers for Disease Control and Prevention (CDC)
17 Foundation to identify consensus recommendations on respiratory protection for workers
18 without respiratory protection programs, and also the public. NASEM published consensus
19 recommendations in February 2022 [NASEM 2022].

20 The recommendations in the report address both worker and public needs by establishing a
21 framework for oversight and guidance for respiratory protection. The NASEM framework
22 describes six components that align with the industrial hygiene hierarchy of protection
23 model and the NIOSH Conformity Assessment Framework [NIOSH 2018a] that are to be
24 used to link respirators with users. The framework recognizes that workers are more likely
25 to appropriately use PPE when they are confident that the equipment will provide the
26 intended protection based on its conformance with appropriate standards. It presents a
27 comprehensive, tailor-made conformity assessment program as the most effective way to
28 manage risks of a non-conforming PPE to instill confidence in PPE users.

29 The NASEM presented a systematic approach and actions to be taken to ensure respiratory
30 protection of the public and workers not covered by a respiratory protection program,
31 including those exposed to wildfire smoke. The committee’s proposed framework and its
32 associated recommendations constitute a template for action, detailing the steps that need
33 to be taken. The report provides several recommendations to meet the respiratory
34 protection needs of workers and the public, and to foster collaboration between partners to
35 holistically address the needs of workers and the public.

36 Five overarching recommendations involve workers:

- 37 1. Build on the foundation of OSHA’s respiratory protection programs and NIOSH, as the
38 approving authority for respirators.
- 39 2. Have OSHA serve as the coordinating entity.
- 40 3. Broaden workers’ coverage through changes to the Occupational Safety and Health
41 Act language and interpretation.
- 42 4. Establish new OSHA standards for triggering coverage.

1 5. Expand NIOSH conformity assessment capacity and NIOSH research.

2 Three recommendations concerning workers emphasize leadership by OSHA, states, and/or
3 Congress, and they focus on ensuring protection from inhalation hazards for all workers.

4 Two recommendations center on generating and using NIOSH Approved® respirators. Two
5 recommendations focus on meeting expanded worker needs for respirator access, guidance,
6 and training on respirator use. Finally, two recommendations concentrate on building a
7 strong scientific foundation.

8 The NASEM Consensus study report provides a comprehensive strategy for addressing
9 respiratory protection needs for workers without respiratory protection programs, and also
10 the public. Federal and private sector coordination is required at many levels to ensure all
11 workers are protected.

12 During the 2023 Canadian wildland fires that resulted in a significant wildland fire smoke
13 incursion to the United States, NIOSH, CDC’s NCEH, and the EPA authored a *NIOSH Science*
14 *Blog* post to help workers and the public better understand how they could protect
15 themselves, including choosing the right respirator [NIOSH 2023d].

16 **1.4 History of Other Government**
17 **Organizations’ Related Activities**

18 Many U.S. federal agencies have statutory authorities related to mitigating risks from
19 wildland fires. A broad coalition of a majority of these federal organizations, along with
20 select non-federal interested parties (e.g., states, municipalities, tribes, and nonprofits), was
21 established in 2021 under the Infrastructure Investment and Jobs Act (Public Law 117-58).
22 Co-chaired by representatives from the USDA, DOI, and the U.S. Department of Homeland
23 Security (DHS), the Wildland Fire Mitigation and Management Commission was charged
24 with developing recommendations to “improve federal policies relating to (1) the
25 prevention, mitigation, suppression, and management of wildfires in the United States; and
26 (2) the rehabilitation of land in the United States devastated by wildfires.”

27 The authorizing language expressly required representation from the Bureau of Land
28 Management, the National Park Service, the Bureau of Indian Affairs, the U.S. Fish and
29 Wildlife Service, and the Forest Service, and it allowed for including representatives from
30 other federal agencies, as deemed appropriate by the co-chairpersons (Public Law 117-58).
31 Although the Commission did not have representation from either the U.S. Department of
32 Health and Human Services (HHS) or the U.S. Department of Labor (DOL), in addition to a
33 number of recommendations related to protecting wildland firefighters, the Commission
34 recommended the “completion of a human health risk assessment for worker exposure to
35 wildland fire smoke and smoke from wildfires in the built environment” in its final report.
36 The authors of the report argued that while OSHA may use the existing standard for
37 particulates not otherwise regulated (PNOR) to protect outdoor workers from wildfire
38 smoke, this is not appropriate due to: (1) the unique attributes of wildland fire smoke, (2)
39 the smaller particle size (aerodynamic equivalent diameter, or $d_{ae} \leq 2.5$ micrometers (μm),
40 and (3) co-pollutants to which workers are exposed [Wildland Fire Mitigation and

1 Management Commission 2023]. Note, the OSHA standard for respirable PNOR ($d_{ae} \leq 4.0$
2 μm) is $5.0 \text{ mg}/\text{m}^3$ [Air contaminants, 2017].

3 In the year before the Wildland Fire Mitigation and Management Commission was
4 established, the U.S. Government Accountability Office (GAO) initiated a performance audit
5 to examine agency efforts to manage human health risks from wildfire smoke [GAO 2023].
6 The GAO's report, published 6 months before the release of the Commission report, focused
7 on the work of the EPA in protecting human health from wildfire smoke and coordinating
8 its activities with the USDA Forest Service and DOI. The report included six
9 recommendations to help communities prepare for and manage risks from wildfires and
10 wildfire smoke, as well as increase coordination between the EPA, USDA, and DOI to align
11 land management, wildfire risk mitigation, and air quality goals [GAO 2023].

12 Two of the recommendations explicitly addressed public health risks to wildfire smoke:

13 **Recommendation 1:** "The Administrator of the EPA should develop and document a
14 coordinated approach for EPA's actions to help communities prepare for and respond to the
15 air quality and public health risks of wildfire smoke. The approach should align with leading
16 practices for collaboration, including establishing goals, identifying and leveraging
17 resources, and clarifying key stakeholder roles and responsibilities."

18 **Recommendation 5:** "The Administrator of the EPA should, in consultation with federal
19 land management agencies, identify and develop additional information on reducing risks
20 from wildfire smoke to air quality and public health through wildfire risk mitigation" [GAO
21 2023].

22 Although the report was focused on managing and mitigating risk of wildfire smoke
23 exposure to communities, successful implementation of the recommended actions is also
24 likely to benefit the health of outdoor workers.

25 **1.4.1 Fire Management within the USDA Forest Service** 26 **and the Department of the Interior**

27 The USDA Forest Service and DOI have several offices with responsibilities related to
28 wildfire management and response [NIFC 2024]. A detailed description of these
29 organizations falls outside the scope of this hazard review; however, two groups within
30 these departments bear responsibilities with a direct or indirect impact on wildland fire
31 smoke.

32 The Wildland Fire Leadership Council (WFLC) is made up of representatives from USDA,
33 DOI, DHS, and the Department of Defense, as well as elected officials from states, tribes,
34 counties, and municipal governments [Forests and Rangelands 2016]. Guided by a formal
35 cohesive wildland fire management strategy, the WFLC coordinates activities and provides
36 recommendations related to wildland fire management. These recommendations include
37 measures to prevent and safely respond to wildland fires, as well as actions communities
38 can take to reduce risk from wildfires and wildfire smoke [WFLC 2023].

1 Similarly, the National Wildfire Coordinating Group (NWCG), based within the National
2 Interagency Fire Center (NIFC), includes members from federal and non-federal agencies
3 and organizations and works to establish standards and recommendations related to
4 wildland fire operations [NWCG 2023]. NWCG has established a committee of subject
5 matter experts and other interested parties focusing on smoke (1 of 17 committees within
6 the larger organization). The committee facilitates collaboration between programs and
7 provides a forum to discuss strategies and guidance in managing smoke from wildland fires
8 [NWCG, no date].

9 **1.4.2 Federal Efforts to Protect** 10 **Communities from Wildland Fire Smoke**

11 **1.4.2.1 U.S. Environmental Protection Agency**

12 The EPA conducts research and provides guidance specific to assessing and mitigating
13 health risks of wildland fire smoke [EPA 2019b]. The agency maintains several useful
14 communication materials related to wildland fire smoke on the website [AirNow](#), including a
15 fire and smoke map developed by the U.S. Forest Service (USFS) and EPA that shows recent
16 and active fires, alongside air quality data throughout the United States (see AirNow's
17 [Wildfires](#)). The website includes both data reported from states using federal reference
18 monitoring methods, and data from private citizens using less expensive and likely less
19 accurate sensors [Barkjohn et al. 2022]. See Section 4.4 for a more in-depth overview of
20 these exposure assessment tools. Air quality data collected and reported by states are used
21 to derive an Air Quality Index (AQI), a tool to communicate air quality to the general public
22 [EPA 2018]. As described in Section 1.4.3.3, some states have incorporated the use of the
23 AQI during wildfire smoke events to protect outdoor workers.

24 The EPA first published guidance for public health officials on wildfire smoke in 2001 in
25 response to public concerns of the potential health risks of smoke from wildfires that
26 occurred in California in 1999. This initial document was developed following a workshop
27 held at the University of Washington in June 2001 with subject matter experts convened by
28 California's Office of Environmental Health and Hazard Assessment and the EPA. Updated in
29 2008, 2012, 2016, 2019, and 2021, this guide includes practical strategies and
30 recommendations to limit exposure to wildfire smoke and mitigate risk to the general
31 public, as well as at-risk populations, including outdoor workers [Stone and Sacks 2024;
32 EPA 2021a].

33 Within the Office of Research and Development, the EPA has established a formal strategic
34 research action plan and framework related to wildland fire [EPA 2019b]. The EPA has a
35 major focus on the human health risks of exposure to wildfire smoke, but the agency also
36 conducts research to better characterize the impact of wildfires on drinking water and
37 ecological systems. Its work involves extensive collaboration with many federal agencies
38 such as USDA, DOI, HHS, the National Aeronautics and Space Administration (NASA), the
39 Department of Energy, and the National Institute of Standards and Technology (NIST).
40 Examples of tangible products and tools from these collaborations include developing an

1 assessment to compare the impacts of prescribed fire versus wildfire (USDA and DOI), and
2 the integration of smoke plume models into a hazard mapping system (National Oceanic
3 and Atmospheric Administration, or NOAA) [EPA 2019b; EPA 2021b].

4 **1.4.2.2 National Center for Environmental Health**

5 The CDC's NCEH conducts research to better assess exposures to wildfire smoke and
6 understand the health impacts of these exposures, along with providing guidance to
7 mitigate health risk [CDC 2023; Michael et al. 2023; Mirabelli et al. 2022; Vaidyanathan et al.
8 2018]. NCEH has developed a public-facing, near-real time tool identifying areas and
9 populations vulnerable to wildland fire that can be accessed through the [National](#)
10 [Environmental Public Health Tracking Network](#). Although much of this work is focused on
11 community impacts of wildfire smoke, NCEH also provides guidance on the potential for
12 private water wells to become contaminated after a wildfire and on hazards workers might
13 encounter during cleanup efforts after a wildfire. Guidance for the public is available at
14 [Safety Guidelines: Wildfires and Wildfire Smoke | Wildfires | CDC](#).

15 **1.4.2.3 National Oceanic and Atmospheric Administration**

16 In developing tools and technologies to forecast and track smoke from wildfires, the work of
17 NOAA provides support in protecting communities and outdoor workers from wildland fire
18 smoke. In addition to their hazard mapping system, NOAA has recently released their
19 [Regional Advanced Visible Emissions](#) (RAVE) product that estimates wildfire emissions
20 using heat signatures. Additionally, a new satellite is being developed with an anticipated
21 launch date in the early 2030s equipped with advanced instrumentation that will improve
22 air quality monitoring and forecasting [NESDIS 2024].

23 **1.4.2.4 National Institute of Standards and Technology**

24 NIST conducts research on hazards of smoke from wildland fire occurring in the wildland-
25 urban interface. This work includes characterization of emissions from WUI fires, as well as
26 modeling of the spread of these fires within communities [NIST, no date]. As fires in the
27 WUI may result in greater emissions of hazardous air pollutants relative to wildfires or
28 prescribed fires, this research has the potential to provide valuable information in efforts to
29 protect communities and outdoor workers.

30 **1.4.2.5 Other Federal Partners**

31 Several additional federal agencies conduct research or provide tools that may inform
32 efforts to protect communities and workers from wildland fire smoke. This work includes
33 National Science Foundation's research to improve the characterization of components of
34 wildfire smoke [National Science Foundation 2018], USFS's modeling of fire and smoke
35 behavior [USFS, no date, b], and NASA's detection of active fires [NASA, no date].

36 **1.4.3 Authorities and Efforts to Protect Outdoor Workers**

37 Within the DOL, OSHA and the Mine Safety and Health Administration (MSHA) establish and
38 enforce health and safety standards to protect workers in the United States. [Public Law 91-
39 596; Public Law 91-173]. NIOSH is an institute within CDC and HHS that is responsible for

1 conducting occupational health and safety research and making recommendations to
2 improve the health and safety of workers [Public Law 91-596]. Although generally
3 characterized as a non-regulatory agency, guidance and recommendations issued by NIOSH
4 are often used by other agencies responsible for developing and enforcing workplace safety
5 and health regulations. NIOSH is also directly responsible for several regulations, including
6 the approval of respiratory protective equipment [NIOSH 2018a]. NIOSH’s efforts in
7 protecting workers from wildland fire smoke are described in Section 1.3. Apart from these
8 agencies focused on occupational health and safety, other federal agencies establish best
9 practices or guidelines to protect their own workforce. For example, the U.S. Army Public
10 Health Command has developed chemical exposure guidelines for deployed military
11 personnel for a variety of substances, including particulate matter [APHC 2010]. The
12 National Wildfire Coordinating Group has also recommended occupational exposure limits
13 for carbon monoxide (CO) and PM_{4.0} to protect wildland firefighters [Reinhardt and Broyles
14 2019; USDA 2013].

15 **1.4.3.1 Occupational Safety and Health Administration (OSHA)**

16 OSHA is part of a coalition of federal agencies known as Weather-Ready Nation
17 Ambassadors with the shared mission of “working with the National Oceanic and
18 Atmospheric Administration (NOAA) and other Ambassadors to strengthen national
19 preparedness for and resilience against extreme weather” [OSHA, no date, a]. To this end,
20 OSHA and NOAA have collaborated on public education to improve the way people prepare
21 for and respond to wildfires. OSHA has primarily focused on employer responsibilities for
22 the safety and health of workers and protecting workers from anticipated hazards
23 associated with wildfire response and recovery operations. OSHA notes the following: “Each
24 employer is responsible for the safety and health of its workers and for providing a safe and
25 healthful workplace for its workers. Employers are required to protect workers from the
26 anticipated hazards associated with the response and recovery operations for wildfires that
27 workers are likely to conduct” [OSHA, no date, a]. For additional OSHA information about
28 wildfires, see OSHA’s [Wildfires](#).

29 In February 2024, OSHA published a notice of proposed rulemaking (NPRM) to issue a new
30 safety and health standard, titled Emergency Response, to replace the existing Fire Brigades
31 Standard. Like the initiatives described above, this standard is also primarily focused on
32 emergency responders (instead of other outdoor workers). The new standard is intended to
33 address a broader scope of emergency responders and include programmatic elements to
34 protect emergency responders from various occupational hazards. For more information,
35 see OSHA’s [Emergency Response Rulemaking](#).

36 **1.4.3.2 National Institute of Environmental Health Sciences (NIEHS)**

37 The National Institute of Environmental Health Sciences (NIEHS) is part of the National
38 Institutes of Health. The mission of NIEHS is to discover how the environment affects people
39 to promote healthier lives. To achieve this, NIEHS “focuses on basic science, disease-
40 oriented research, global environmental health, and multidisciplinary training for
41 researchers” [NIH 2024].

1 Examples of research supported by NIEHS about the hazards of wildfire smoke can be found
2 in its curated Wildfire Smoke Collection of peer-reviewed manuscripts published in the
3 NIEHS Environmental Health Perspectives. The collection includes research published
4 through 2020 (see NIEHS’s [Wildfire Smoke Collection](#)). NIEHS also offers career and worker
5 training related to wildfire smoke. The NIEHS Worker Training Program and its grant
6 awardees provide resources and training in support of wildfire response operations in the
7 United States. These resources, aimed at protecting the health and safety of those
8 responding to wildfires, can be found at NIEHS’s [Wildfires](#) website.

9 A more recent resource developed by NIEHS broadly addresses the areas of climate change
10 and their effects on human health. The NIEHS Global Environmental Health Program
11 released the Climate Change and Human Health Glossary in September 2023. The
12 searchable online resource includes nearly 300 terms and definitions relevant to the science
13 of climate change and health research and policy. The glossary is designed to encourage
14 consistency in the use of specific terms, improve global understanding of these topics,
15 encourage data sharing, and inform decision-making to protect health from climate-related
16 effects (see NIEHS’s [Climate Change and Human Health Glossary](#)).

17 **1.4.3.3 State Occupational Health and Safety Programs**

18 Three states with OSHA-approved state workplace health and safety programs—California,
19 Oregon, and Washington—have recently enacted rules that require employers to take
20 specific actions to protect outdoor workers from wildfire smoke [California Code of
21 Regulations 2019; Oregon Administrative Rules 2022a,b; Washington Administrative Code
22 2024a,b]. These rules are similar in that specific requirements are linked to levels of EPA’s
23 AQI for PM_{2.5} (Figure 1–8). Additional controls are required with increasing AQI values in
24 each of these states including training, feasible engineering and administrative controls, and
25 voluntary or mandatory respirator use. See Chapter 5 for more information about these
26 state regulations.

Air Quality Index (AQI) Categories for PM _{2.5}	AQI Values	PM _{2.5} Concentration (µg/m ³)
Good	0–50	0–9.0
Moderate	51–100	9.1–35.4
Unhealthy for Sensitive Groups	101–150	35.5–55.4
Unhealthy	151–200	55.5–125.4
Very Unhealthy	201–300	125.5–225.4
Hazardous	301+	225.5+

AQI Threshold of Employer Requirements to Protect Outdoor Workers



Figure 1–8: EPA Air Quality Index (AQI) and thresholds for requiring training and feasible engineering and administrative controls to protect outdoor workers in the states of Washington, Oregon, and California

Source: [AirNow.gov; California Code of Regulations 2019; Oregon Administrative Rules 2022a,b; WAC 2024a,b].

Note, thresholds for requirements related to availability and use of respirators is initiated at higher AQI values in each state.

1.4.3.4 Air Quality Index

The AQI is a tool that allows the EPA to communicate to the public information about air quality in a way that is relatively easy to understand. The AQI is divided into six color-coded bins that include descriptions of increasing levels of concern (Good, Moderate, Unhealthy for Sensitive Groups, Unhealthy, Very Unhealthy, and Hazardous) (see Figure 1–8).

Individual AQIs are calculated for five of the six criteria air pollutants (see Section 1.2.4): particulate matter (both PM_{2.5} and PM₁₀), ozone, sulfur dioxide, nitrogen dioxide, and CO [EPA 2015; EPA 2018; 42 USC 7408]. Note that the EPA has not established an AQI for lead, which is currently designated as a criteria pollutant, and the set of pollutant AQIs presented on [AirNow](#) will vary based on season and geographic location. An AQI value of 100 represents the upper end of the “moderate” level of concern, and roughly corresponds to the short-term NAAQS for each of the five pollutants. With increasing concentrations above the NAAQS, the EPA has established ranges and breakpoints of pollutant concentrations that correspond to increasing values of the AQI [EPA 2018; EPA 2024b].

1.4.3.5 National Ambient Air Quality Standards (NAAQS)

Several states are using the AQI in their rules to protect outdoor workers, and given that the AQI is derived from NAAQS, it is worthwhile to evaluate the stated purpose of these standards. Simply put, under 42 USC 7409, the Administrator of the EPA is to use their

1 judgment in establishing and revising air pollution standards that are requisite (i.e., no
2 more or less stringent than necessary), to protect public health with an adequate margin of
3 safety. In considering the definition of “public health” under this statute, it is instructive to
4 review the NAAQS for ozone in which outdoor workers are specifically identified as an at-
5 risk population, and for which the 8-hour averaging time corresponds to a typical work shift
6 [EPA 2020]. Further, while the EPA has not explicitly established outdoor workers as an at-
7 risk population in their particulate matter science or policy assessments [EPA 2022; EPA
8 2023], outdoor workers are explicitly included as an at-risk group in the wildfire smoke
9 guide for public health officials [EPA 2021a].

10 **1.4.4 Activities Outside the United States**

11 In recent years Europe, Canada, and Australia have been impacted by wildfire seasons and
12 increased exposure to wildland fire smoke. The European Commission reported that in
13 2022, 30 European countries experienced wildfires affecting a total of nearly 900,000
14 hectares (2.2 million acres) of land burned [European Commission 2023]. In 2023, Ukraine,
15 Greece, Italy, Spain, Portugal, and several other countries all experienced greater frequency
16 and intensity of wildfires [Statista 2024]. Canada and Australia also experienced significant
17 wildfire activity that received worldwide attention. The governments of both countries have
18 developed guidance for employers and workers to minimize exposures and prevent
19 associated health effects from working outdoors in areas where wildland fire smoke may be
20 present.

21 Safe Work Australia has safety and health recommendations about the hazards of wildland
22 fire smoke under the topic of “working near bushfires” [Safe Work Australia 2024]. The
23 recommendations reference the following two resources:

- 24 • The Australian Fire Danger Ratings System provides information about the risks of
25 bushfires in the local area.
- 26 • The Australian Warning System provides warnings during emergencies such as
27 bushfires.

28 The Safe Work Australia recommendations indicate that a worker has the right
29 to stop unsafe work if facing a serious risk to their health and safety, and they tell their
30 employer when it is unsafe to work. In such situations, the worker must notify the employer
31 as soon as possible and be available to do suitable alternative work to reduce or eliminate
32 the risk of exposure to wildland fire smoke (see Safe Work Australia’s [Working Near
33 Bushfires](#)).

34 The Canadian Centre for Occupational Health and Safety (CCOHS) also provides information
35 about the hazards of wildland fire smoke and the protective measures that employers and
36 workers can take to minimize exposures [CCOHS 2024]. These measures align with the
37 hierarchy of controls for occupational hazards. The CCOHS also recommends that
38 employers and outdoor workers routinely check the Air Quality Health Index (AQHI) or
39 other indicators of smoke levels in the work area. Along with understanding the AQHI,

1 employers are advised to train workers on emergency response procedures and preparing
2 for possible evacuation (see CCOHS's [Health and Safety Report](#)).

3 **1.5 Summary and Discussion**

4 Increases in the size and severity of wildfires, along with an increase in the length of
5 wildfire season have likely resulted in increasing numbers of outdoor workers being
6 exposed to smoke from these fires more frequently and at higher concentrations. These
7 trends are expected to continue due to a buildup of natural fuel, an increase in the size of
8 the wildland-urban interface, and weather conditions that increase the potential for
9 wildfires (e.g., drought, strong surface winds, and higher global temperatures). Over the
10 past 20 years, NIOSH has conducted intramural research and funded extramural research to
11 evaluate and mitigate risks from exposure to wildland fire smoke among outdoor workers,
12 with some emphasis on farmworkers and wildland firefighters. NIOSH plays a critical role in
13 approving respirators that may be used to protect outdoor workers from wildland fire
14 smoke, as well as communicating the benefits and limitations of such personal protective
15 equipment.

16 Many agencies, including NIOSH, have developed public health guidance to better
17 communicate the potential health risks of exposure to wildland fire smoke and mitigate
18 exposures to at-risk populations, including outdoor workers. The rules enacted by the
19 states of California, Oregon, and Washington require employers to take specific actions to
20 protect outdoor workers during wildland fire smoke events based on specific values of the
21 AQI for PM_{2.5}. As noted, the lower category breakpoints of the AQI are directly tied to the
22 NAAQS, which are set at a level to protect the general population with an adequate margin
23 of safety and have not yet been used in regulation to protect workers. In its final wildfire
24 smoke legislative rule analysis, the state of Washington contrasts existing occupational
25 health standards for PNOR with the NAAQS for PM_{2.5}, citing language in the Washington
26 Administrative Code (WAC). This code that “nuisance dusts” have “little adverse effect on
27 the lungs and do not produce significant organic disease or toxic effect ...” [WAC 296-841-
28 099], while noting the large body of evidence of adverse health effects caused or likely to be
29 caused by exposure to PM_{2.5} [L&I 2023]. However, the only difference between the particles
30 that comprise PM_{2.5} regulated by EPA and PNOR regulated by the state of Washington is the
31 size fraction. PM_{2.5} includes particles that are 2.5 µm in diameter or smaller, and PNOR
32 includes particles that are 4.0 µm in diameter or smaller. Neither agency requires a
33 component analysis [Air contaminants 2017; EPA 2024b]. Further, the health effects of
34 exposure to particulate matter have been extensively studied and characterized, with
35 studies of PM_{2.5} dominating the literature over the past 25 years [EPA 2022, 2024c;
36 Landrigan et al. 2018]. Finally, using PM_{2.5} as a metric of exposure to wildland fire smoke
37 has an advantage in protecting outdoor workers: an existing national air quality monitoring
38 and reporting network.

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Chapter 2: Sources, Population, and Exposures

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Key Chapter Takeaways

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- Smoke from wildland and wildland-urban interface fires contains a complex mixture of hazardous chemicals.
- Outdoor workers who work outside of physical structures encompass a wide range of occupations, including farmworkers and construction, extraction, transportation, maintenance, and forestry workers.
- Exposures to wildland fire smoke can occur through inhalation, dermal absorption, or ingestion.
- Outdoor workers may experience occupational health inequities linked to social, economic, or environmental disadvantages that place some individuals and groups at greater risk of exposure.

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The hazards experienced by outdoor working populations are the focus of this hazard review. The characteristics of the sources, population, and exposures are fundamental to identifying these hazards. This chapter discusses several topics necessary to identify and better understand the hazards associated with occupational exposures to wildland fire smoke, including the chemical composition of wildland fire smoke (Section 2.1), the types and numbers of outdoor workers exposed (Section 2.2), the routes of exposure (Section 2.3), and health equity considerations (Section 2.4).

2.1 Chemical and Physical Properties of the Smoke

2.1.1 Composition of Wildland Fire Smoke

Smoke from wildland fires is a complex mixture of several gas- and particulate-phase chemicals, many of which are toxic air pollutants. The chemical composition of wildland fire smoke strongly depends on the characteristics of combustible materials that fuel the fire in the wildland and at the wildland-urban interface (WUI). WUI fires occur at the intersection of human development zones and undeveloped wildland vegetation and often involve human-made urban materials such as building materials, vinyl and plastic materials, furniture, vehicles, electronics, and other human-made structures. Measurements of primary combustion products from wildland as well as WUI fires have shown that the emission factors of most volatile organic compounds (VOCs) and some polycyclic aromatic hydrocarbons (PAHs) were greater in the WUI fires compared with the wildland fires containing natural fuels [Jaffe et al. 2020].

Table 2-1 shows various classes of pollutants found in fresh emissions from the combustion of biomass, natural vegetation, and various human-made materials found in the WUI zone. Some of these pollutants have also been measured in regional urban air a considerable distance from wildland fires. Some species undergo chemical transformation with time as they are transported away from the fires, as discussed below.

Table 2–1. Primary pollutants emitted from combustible natural and human-made materials serving as fuel for wildland and WUI fires. Pollutants were measured in fresh emissions close to the fire and/or in regional urban air downstream of fires. Adapted from NAS [2022]

Primary pollutant	Near the fire source*							Regional urban air [‡]
	Natural wildland vegetation or biomass	Insulation (polyurethane/ phenolic/ polystyrene foam, glass wool) [†]	Vinyl building products (siding or windows) [†]	Upholstery and furniture [†]	Flooring (polyamide/ vinyl) [†]	Vehicle (battery, plastics, rubber, fluids, rubber, upholstery, fuel) [†]	Acrylic clothing [†]	
Particulate matter (PM)	✓	✓	✓	✓	✓	✓	✓	✓
Hydrogen cyanide (HCN)	✓	✓	•	✓	•	✓	✓	✓
Carbon monoxide (CO)	✓	✓	✓	•	•	✓	✓	•
Nitric oxide (NO)	✓	✓	•	✓	•	✓	✓	✓
Nitrogen dioxide (NO ₂)	✓	✓	•	✓	•	✓	✓	✓
Sulfur dioxide (SO ₂)	✓	•	•	•	•	✓	•	✓
Ammonia (NH ₃)	•	✓	•	✓	•	•	✓	✓
Halides	✓	✓	✓	•	✓	•	•	✓
Hydrochloric acid (HCl)	✓	✓	✓	✓	✓	✓	•	✓
Phosphoric acid (H ₃ PO ₄)	•	✓	•	✓	•	•	•	✓
Polycyclic aromatic hydrocarbons (PAHs)	✓	✓	•	•	•	✓	•	✓

Primary pollutant	Natural wildland vegetation or biomass	Insulation (polyurethane/ phenolic/ polystyrene foam, glass wool) [†]	Vinyl building products (siding or windows) [†]	Upholstery and furniture [†]	Flooring (polyamide/ vinyl) [†]	Vehicle (battery, plastics, rubber, fluids, rubber, upholstery, fuel) [†]	Acrylic clothing [†]	Regional urban air [‡]
Volatile organic compounds (VOCs)	✓	✓	•	✓	•	✓	✓	✓
Semivolatile organic compounds (SVOCs)	✓	✓	•	✓	•	✓	✓	✓
Dioxins	•	✓	✓	✓	•	✓	•	•
Dibenzofuran	•	✓	✓	✓	•	✓	•	•
Acrolein	✓	✓	•	•	•	✓	•	✓
Formaldehyde	✓	✓	•	•	•	✓	•	✓
Acetaldehyde	✓	•	•	•	•	•	•	✓
Iscocyanates	•	✓	•	✓	•	✓	✓	•
Benzene, toluene	✓	•	•	✓	•	•	•	✓
Particulate metals	•	•	•	•	•	✓	•	✓

1 * Chemicals found in fresh emissions at or near the fire source (near-field scale in Figure 2–1).

2 † Combustible materials present in typical WUI settings.

3 ‡ Chemical, emitted from the fires, but found in regional outdoor air far away from the fires (Figure 2–1).

2.1.1.1 Gas-phase Pollutants

CO₂ constitutes the largest component by mass of emissions from wildland vegetation and biomass fires, making up approximately 80% of the fraction, followed by ~12% CO, ~3% VOCs, ~1.6% PM_{2.5}, and ~0.4% CH₄ [Akagi et al. 2011; Andreae 2019; Jaffe et al. 2020]. The composition is highly variable depending on the nature of vegetation, combustion conditions, geographical region, and meteorological conditions [Jaffe et al. 2020]. Non-carbonaceous gas-phase emissions from the combustion of biomass vegetation include inorganic species such as NO_x, HCN, NH₃, and HONO. Oxidized species such as CO₂, nitrogen oxides (NO_x), nitrous acid (HONO), and sulfur oxides (SO_x) are generally formed during *flaming* conditions of the fire, whereas the reduced species such as CO, NH₃, CH₄, and HCN are formed during *smoldering* conditions of the fire.

Both flaming and smoldering states of fire are associated with the emission of many VOC species (>500), many of which have adverse health effects [Hatch et al. 2017]. The mixture of VOCs from the smoke also tends to be highly reactive with the hydroxyl (OH) radical [Jaffe et al. 2020], and the mixture is more reactive than typical emissions in industrial atmospheres. This leads to the formation of secondary organic aerosol species and ozone (O₃) in the ambient atmospheres containing wildfire smoke [Xu et al. 2021].

About 80% of the VOCs are unsaturated compounds with varying degrees of volatilities, oxygenation, heteroatoms (N, F, S, Cl, Br, I), and functional groups (e.g., ketones and other carbonyls, alcohols), with about 60% of the VOCs being oxygenated [Jaffe et al. 2020]. The most common oxygenated VOCs emitted from typical wildland vegetation in the United States are acetaldehyde, acetic acid, formaldehyde, formic acid, and methanol. These gas-phase species have been measured close to fires (near-field) as well as at the regional scale shown in Figure 2–1 [Jaffe et al. 2020; O’Dell et al. 2020; Rice et al. 2023]. Levoglucosan and phenolic compounds such as cresols and guaiacol are also commonly found in fresh wildland fire smoke [Jaffe et al. 2020].

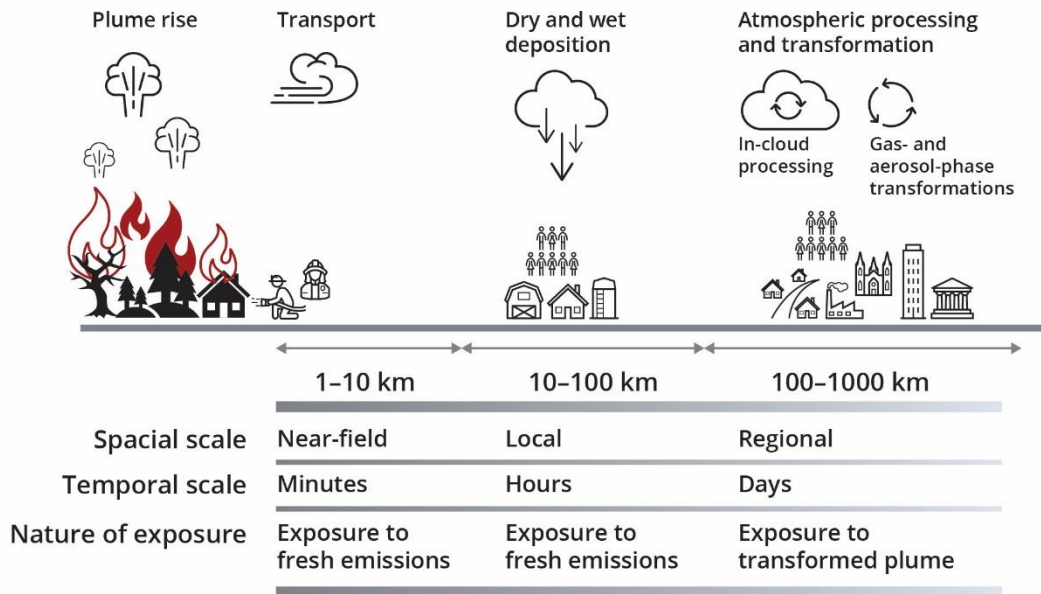


Figure 2–1. Length and time scales associated with transport, deposition, atmospheric transformation of smoke plume and their implications to nature of human exposure [adapted from NAS 2022]

Sekimoto et al. [2018] noted that low-volatility compounds, oxygenates, furans, and ammonia are formed during low-temperature pyrolysis, while emissions from high-temperature pyrolysis often led to the enrichment of aliphatic hydrocarbons, HONO, isocyanic acid (HNCO), PAHs, and HCN.

2.1.1.2 Particulate-phase Pollutants

Emissions of particulate species from wildland fires are subjected to large variations in particle size, morphology, chemical composition, and volatility, all of which determine their effect on exposure concentrations and human health. Smoke particles from the fire are typically in the sub-micrometer range. The particle diameter of aged wildland fire smoke measured by Laing et al. [2016] at a central Oregon ground location (at a regional scale as shown in Figure 2–1) was typically small, with median diameters in the range of approximately 50–200 nm [Jaffe et al. 2020; Laing et al. 2016]. Number-weighted size distributions measured by Laing et al. [2016] ranged from unimodal to bimodal and had geometric mean diameters ranging from 138 to 229 nm (geometric standard deviation ranging from 1.53 to 1.89). These size distribution measurements were consistent with those reported by Janhäll et al. [2010] for aged smoke. The physical characteristics of the smoke particles may vary from fire to fire and depend on the fuel type, fuel moisture content, fire conditions, temperature, meteorological conditions, and geography.

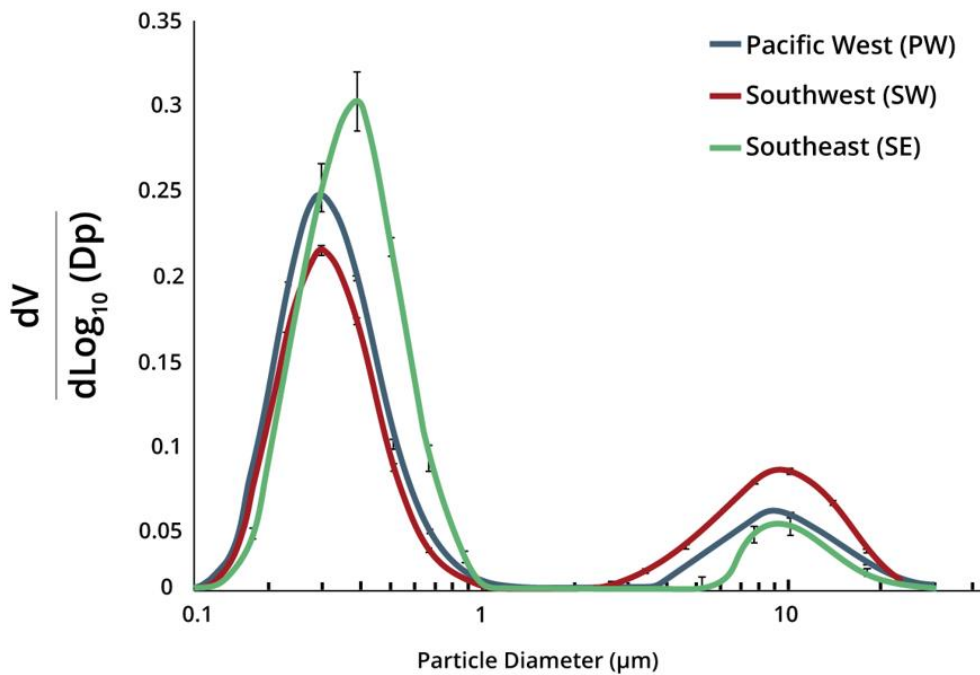


Figure 2–2. Normalized volume-weighted diameter distribution of smoke particles measured by Bian et al. [2020] in the urban air on smoke-impacted days in the Pacific West (PW), Southwest (SW), and Southeast (SE) regions of the United States

Recently, Bian et al. [2020] estimated the normalized size distribution of smoke particles in the urban air of various regions in the United States (measurements conducted at regional and continental spatial scales shown in Figure 2–2). They used the differences between the mean volume size distributions for smoke-impacted and nonsmoke-impacted days measured at various ground locations in the United States. The modal diameters of the size distribution were similar for the Pacific West, Southwest, and Southeast regions as shown in Figure 2–2. The mode of sub-micrometer particle diameter distribution was about 300 nm for the Southwest and Southeast, whereas the modal diameter was around 400 nm for the Pacific West. A coarse mode with fewer particles with a modal diameter of around 9–10 µm was also observed in all regions. The difference in the modal diameter of the fine mode was attributed to the difference in combustion behaviors, with more flaming-type combustion conditions of the wildland fires in the Pacific West and Southwest, compared with smoldering flame conditions of fires in the Southeast, as might be associated with prescribed burning [Bian et al. 2020]. Reid et al. [2005] have reported a similar observation that the flaming phase of burning usually produces smaller particles, while the smoldering phase generates larger smoke particles. The predominant fraction of the smoke particles, by number and mass, are in the sub-micrometer diameter range.

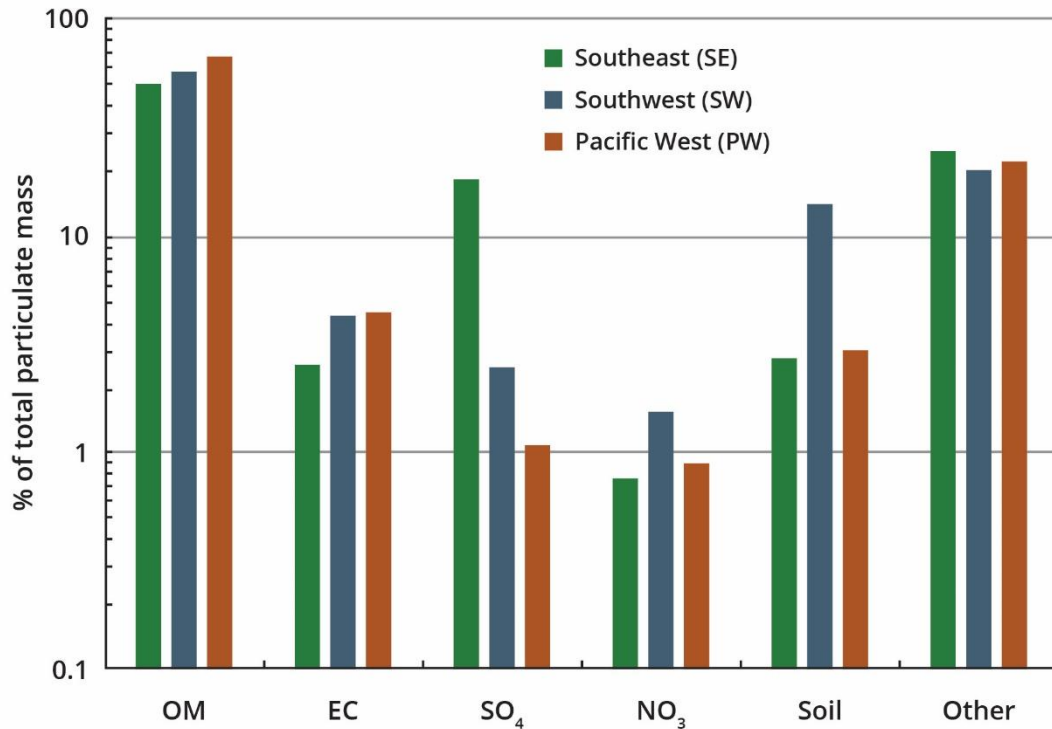


Figure 2–3. Abundance (on y-axis) of organic matter (OM), elemental carbon (EC), sulfates (SO₄), nitrates (NO₃), soil, and other components as a fraction of total particulate mass in urban air of various regions of the United States measured by Bian et al. [2020]. Organic matter was the largest component of PM

PM from smoke particles in regional urban air primarily consists of organic matter (OM), constituting between 50% and 75% of the total mass, along with 3%–5% elemental carbon (EC), 1%–15% SO₄, 0.8%–1.5% NO₃, with the remaining fraction made of inorganic ions (such as K and Cl) and trace metals [Bian et al. 2020; Jaffe et al. 2020]. Bian et al. [2020] reported OM content to be the largest component of regional PM_{2.5} smoke across all regions in the United States (Figure 2–3), accounting for 68% in the Pacific West, 57% in the Southwest, and 48% in the Southeast regions. However, reliable quantification of particle composition remains limited, with many studies omitting either organic or inorganic fractions. Measuring the representative chemical composition of wildland and WUI smoke can be challenging, especially at near-field scale (Figure 2–1), because of the range of volatilities of the gas- and particulate-phase species that can undergo condensation or evaporation, depending on sampling conditions. According to Hatch et al. [2018], about 40% of the particulate mass may be lost because of the evaporation of semi volatile compounds. While toxic metals are present in PM at extremely low concentrations, they may be present at higher concentrations in emissions from fires occurring near urban areas or contaminated sites, or at the WUI [Jaffe et al. 2020; NAS 2022].

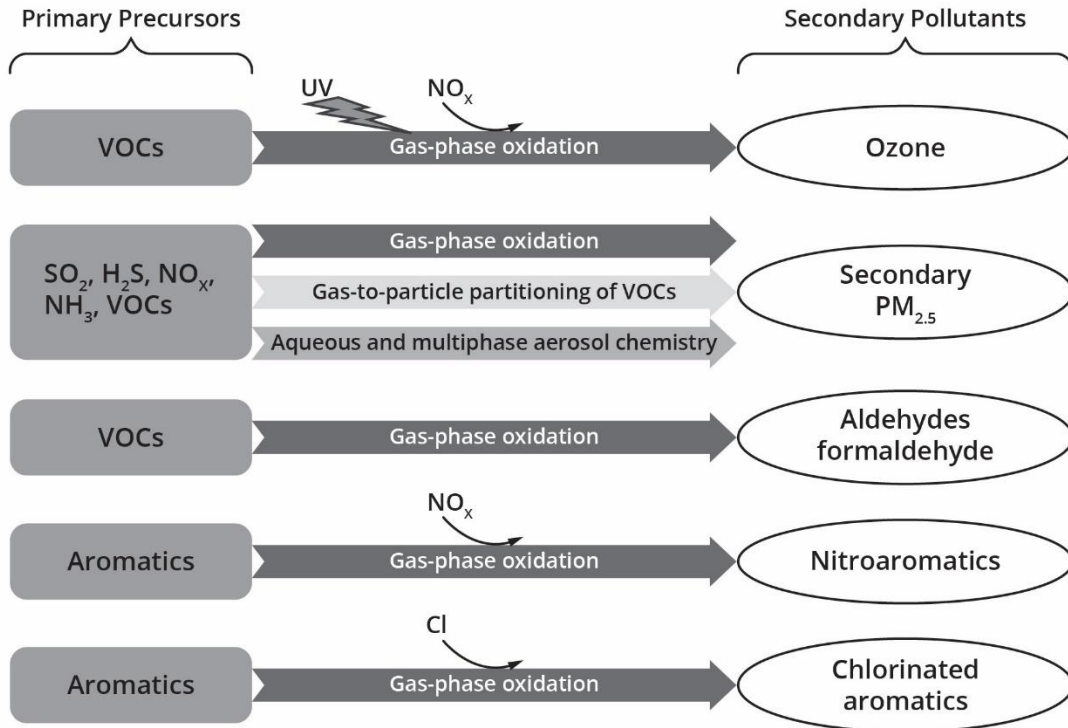
2.1.1.3 Atmospheric Transport and Chemical Transformation

The spatial and temporal scales associated with the transport, transformation of, and resulting exposure to wildland and WUI smoke are key to understanding the emissions of individual wildland fire events (see Figure 2–1). The atmospheric transport characteristics depend on the local or regional meteorological conditions, as well as on the nature of the wildland fire (e.g., prescribed burn, wildland, or WUI fire). The concentration of chemical components of smoke decreases with distance downwind of the fire, through dilution and deposition of larger particles to the ground, and the components undergo chemical and phase changes in the atmosphere [Jaffe et al. 2020; NAS 2022]. Downwind of the immediate fire, at the near-field scale, smoke concentration (in the aerosol phase) is reduced because of the dilution from the clean air, which also reduces the primary organic aerosol content through evaporation and increases the gas-phase VOC concentration. This significantly affects the concentration inhaled by the exposed worker population in the near-field zone.

The level of exposure also depends largely on (1) the nature of the plume rise, (2) whether the emissions are lofted above the mixing layer (a layer of the atmosphere between a stable atmospheric layer and the ground, within which pollutants are mixed by turbulence and diffusion), and (3) whether the emissions are then transported over large distances before descending and mixing near the ground level or are trapped near the ground level in the near-field (see Figure 2–1). The major factor in determining transport altitude is the plume rise or the plume injection height, which in turn depends on the overall heat released by the fire. Meteorological conditions, transport altitude, and location of the plume relative to the mixing layer affect the concentration of the chemicals, aerosol dynamics and gas-particle partitioning, photochemistry, cloud processing, and multiphase chemistry [Jaffe et al. 2020]. Ground-level ozone and oxygenated aerosol can be produced through photochemical oxidation and secondary chemistry in a wildland fire plume in the presence of other anthropogenic pollutants in the atmosphere downwind of the fire [NAS 2022].

The concentration of primary species (particularly the hazardous air pollutants) from the fresh smoke can decrease through several chemical and physical processes that take place on timescales of minutes to days as the smoke is transported and transformed downwind. Conversely, the formation of new secondary species can increase with toxic potential through atmospheric processing (see Figure 2–4). In sharp contrast to anthropogenic sources involving near-complete combustion of fuel, such as internal combustion engines, wildland and WUI fires are distinct as an emission source because of the prevalence of incomplete combustion and thermal degradation of oxygenated fuel components such as cellulose. This leads to the formation of water-soluble oxygenated species such as HONO, HNO₃, HCl, formaldehyde, glycolaldehyde, acetic acid, phenols, furfurals, isocyanic acid, and amines [NAS 2022]. As noted earlier, measurements have suggested that nearly 60% of the measured VOCs from wildland fire fuels are oxygenated [Gilman et al. 2015].

Several factors affect the atmospheric fate of water-soluble compounds in the smoke, which include competitive kinetics of various gas-phase photochemical reactions, reactive uptake of water-soluble compounds in clouds and liquid aerosol particles, and removal by wet deposition [NAS 2022].



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Figure 2–4. Secondary gas-phase and particulate-phase species formed downstream of wildland and WUI fires

Combustion of human-made materials in WUI fires introduces several types of compounds into the smoke, including reactive halogenated compounds (HCl, HBr, HF), dioxins, phenols from the degradation of polymers, aldehydes, nitrogen-containing organics such as isocyanates, brominated and fluorinated organics, and metals. These species could alter the rate of chemical transformations in WUI plumes, changing the lifetime of primary smoke species with toxic potential and the formation and abundance of secondary species with toxic potential.

Oxidation of primary organic precursors can also produce secondary species with toxic potential. Elevated concentrations of reactive chlorine and nitrogen, transition metals, and OH radical are likely present in wildland and WUI plumes and will react with unsaturated organic compounds to form more oxygenated, chlorinated, and nitrated products that may have increased toxicity [NAS 2022]. Figure 2–4 shows various primary precursors that lead to secondary pollutants in the presence of UV and reactive nitrogen, oxygen, halogen, and OH species.

2.1.2 Air Pollutants Emitted During Wildland Fires

In addition to the six criteria air pollutants discussed in Chapter 1, EPA is required to regulate 188 hazardous air pollutants (HAPs), under the Clean Air Act, that pose a risk of serious health effects [EPA 2023a]. Even though anthropogenic emissions of criteria air pollutants and HAPs have declined for decades because of air quality management, biogenic emissions from the increasing frequency of wildland fires may counter these reductions [Jerrett et al. 2022; Sarangi et al. 2023]. Wildland fire emissions are complex mixtures of organic and inorganic compounds in the gaseous and particulate phases, including the criteria air pollutants and many HAPs such as VOCs, PAHs, metals and metalloids, inorganic acids, dioxins, and polychlorinated biphenyls (PCBs) [EPA 2023b; NAS 2022]. The following section describes wildland fire emissions of criteria air pollutants and HAPs reported in the literature. For a summary of the criteria air pollutants and HAPs from the wildland and WUI fires, see Tables 2–2 and 2–3.

2.1.2.1 Criteria Air Pollutants

Carbon Monoxide

Carbon monoxide (CO) is a ubiquitous air pollutant formed from the incomplete combustion of carbon in fuel and is considered one of the most common acute hazards (asphyxiant) to firefighters and other personnel within a downwind near-field scale (see Figure 2–1) [Adetona et al. 2016; Miranda et al. 2012; NAS 2022]. Generally, time-weighted average (TWA) exposures of firefighters and associated personnel to CO during wildland fires do not exceed OSHA permissible exposure limits (PELs) or NIOSH recommended exposure limits (RELs) [Adetona et al. 2016]. During some wildland fires, CO concentrations have exceeded the OSHA PEL of 50 ppm and the NIOSH REL ceiling limit of 200 ppm [Adetona et al. 2016]. Exposures to the public on a local or regional scale (see Figure 2–1) are typically low because of oxidation of CO and air dilution during downwind transport [Adetona et al. 2016; NAS 2022]. Recently, Lill et al. [2022] reported that CO concentrations were significantly greater on smoke-impacted days (265 ppb) compared with non-smoke-impacted days (150 ppb) in Boise, Idaho, during wildland fires in the summer of 2018 on a local and regional spatial scale. This finding was consistent with a study by Lindaas et al. [2017] that observed significant increases of CO in the Colorado Front Range during the summer of 2015 when aged smoke plumes passed through the region.

CO also serves as an important indicator species for the oxidation of hydrocarbons and chlorinated hydrocarbons during wildland fires [NAS 2022] and has been used as a marker species to identify smoke-impacted regions [Jaffe et al. 2022]. Approximately 80% of emissions by mass from biomass wildland fires are CO₂, and CO constitutes the largest portion (~60%) of the non-CO₂ emissions [Akagi et al. 2011; Andreae et al. 2019; Jaffe et al. 2020]. Oxidation of CO to CO₂ by photochemically produced OH radicals is inhibited by competitive oxidative reactions with other organic compounds present during wildland fires, including formaldehyde and methane [NAS 2022]. In addition, the presence of halogens (e.g., chlorine and bromine) and halogen-containing species (e.g., chloromethane)

1 from the combustion of human-made materials further inhibits CO oxidation by quenching
2 combustion reactions and by competitive OH oxidative reactions.

3 Verma et al. [2009b] reported that 24-hr mean CO concentrations on a local scale increased
4 nearly three-fold in Los Angeles during the October 2007 wildland fires in Southern
5 California.

6 Occupational exposure assessment of firefighters and other personnel during wildland
7 firefighting operations offers the most robust studies for near-field exposures to CO and
8 other pollutants. Gaughan et al. [2014] monitored 17 firefighters with mixed job tasks
9 during the 2006 Red Eagle Fire in Montana and did not observe CO concentrations
10 exceeding the NIOSH 10-hr REL of 35 ppm. However, nine samples did exceed the NIOSH
11 ceiling limit of 200 ppm [Gaughan et al. 2014]. Semmens et al. [2021] reported that only
12 1.6% of 246 wildland firefighters who were monitored from 2015 to 2017 in the western
13 and southeastern United States had CO exposures exceeding the ACGIH 8-hr threshold limit
14 value (TLV) of 25 ppm.

15 **Nitrogen Oxides and Sulfur Oxides**

16 Vegetative biomass combustion is the primary source of nitrogen oxides (NO_x) in the air,
17 with 90%–95% emitted as nitric oxide (NO) and 5%–10% emitted as nitrogen dioxide
18 (NO₂) [NAS 2022]. NO_x emissions are also enhanced by higher modified combustion
19 efficiencies of wildland fires [Xu et al. 2021]. NO is rapidly oxidized to NO₂, which
20 contributes to the formation of smog and acid rain [NAS 2022]. Oxidation of NO_x and SO_x
21 may produce secondary species with toxic potential (e.g., nitrophenols, nitro- and dinitro-
22 PAHs, caustic nitrates, and sulfates).

23 Bian et al. [2020] investigated non-smoke versus smoke-impacted days for April through
24 September from 2008–2017 in three U.S. regions (Pacific West, Southeast, and Southwest)
25 and found that NO_x and SO_x concentrations increased on smoke-impacted days. SO_x
26 emissions were higher in the Southeast relative to other regions studied because of
27 vegetation and resuspension of SO_x deposited in soil from fossil fuel combustion.
28 Conversely, NO_x emissions were higher in the Pacific West where flaming fires were more
29 common [Bian et al. 2020].

30 Reactive nitrogen species from the oxidation of NO_x are formed rapidly in wildland fire
31 smoke plumes [Alvarado et al. 2010]. Lindaas et al. [2017] observed ~100% increase in
32 peroxyacetyl nitrate (PAN) and propionyl peroxy nitrite (PPN) in the Colorado Front Range
33 during the summer of 2015 when aged smoke plumes passed through the region. Lill et al.
34 [2022] reported ~40% increase in PAN and PPN concentrations on smoke-impacted days in
35 Boise, ID during wildland fires in the summer of 2018.

36 Multiple studies have reported elevated concentrations of NO_x on a local scale during
37 wildland fire smoke-impacted days. Na and Cocker [2008] reported that NO₂ concentrations
38 were significantly higher in Riverside, CA, approximately 30 km from wildland fires in
39 October 2003 (maximum observed concentration: 87 ppb) than during the week after the
40 fire subsided (maximum observed concentration: 63 ppb). Verma et al. [2009b] reported

1 24-hr mean NO concentrations increased nearly 3-fold in Los Angeles during October 2007
2 southern California wildland fires. Interestingly, NO₂ concentrations were not higher on
3 smoke-impacted days.

4 **Ozone**

5 Ozone (O₃) is a secondary air pollutant that is formed through multiple photochemical
6 reaction pathways involving interactions of ultraviolet (UV) light with precursors emitted
7 during wildland fires such as CO, NO_x, and VOCs (see Figure 2–4). NO_x is considered the
8 limiting precursor for O₃ formation during wildland fires. O₃ formation is enhanced when
9 vegetative biomass smoke containing high concentrations of VOCs is mixed with NO_x-rich
10 smoke emanating from WUI fires [Xu et al. 2021]. Ground-level O₃ concentrations are
11 elevated during dry, summer months because of increased emissions of O₃ precursors from
12 anthropogenic sources (e.g., fossil fuel combustion) and natural sources (e.g., wildland
13 fires).

14 In the intermountain western United States, wildland fires increase summer mean daily
15 maximum 8-hr average (MDA8) O₃ concentrations by an estimated 0.3–1.5 ppbv (parts per
16 billion by volume) and potentially increase by 5–40 ppbv [Dreessen et al. 2016; Gong et al.
17 2017; Lu et al. 2016]. In addition, Lu et al. [2016] reported wildland fires to account for
18 about one third of summer days (during 1989–2010) exceeding the EPA National Ambient
19 Air Quality Standards (NAAQS) MDA8 O₃ concentration of 70 ppbv [EPA 2024b].

20 Wildland fire emissions may lead to increased production of ground-level O₃ on a local and
21 regional scale. Na and Cocker [2008] reported that O₃ concentrations were significantly
22 higher in Riverside, CA, approximately 30 km from the October 2003 wildland fires
23 (maximum observed concentration: 78 ppb) than during the week after the fire subsided
24 (maximum observed concentration: 51 ppb). Lill et al. [2022] reported that mean O₃
25 concentrations (determined by using aircraft measurement in the boundary layer during
26 aircraft ascent and descent) were significantly higher by ~13 ppb on smoke-impacted days
27 in Boise, Idaho, during the summer of 2018 wildland fires, which was consistent with
28 studies of O₃ enhancement on smoke-impacted days from 2006–2017. Conversely, Verma et
29 al. [2009b] did not observe increased levels of O₃ during the October 2007 fires in Southern
30 California.

31 Wentworth et al. [2018] had the opportunity to investigate O₃, VOCs, and PAHs emissions at
32 near-field scale to a large boreal wildland fire in Alberta, Canada, during May 2016.

33 Although O₃ production is commonly observed downwind of wildland fires [Brey and
34 Fischer 2016; Jaffe and Wigder 2012], they did not find an increase in O₃ near the boreal
35 wildland fire [Wentworth et al. 2018]. They note this is consistent with studies suggesting
36 that O₃ production during boreal wildland fires is inhibited by optically thick aerosols
37 hindering photochemical reactions [Verma et al. 2009a] and/or by sequestration of NO_x in
38 cooler climate boreal forests [Alvarado et al. 2010; Wentworth et al. 2018].

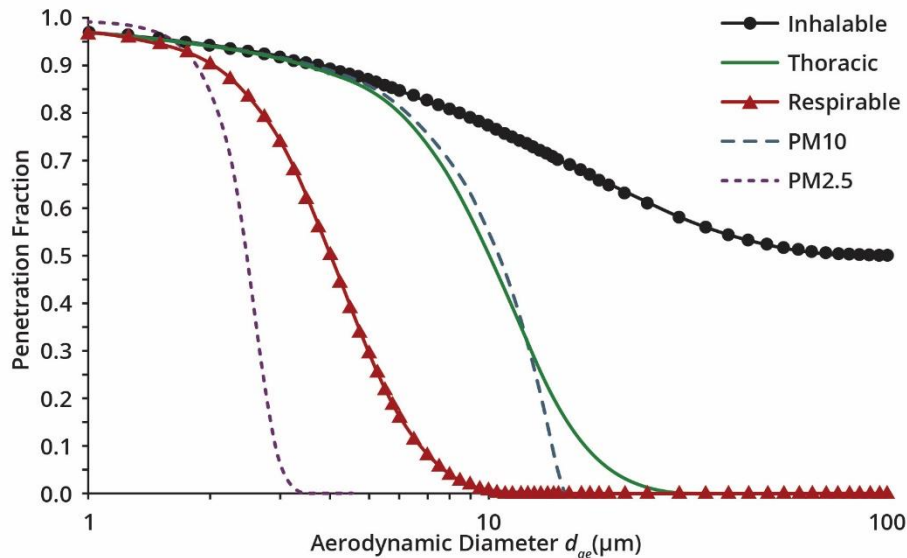
1 **Particulate Matter**

2 **Particle Size-Selective Criteria and Standards**

3 EPA updated its Total Suspended Particulate standards [EPA 1971] to a PM₁₀ standard [EPA
4 1987]. PM₁₀ better captured anthropogenic sources and considered human airway
5 penetration [Miller et al. 1979]. EPA further proposed a smaller particle size fraction and
6 standard (PM_{2.5}) based on strong epidemiological evidence reporting associations between
7 ambient PM and a range of serious adverse human health effects [EPA 1997]. PM_{2.5} (fine)
8 and PM₁₀ (coarse) particles in the atmosphere may be differentiated by source and
9 formation processes, and chemical and physical properties, including behavior in the
10 atmosphere [EPA 1997]. The air quality fractions for PM₁₀ [EPA 1987] and PM_{2.5} [EPA 1997]
11 are shown in Figure 2–5, based on approximations in Hinds [1999].

12 Particle penetration and deposition within the human respiratory system are influenced by
13 aerodynamic particle diameter. The health-based particle-size fractions (inhalable, thoracic,
14 and respirable) determine where particles are capable of penetrating within the human
15 respiratory system [ACGIH 1999]. Inhalable particles are those that may enter the mouth or
16 nose during breathing; thoracic particles pass the larynx and into the conducting airways;
17 and respirable particles may penetrate the unciliated or gas-exchange regions of the human
18 respiratory system [ACGIH 1999; CEN 1993; ISO 1995]. The thoracic fraction is defined as a
19 subfraction of the inhalable fraction, and the respirable fraction is defined as a subfraction
20 of both the inhalable and thoracic fractions. These health-based particle-size fractions are
21 routinely used in occupational exposure sampling for particulate matter.

22 The inhalable fraction is represented by a curve with a penetration of 1.0 at very small
23 particle sizes (less than 1 µm), reduces to 0.58 for 30 µm and retains a penetration of ~0.5
24 for particles up to and including 100 µm diameter. The thoracic fraction is represented by a
25 cumulative lognormal curve with a penetration of 0.5 at 10 µm. The respirable fraction is
26 represented by a cumulative lognormal curve with a penetration of 0.5 at 4 µm. These
27 health-based particle penetration curves and their mathematical approximations are
28 discussed in ACGIH [1999], CEN [1993], ISO [1995], Hinds [1999], and Vincent [2007]. They
29 are also shown in Figure 2–5.



1
2 **Figure 2–5. Reproduced from NIOSH [2024b]. The inhalable, thoracic, respirable,**
3 **PM₁₀, and PM_{2.5} particle penetration curves**

4 Consideration was given to the thoracic fraction (human airway particle penetration
5 passing the larynx) in the development of the PM₁₀ air quality standard [Miller et al. 1979],
6 but the PM_{2.5} standard was not based on the long-established respirable aerosol criterion
7 [Vincent 2012]. As noted, the PM_{2.5} size fraction was focused from a particle source rather
8 than a human airway penetration (sink) perspective. PM_{2.5} more closely captures PM
9 components contributed from fixed and mobile combustion sources and excludes coarser
10 particles in the atmosphere that are mechanically generated.

11 The thoracic and PM₁₀ size fractions are in close agreement (up to ~13 μm diameter), but
12 with a sharper cut-off for PM₁₀. The PM_{2.5} and respirable-size fractions are in close
13 agreement up to ~2 μm particle diameter, but the PM_{2.5} fraction curve drops sharply after
14 ~2 μm . The respirable fraction curve has 0.5 penetration at 4 μm ; whereas the PM_{2.5}
15 fraction curve has zero penetration. As noted in Figure 2–2 and in Bian et al. [2020], the
16 primary mode observed in the volume-weighted particle size distribution from wildland
17 fire smoke at the regional scale is predominantly sub-1 μm . It suggests that particulate mass
18 in PM_{2.5} and the respirable particle fractions (despite differences greater than 2 μm
19 aerodynamic diameter) of the PM on smoke-impacted days will likely be comparable (see
20 Figure 2–5). A smaller second mode in the volume-weighted particle-size distribution was
21 also observed (see Figure 2–2), centered ~9–10 μm , though it will not contribute to the
22 PM_{2.5} fraction. This secondary mode could, however, be accounted for in the PM₁₀, thoracic,
23 or inhalable size fractions. PM from wildland fire smoke may not be completely captured by
24 either the PM_{2.5} or respirable size fractions.

1 **PM from Wildland and WUI fires**

2 Wildland fires are estimated to account for up to 25% of total PM_{2.5} concentrations across
3 the United States and account for nearly 50% of total PM_{2.5} in the western United States
4 [Burke et al. 2021; Zhang et al. 2023]. Sarangi et al. [2023] recently estimated that the
5 summertime emission contribution from wildland fires to total surface-level PM_{2.5} is
6 expected to nearly double by 2050 in North America.

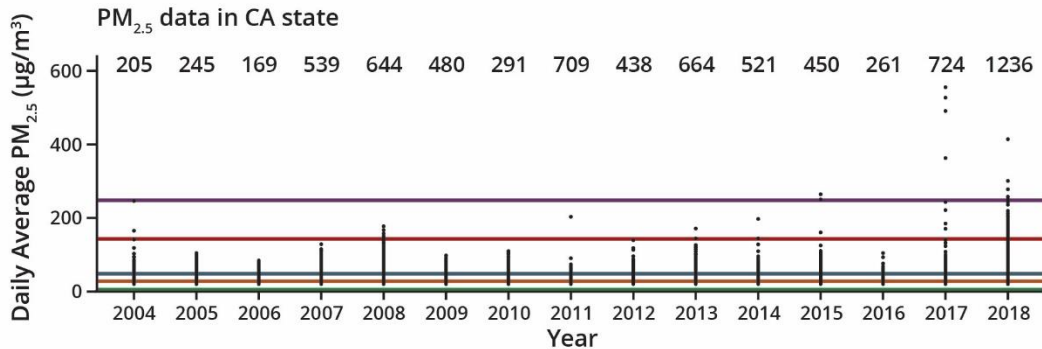
7 PM_{2.5} is a reliable surrogate for wildfire smoke exposure. In Washington state, peak PM_{2.5}
8 concentrations normally occur in July–September during wildland fires and high heat
9 indexes (>85 °F) when agricultural worker population counts are the highest [Austin et al.
10 2021]. Elevated PM_{2.5} concentrations are also reliable indicators of elevated HAPs on
11 smoke-impacted days [O’Dell et al. 2020]. Rice et al. [2023] reported that cadmium and
12 seven VOCs (acetaldehyde, acrolein, benzene, 1,3-butadiene, carbon tetrachloride,
13 formaldehyde, trichloroethylene) were significantly correlated with total PM_{2.5} on smoke-
14 impacted days.

15 Childs et al. [2022] estimated that from 2006–2020, wildland fire smoke contributions to
16 average daily PM_{2.5} increased up to 5 µg/m³ in the western United States. They estimate that
17 nearly 16.4 million people from 2016–2020 experienced at least 1 day of smoke PM_{2.5}
18 greater than 50 µg/m³, nearly 8 million people experienced at least 1 day of smoke PM_{2.5}
19 greater than 100 µg/m³, and nearly 1.5 million people experienced at least 1 day of smoke
20 PM_{2.5} greater than 200 µg/m³ [Childs et al. 2022]. The states with the greatest increase in
21 days with extreme smoke PM_{2.5} (>200 µg/m³) were California, Idaho, Montana, Nevada,
22 Oregon, and Washington [Childs et al. 2022].

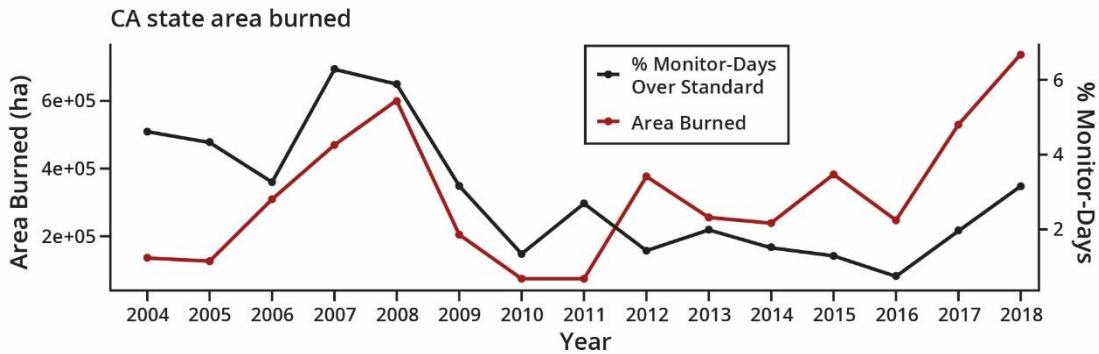
23 Jaffe et al. [2020] explored the increasing importance of seasonal wildland fire PM_{2.5}
24 emissions on U.S. air quality versus PM_{2.5} emissions during prescribed fires. In 2017,
25 wildland fires burned more than 4 million hectares. The top five states for annual area
26 burned are in the western United States (California, Idaho, Montana, Nevada, and Oregon).
27 Conversely, prescribed fires burned more than 5 million hectares. The top five states for
28 annual area burned are in the central and southeastern United States. (Alabama, Florida,
29 Georgia, Oklahoma, and Texas). Yet, the maximum 24-hr mean PM_{2.5} concentrations
30 observed during prescribed fires (29–49 µg/m³) were an order of magnitude lower than the
31 maximum 24-hr mean PM_{2.5} concentrations during wildland fires (125–550 µg/m³) [Jaffe et
32 al. 2020]. They surmise that fuel type and burning conditions may explain the differences in
33 PM_{2.5} emissions, as well as the prevalence of prescribed burns during winter and spring
34 seasons. The authors also note that because of the locations of EPA Air Quality System
35 (AQS) monitors in the western United States relative to many wildland fires, the reported
36 PM_{2.5} concentrations may have underestimated wildland fire PM_{2.5} emissions.

37 Figure 2–6 illustrates the relationship between the annual area burned by wildland fires
38 and daily PM_{2.5} concentration measured using AQS monitor data in California from 2004–
39 2018. The percentage of monitor-days exceeding the EPA NAAQS standard of 35 µg/m³
40 [EPA 2024b] occurred in 2007, 2008, 2017, and 2018, when fire activity was highest. During
41 this time, a significant number of monitor-days exceeded the EPA Air Quality Index (AQI)

1 thresholds for Unhealthy and Very Unhealthy 24-hr mean PM_{2.5} concentrations [Jaffe et al.
 2 2020].



3



4

5 **Figure 2–6. PM_{2.5} data and area burned in California**

6 (Top) Box and whisker plots of all daily PM_{2.5} concentrations by year for air quality monitors in California.
 7 The numbers at the top of the panel show the total number of monitor-days above the daily PM_{2.5}
 8 standard (35 µg/m³). Colored horizontal lines show the six AQI cut points: Good, <12 µg/m³; Moderate,
 9 <35.4 µg/m³; Unhealthy for Sensitive Groups, <55.4 µg/m³; Unhealthy, <150.4 µg/m³; Very Unhealthy,
 10 <250.4 µg/m³; Hazardous, >250.4 µg/m³.

11 (Bottom) Annual area burned (left y-axis) and percentage of all monitor-days that exceeded the daily
 12 PM_{2.5} standard (right y-axis). All PM_{2.5} data from the EPA AQS system are included (regulatory and non-
 13 regulatory). Sources: Burned area for each state is from the National Interagency Fire Center, and PM_{2.5}
 14 data are from the EPA AQS database. Reprinted with permission from Taylor and Francis,
 15 <https://doi.org/10.1080/10962247.2020.1749731>; Copyright: 2020 Air and Waste Management Association.

16

17 Multiple studies have investigated PM_{2.5} exposures at wildland firefighter incident
 18 command posts (also known as base camps), which are analogous to communities and
 19 workers exposed to wildfire smoke on a near-field scale (see Figure 2–1). McNamara et al.
 20 [2012] measured PM_{2.5} concentrations at three wildland firefighter incident command posts
 21 in California, Oregon, and Washington during the 2009 fire season and reported that PM_{2.5}
 22 concentrations were consistently higher overnight. The daytime mean PM_{2.5} concentrations
 23 ranged from 4.7–37.1 µg/m³, while the nighttime average PM_{2.5} concentrations ranged from
 24 11.0–44.0 µg/m³. Most 24-hr mean PM_{2.5} concentrations observed were below the EPA

1 NAAQS standard of 35 $\mu\text{g}/\text{m}^3$ [McNamara et al. 2012]. Navarro et al. [2019] collected area
2 air samples at wildland firefighter incident command posts to monitor $\text{PM}_{2.5}$ during the
3 2015 Willow Fire in California. They reported that $\text{PM}_{2.5}$ concentrations were highest during
4 the day, with daytime concentrations ranging from 19–105 $\mu\text{g}/\text{m}^3$ and nighttime
5 concentrations ranging from 7–24 $\mu\text{g}/\text{m}^3$ [Navarro et al. 2019]. The authors note that the
6 24-hr mean $\text{PM}_{2.5}$ was 23 $\mu\text{g}/\text{m}^3$, which was within the range of the daily mean
7 concentrations reported by McNamara et al. [2012]. Navarro et al. [2019] considered
8 exposures to personnel at incident command posts as representative of exposures to
9 communities at a near-field scale. The 24-hr mean $\text{PM}_{2.5}$ concentrations, when compared
10 with the EPA AQI (see caption in Figure 2–6), were considered Moderate for 5 days,
11 Unhealthy for Sensitive Groups for 4 days, and Unhealthy for 2 days [Navarro et al. 2019].

12 **Lead**

13 Lead (Pb) in the $\text{PM}_{10-2.5}$ fraction (which is a coarse particle subfraction within the thoracic
14 fraction) is primarily emitted from the combustion of human-made materials during WUI
15 fires such as vehicle batteries and legacy materials in structures (e.g., paint and pipes) [NAS
16 2022]. See Table 2–1 for more information.

17 Boaggio et al. [2022] used $\text{PM}_{2.5}$ measurements in California from 2006–2018 to identify
18 metals and other inorganic chemicals associated with elevated $\text{PM}_{2.5}$ in smoke plumes. They
19 observed lead concentrations in total $\text{PM}_{2.5}$ were elevated during some wildland fires but
20 concluded that the average concentrations did not significantly increase across all smoke-
21 impacted days during the 13-year study [Boaggio et al. 2022]. They also observed that the
22 most destructive fires to structures and vehicles, the October 2017 and Camp 2018 Fires,
23 produced the highest concentrations of particulate lead [Boaggio et al. 2022].

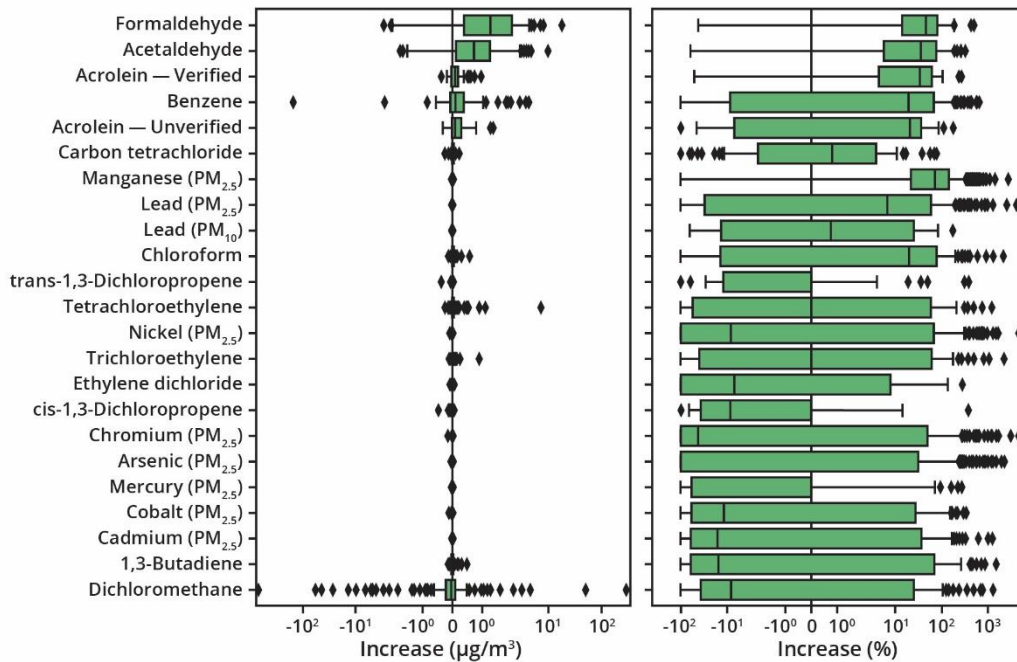
24 Elevated concentrations of toxic metals, including lead, were measured in the regional
25 urban air during the Camp 2018 Fire in California [CARB 2021]. High concentrations of
26 particulate lead (in $\text{PM}_{2.5}$), about 4–50 times higher than those on nonsmoke-impacted days,
27 were measured at the San Jose and Sacramento-Del Paso sites during the fires.

28 Rice et al. [2023] reported that particulate lead measured in $\text{PM}_{2.5}$ and PM_{10} did not
29 significantly increase on smoke-impacted days from 2006–2020 in the western United
30 States (see Figure 2–7).

31 **2.1.2.2 Hazardous Air Pollutants (HAPs)**

32 As noted earlier, HAPs are chemicals known or suspected to cause cancer or other serious
33 health effects that are regulated by the EPA [EPA 2023a]. In a recent study, Rice et al. [2023]
34 investigated elevated concentrations of individual HAPs on wildland fire smoke-impacted
35 days compared with non-smoke-impacted days. They examined whether the measured
36 concentrations of HAPs during wildland fire smoke events exceeded the available HAP
37 reference concentrations for acute and chronic human health effects. Their study collated
38 daily average measurements from 309 EPA AQI monitoring stations in the western United
39 States from 2006 to 2020 during fire seasons (April–December) and investigated the
40 influence of four major California fires on HAPs concentrations in San Jose, CA, from

1 2017–2020. Figure 2–7 displays the 21 HAPs surveyed for this study, including 13 VOCs
 2 (acetaldehyde, acrolein, benzene, 1,3-butadiene, carbon tetrachloride, chloroform,
 3 dichloromethane, cis-1,3-dichloropropene, trans-1,3-dichloropropene, ethylene dichloride,
 4 formaldehyde, tetrachloroethylene, and trichloroethylene) and 8 metals and metalloids
 5 (arsenic, cadmium, chromium, cobalt, lead, manganese, mercury, and nickel). The findings
 6 from this study are discussed in the following sections where appropriate.



7
 8 **Figure 2–7. Box-and-whisker plots of absolute and percentage mean differences**
 9 **between smoke-impacted and nonsmoke-impacted days at each included U.S. EPA Air**
 10 **Quality System monitoring station per year**

11 The green-shaded boxes show the 25th, 50th, and 75th percentiles of the distribution of station- and year-
 12 specific differences, and whiskers extend 1.5 times the interquartile (25th–75th percentile) range.

13 Reprinted with permission from Rice et al. [2023; DOI: 10.1021/acs.est.3c04153]. Copyright 2023,
 14 American Chemical Society.

15
 16 **2.1.2.3 Volatile Organic Compounds**

17 VOCs constitute the second-largest portion (~15%) of non-CO₂ emissions from biomass
 18 wildland fires [Jaffe et al. 2020]. More than 500 VOCs have been identified in smoke [Hatch
 19 et al. 2017], and it has been documented in laboratory and field studies that VOC emission
 20 factors increase with decreased combustion efficiency [Aurell and Gullett 2013; Sekimoto et
 21 al. 2018]. Although VOCs represent only a fraction of the total emissions, many are
 22 associated with adverse health effects [Jaffe et al. 2020; O’Dell et al. 2020; Rice et al. 2023].
 23 VOCs may also undergo photochemical oxidation in smoke plumes to form secondary
 24 organic aerosols and O₃ [NAS 2022; Xu et al. 2021]. Oxygenated species (e.g., acetaldehyde,

1 acrolein, and formaldehyde) are emitted in the highest amounts during fire events from
2 pyrolysis of biomass, accounting for nearly 70% of emitted VOCs [O'Dell et al. 2020; Permar
3 et al. 2021; Rice et al. 2023; Sekimoto et al. 2018; Xu et al. 2021]. Chlorinated hydrocarbon
4 (e.g., carbon tetrachloride, chloroform, and trichloroethylene) emissions may be higher at
5 the WUI from the combustion of anthropogenic sources [NAS 2022; Rice et al. 2023].

6 The age of the smoke may also affect the concentrations of the VOCs as they are transported
7 downwind. O'Dell et al. [2020] used measurements obtained during Wildfire Experiment for
8 Cloud Chemistry, Aerosol Absorption, and Nitrogen (WE-CAN) experiments to estimate
9 variation in VOC concentrations as a function of smoke plume age [O'Dell et al. 2020]. In
10 young smoke (<1 day of aging, representing near-field to local-level exposures in
11 Figure 2–1), they noted the concentration of formaldehyde, acrolein, and benzene exceeded
12 the California EPA reference exposure concentrations for no adverse effects. The
13 concentrations exceeded the reference exposure limits for fresh concentrated plumes with
14 physical ages as brief as 20 min. They did not find VOCs (classified as HAPs) above the
15 California EPA reference limit in medium (1–3 days) and old (>3 days) smoke. The young
16 physical age noted in this study applies to the near-field exposures, including some adjacent
17 regions within the local scale (Figure 2–1), where the population of outdoor workers could
18 reside. O'Dell et al. [2020] also noted that none of these HAP VOCs exceeded the OSHA
19 short-term and permissible exposure limits.

20 Multiple studies have investigated the effects of wildland fire emissions on VOC
21 concentrations at local and regional spatial scales (Figure 2–1). Na and Cocker [2008]
22 reported acetaldehyde and formaldehyde concentrations in Riverside, CA, approximately 30
23 km downwind of wildland fires in Fall 2003. The maximum observed concentrations during
24 wildland fires were more than two times higher than the following week after the fires
25 subsided, reaching 7.5 ppb for acetaldehyde and 11.2 ppb for formaldehyde [Na and Cocker
26 2008]. Lill et al. [2022] measured 121 VOCs in Boise, Idaho, during the summer of 2018
27 wildland fires and found the mean concentrations of acetaldehyde, acrolein, benzene, and
28 formaldehyde were significantly higher on smoke-impacted days.

29 Rice et al. [2023] reported the median concentrations of acetaldehyde, acrolein, chloroform,
30 formaldehyde, and tetrachloroethylene all significantly increased on smoke-impacted days
31 from 2006–2020 across the western United States (see Figure 2–7). The VOCs with the
32 greatest absolute and percentage median concentration increases were acetaldehyde (0.73
33 $\mu\text{g}/\text{m}^3$ [36%]) and formaldehyde (1.3 $\mu\text{g}/\text{m}^3$ [46%]) [Rice et al. 2023]. In addition, Rice et al.
34 [2023] reported that acetaldehyde, acrolein, and formaldehyde were consistently elevated
35 on smoke-impacted days in San Jose, California, and linked to four major wildland fires from
36 2017–2020. The highest formaldehyde concentrations in San Jose from 2006–2020
37 occurred during the 2018 Camp Fire (11.2 $\mu\text{g}/\text{m}^3$) and the August 2020 Complex Fire (11.4
38 $\mu\text{g}/\text{m}^3$) [Rice et al. 2023]. Concentrations of 1,3-butadiene, benzene, carbon tetrachloride,
39 dichloromethane, and ethylene dichloride were not significantly elevated on smoke-
40 impacted days. It has been reported that 1,3-butadiene and benzene have higher
41 concentrations in winter in the absence of smoke. In addition, trace concentrations and

1 short atmospheric lifetimes (e.g., benzene) may present difficulties in monitoring some
2 VOCs further from the emission sources [Rice et al. 2023].

3 Few studies have researched VOC air concentrations affected by wildland fire smoke at a
4 near-field scale. Wentworth et al. [2018] investigated 65 total VOCs measured by air quality
5 monitoring stations in Alberta, Canada, in proximity (<10 km) to a large boreal wildland fire
6 in May 2016. They reported that the daily average of summed VOCs (Σ VOCs) of 63 ppbv on
7 smoke-impacted days (with the highest recorded daily-average Σ VOCs of 112 ppbv) was a
8 minor to moderate increase over the daily-average Σ VOCs of 46 ppbv on nonsmoke-
9 impacted days. The major constituents of the Σ VOCs measured on smoke-impacted days
10 were acetaldehyde, acetone, benzene, butene, formaldehyde, and methanol [Wentworth et
11 al. 2018].

12 **2.1.2.4 Polycyclic Aromatic Hydrocarbons**

13 Polycyclic aromatic hydrocarbons (PAHs) are volatile and semi-volatile organic compounds
14 (SVOCs) that can be released from the combustion of vegetative biomass or combustible
15 human-made materials in structures and vehicles (e.g., insulation, upholstery, carpet,
16 plastics) [Table 2-1].

17 Although PAHs can remain airborne for 5–10 days and transport long distances, higher
18 emissions are commonly associated with incomplete combustion during smoldering fires
19 [NAS 2022]. Wentworth et al. [2018] investigated 23 total PAHs measured in Alberta,
20 Canada, near-field to a large boreal wildland fire in May 2016, finding the daily average of
21 summed PAHs (Σ PAHs) was significantly higher on smoke-impacted days. They observed
22 significant increases in daily-average Σ PAHs on smoke-impacted days by nearly a factor of
23 60, with the daily-average Σ PAHs of 852 ng/m³ compared with the daily-average Σ PAHs of
24 50 ng/m³ on nonsmoke-impacted days [Wentworth et al. 2018]. The highest maximum
25 daily-average Σ PAHs measured on smoke-impacted days was 2,883 ng/m³. Notably,
26 naphthalene accounted for 30%–80% of Σ PAHs measured in this study. When naphthalene
27 was excluded from the Σ PAHs measurement, the daily-average Σ PAHs fell to 399 ng/m³ on
28 smoke-impacted days and 16 ng/m³ on non-smoke-impacted days [Wentworth et al. 2018].

29 Navarro et al. [2019] collected area air samples to measure 17 PAHs at a wildland
30 firefighter incident command post during the 2015 Willow Fire in California. They reported
31 the highest PAHs measured were naphthalene (mean 284 ng/m³; range 80–2,515 ng/m³)
32 and retene (mean 16 ng/m³; range <3–268 ng/m³). They also found that naphthalene and
33 Σ PAH concentrations were higher during the daytime and highest during peak fire activity
34 in the afternoon [Navarro et al. 2019].

35 Fewer studies report PAH concentrations on smoke-impacted days at a local or regional
36 spatial scale. Verma et al. [2009a] found that retene was significantly elevated in Los
37 Angeles during the October 2007 Southern California wildland fires. But they did not find
38 that other PAH concentrations were affected on smoke-impacted days.

2.1.2.5 Metals and Metalloids

Commonly measured inorganic metals and metalloids in smoke-impacted outdoor air downwind of wildland fires include arsenic, cadmium, chromium, cobalt, lead, manganese, mercury, and nickel. Metals and metalloids are emitted at higher concentrations during WUI fires from non-combustible anthropogenic sources such as structures and vehicles [CARB 2021; Jaffe et al. 2020; NAS 2022].

Verma et al. [2009b] reported that magnesium, manganese, and potassium were significantly elevated in Los Angeles during the October 2007 Southern California wildland fires. Boaggio et al. [2022] reported that aluminum, iron, manganese, potassium, and titanium were significantly elevated on smoke-impacted days for 8+ years from 2006–2018 in California during the April–December fire season. They also reported that arsenic, chromium, copper, lead, nickel, and zinc concentrations were episodically elevated over the 13-year study during some fire events, but these increases were not consistent across all smoke-impacted days [Boaggio et al. 2022]. The emissions of trace metals and metalloids also differed based on how destructive the fires were to structures and vehicles. Elevated concentrations of aluminum, iron, manganese, titanium, and zinc were observed during the least destructive June 2008 fires and the 2008 Carr Fire, while elevated concentrations of arsenic, chromium, copper, magnesium, and nickel were observed in the more destructive 2015 fires, October 2017 fires, and the 2018 Camp Fire [Boaggio et al. 2022]. The California Air Resources Board (CARB) compared the air quality on a local and regional scale during the 2018 Camp Fire (a significant WUI fire) with three other large wildland fires in 2018 that mostly burned vegetation. The wildland fires burned more acreage than the Camp Fire but destroyed far fewer buildings. All four fires were connected to increases in metals measured in PM_{2.5}; however, only the Camp Fire resulted in significantly higher concentrations of iron, manganese, and zinc up to 150 miles away from the fire [CARB 2021].

Rice et al. [2023] reported that manganese in PM_{2.5} significantly increased on smoke-impacted days from 2006–2020 in the western United States. The absolute difference of median manganese concentrations (0.0006 µg/m³) on smoke-impacted days had the highest percentage increase (72%) of all metals and metalloids included in the study. However, other metals and metalloids measured in PM_{2.5} by EPA AQS monitoring stations (arsenic, cadmium, chromium, cobalt, mercury, and nickel) did not significantly increase on smoke-impacted days in the western United States from 2006–2020 [Rice et al. 2023]. Metals and metalloids, except for lead, were not reported in PM₁₀ [Rice et al. 2023].

2.1.2.6 Other Hazardous Air Pollutants in WUI fires

The previous sections describe HAPs that are routinely measured in smoke-impacted ambient air or by area sampling during wildland and WUI fires. Other groups of HAPs could be emitted by the combustion of human-made materials (see Table 2–1) at the wildland-urban interface. Hotter and drier weather and an increase in the frequency and duration of droughts (particularly in the western United States), has led to drier soil and vegetation that poses a higher risk of fire ignition [Peterson et al. 2021]. The prevalence and intensity of

1 wildland fire events also increase the probability of these fires coming into contact with
2 urban communities containing contaminated soils or human-made materials and structures
3 (see Table 2–4). This can lead to the emission of HAPs such as polychlorinated biphenyls
4 (PCBs), flame retardants (e.g., tris[1-chloro-2-propyl] phosphate and tris[2-chloroethyl]
5 phosphate), polychlorinated dibenzo-p-dioxins (PCDDs), polychlorinated dibenzofurans
6 (PCDFs), isocyanates, and per- and polyfluoroalkyl substances (PFAS) [NAS 2022]. In
7 addition, volatilization of PCBs, dioxins, and furans from soil and water is accelerated
8 during wildland fires and these chemicals may have lengthy tropospheric half-lives
9 dependent on the substituents or the particle-bound fraction [NAS 2022].

10 Ruokojärvi et al. [2000] measured high concentrations of PAHs (6.4–470 mg/m³), PCBs
11 (0.5–56 µg/m³), and PCDDs and PCDFs (12–160 ng/m³) during simulated house fires.
12 Recently, Fent et al. [2020] collected area air samples during controlled residential fires and
13 measured significant concentrations of flame retardants in most samples while PCDDs and
14 PCDFs were measured in fewer samples at lower concentrations. These studies suggest the
15 possibility of these chemicals being present in the near-field spatial scales in the WUI fire
16 events, though no studies to date have reported elevated concentrations of PCDFs and
17 PCDDs from fire events in regional urban air far away from the fires.

18 **2.1.2.7 Particulate Carbon**

19 As noted earlier, PM from wildland fire smoke has a significant carbonaceous component.
20 Studies have shown that organic carbon (OC) and elemental carbon (EC) are the largest
21 components of aerosols emitted from biomass burning and were larger global contributors
22 to total particulate carbon compared with fossil fuel burning [Bond et al. 2004; Na and
23 Cocker 2008]. Smoke PM_{2.5} contains 5%–20% EC and greater than 50% OC [Adetona et al.
24 2016; Zhang et al. 2023]. Aurell and Gullett [2013] studied laboratory and prescribed burns
25 in the southeastern United States and observed a linear trend between higher black carbon
26 and brown carbon emissions and higher modified combustion efficiency, suggesting that
27 more intense wildland fires will lead to higher particulate carbon emissions.

28 Bian et al. [2020] investigated the differences in EC fraction of PM_{2.5} on smoke-impacted
29 days from 2008–2017 in three U.S. regions (Pacific West, Southeast, and Southwest). Their
30 findings were consistent with previous studies that showed the EC fraction in smoke PM_{2.5}
31 was higher in the Pacific West and Southwest, and lower in the Southeast where smoldering
32 fires were more prevalent [Bian et al. 2020].

33 On a local scale, Na et al. [2008] reported that OC and EC concentrations were significantly
34 higher than those on nonsmoke-impacted days, in Riverside, CA, approximately 30 km
35 downwind of a significant wildland fire in October 2003. Maximum concentrations reported
36 during the fire event were 68.4 µg/m³ for OC and 5.5 µg/m³ for EC. After the fire, the
37 maximum concentrations observed were 13.7 µg/m³ for OC and 3.7 µg/m³ for EC [Na and
38 Cocker 2008].

Table 2–2. Criteria air pollutants regulated by the EPA that may be emitted during wildland and WUI fire events

Group of pollutants	Phase	Exposure routes	Potential health outcomes	Selected references
Carbon monoxide (CO)	Gas	Inhalation	Oxygen deprivation to critical organs, dizziness, confusion, unconsciousness	EPA 2024a
Nitrogen oxides (NO _x) or Sulfur oxides (SO _x)	Gas	Inhalation	Respiratory irritation, aggravation of lung diseases, asthma development	EPA 2024e; EPA 2024g
Ozone (O ₃)	Gas	Inhalation	Respiratory irritation, difficulty breathing, aggravation of lung diseases, asthma development	EPA 2024c
Particulate matter (PM)	Particulate	Inhalation, ingestion, dermal	Respiratory irritation, difficulty breathing, asthma aggravation, irregular heartbeat, heart attacks	EPA 2024f
Lead (Pb)	Particulate	Inhalation, ingestion	Cardiovascular effects, decreased kidney function, reproductive problems, serious developmental effects to fetuses, infants, and adolescents	EPA 2024d

Table 2–3. Hazardous air pollutants regulated by the EPA that may be emitted in wildland and WUI fires. Adapted from NAS [2022]

Group of pollutants	Common examples	Phase	Exposure routes	Potential health outcomes	Selected references
Volatile organic compounds (VOCs)	Formaldehyde, acetaldehyde, acrolein, benzene, toluene, ethylbenzene, xylenes	Gas	Inhalation, ingestion	Cancer, reproductive and developmental toxicity, neurotoxicity, respiratory irritation, odorants	EPA 2023e; NAS 2022
Polycyclic aromatic hydrocarbons (PAHs)	Benzo(a)pyrene, benzo(a)anthracene, pyrene, benzo(b)fluoranthene, chrysene, fluoranthene	Gas, particulate	Inhalation, ingestion, dermal	Cancer, reproductive and developmental (teratogenic) toxicity, kidney, and liver damage	ATSDR 2014; NAS 2022
Polychlorinated biphenyls (PCBs)	2-Chlorobiphenyl, 2,2-dichlorobiphenyl, aroclors, 2,4,5-trichlorobiphenyl	Particulate	Inhalation, ingestion, dermal	Cancer, neurotoxicity, immune suppression, endocrine disruption, reproductive and developmental toxicity, respiratory toxicity	Ahlborg et al. 1992; ATSDR 2000; NAS 2022
Dioxins and furans	Polychlorinated dibenzo-p-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs)	Gas	Inhalation, ingestion, dermal	Cancer or predisposition to cancer, reproductive and developmental effects, immune suppression, dermal toxicity; endocrine disruption	EPA 2023c; NAS 2022
Plasticizers	Ortho phthalates, adipates, terephthalates, benzoates	Particulate	Inhalation, ingestion	Endocrine disruptors, reproductive and developmental toxicity	EPA 2023d; NAS 2022
Flame retardants	Tris(1-chloro-2-propyl) phosphate, tris(2-tris chloroethyl) phosphate	Gas, particulate	Inhalation, ingestion	Neurotoxicity or neurodevelopmental damage, reproduction and fetal development effects, endocrine and thyroid disruption	ATSDR 2015; NIEHS 2024; NAS 2022

Group of pollutants	Common examples	Phase	Exposure routes	Potential health outcomes	Selected references
Inorganic acid gases and organic gases	Hydrogen chloride (HCl), ammonia (NH ₃), phosgene	Gas	Inhalation	Chemical burns, pulmonary edema, increased risk of laryngeal and lung cancer, chemical pneumonitis, bronchiolitis, reactive airways dysfunction syndrome	ATSDR 2002; CDC 2018; NCI 2022; EPA 2023e; NAS 2022
Metals and metalloids	Arsenic, cadmium, chromium, copper, manganese, mercury, nickel, zinc	Particulate	Inhalation, ingestion, dermal	Neurotoxicity, reproductive and developmental effects, dermal irritation, or allergen; respiratory irritation	EPA 2004; NIOSH 2023; Boaggio et al. 2022; NAS 2022; Rice et al. 2023

NOTE: Table 2–3 focuses on groups of chemicals with sound available data. The lists are meant as common examples and not meant to be exhaustive.

2.2 Population of Outdoor Workers

2.2.1 Definition and Characteristics of Outdoor Workers

Outdoor workers are people working outside a physical structure in rural, suburban, or urban areas. Outdoor work may be carried out with other workers (e.g., construction sites) or in isolation without close or direct supervision. Work may occur in remote locations (e.g., an oil and gas well site or rural highway), at worksites where an employee is physically separated from coworkers (e.g., truck drivers), or may involve interaction with the public (e.g., postal workers). Outdoor workers may be at higher risk of exposure to wildland fire smoke than indoor workers due to a lack of mechanically ventilated physical structures and increased breathing rates from physical exertion in potentially hot ambient temperatures. Outdoor workers may have limited means of communication and a reduced chance of receiving help in an emergency, if working alone, compared with indoor workers. An understanding of the work characteristics of outdoor workers is important for employers to consider when protecting their workers.

2.2.2 Estimating the Number of Outdoor Jobs

This section discusses the burden and likelihood of outdoor work in the United States by occupational group. “Burden” is the proportion of the outdoor workforce in occupational groups. For example, 4.6% of the outdoor workforce are farming, fishing, and forestry workers. “Likelihood” is the percentage of an occupational group that works outdoors. For example, 94.0% of farming, fishing, and forestry workers work outdoors. The burden and likelihood of work was quantified in the United States as outdoor work by occupational group, using the definition of outdoor workers described above. This was operationalized as persons employed in occupations that required spending significant time working outdoors and exposed to the weather, referred to as “outdoor occupations.” The estimated number of U.S. jobs employed in outdoor occupations in 2023 is referred to as “outdoor jobs.”

2.2.2.1 Data Sources to Identify Outdoor Occupations

Data were used from the Occupational Information Network (O*NET) database (version 28.2) to identify occupations that involve spending significant time outdoors. The authors used the ordinal *Frequency Required to Work Outdoors, Exposed to Weather* work context variable (element ID # 4.C.2.a.1.c) from the occupational requirements domain [O*NET 2024].

The 2023 projected data from the Bureau of Labor Statistics (BLS) Employment Projections program [BLS 2023a] was used to estimate employment (number of jobs), in identified outdoor occupations. Employment was estimated at the 6-digit level using the 2018 Standard Occupational Classification (SOC) codes [BLS, no date, b] and aggregated at the SOC major occupational group (2-digit) level.

2.2.2.2 Criteria to Define Outdoor Occupations

O*NET data characterize detailed occupations, each of which is identified with an O*NET occupation code. These codes are based on the 2018 SOC codes. In most cases, the O*NET codes simply correspond to the SOC detailed occupation codes at the 6-digit level. In some cases, however, O*NET detailed occupations are more specific than the SOC detailed codes. These are identified in the O*NET coding system by adding a two-decimal extension to the SOC code (.01, .02, etc.) for the more detailed occupations. Workers employed in these detailed O*NET occupations represent a subset of those employed in the corresponding 6-digit SOC detailed occupation. For SOC detailed occupations that also have more detailed O*NET occupations, O*NET collects data at the 6-digit detailed SOC occupation level, as well as the more detailed O*NET occupation level.

The possible values for the ordinal *Frequency Required to Work Outdoors, Exposed to Weather* variable, which correspond to survey response options, were (1) never, (2) once a year or more but not every month, (3) once a month or more but not every week, (4) once a week or more but not every day, and (5) every day. For each detailed occupation, O*NET reports a standardized average score for this variable that can range from 0 to 100. An outdoor occupation was defined as one with a standardized average score of 75 or more (i.e., ranging from “once a week or more but not every day” to “every day”) on the *Frequency Required to Work Outdoors, Exposed to Weather* O*NET variable. This methodology has been used previously in a NIOSH study that used outdoor work, as determined by O*NET data, as an exposure measure [Cox-Ganser and Henneberger 2021].

2.2.3 List of Employment by Outdoor Occupations

Using the methods described above, 146 outdoor occupations were identified, 132 of which corresponded to a 6-digit SOC code and had employment estimates available from the BLS Employment Projections program. Fourteen of the identified outdoor occupations were O*NET detailed occupations that did not correspond to 6-digit SOC codes. The BLS program does not produce employment estimates for the more specific O*NET detailed occupations, only those that correspond to the standard 6-digit SOC codes.

By considering employment in these 132 outdoor occupations, an estimation was made that the United States had 20.0 million outdoor jobs in 2023. These jobs involve spending significant time working outdoors, and they account for 11.8% of total civilian employment. The SOC major occupational groups that contributed the largest number of all outdoor jobs include the following:

- Construction and extraction occupations (24.9%)
- Transportation and material moving occupations (23.8%)
- Installation, maintenance, and repair occupations (16.9%)
- Building and grounds cleaning and maintenance occupations (8.2%)
- Protective service (7.7%)
- Farming, fishing, and forestry (4.6%)

1 The SOC major occupational groups with the highest proportions of outdoor jobs (i.e., the
2 number of outdoor jobs in the occupational group divided by all jobs in the occupational
3 group) are shown in Table 2–4, along with typical examples of detailed occupations
4 included in the group.

5 Estimated employment in farmworker occupations (SOC 45-2092 and 45-2093) from the
6 Projected Employment program [BLS 2023a] is less than one million jobs. This figure may
7 underestimate individual workers because of the challenges in counting migrant and
8 seasonal farmworkers. The Current Population Survey (CPS) estimated the average annual
9 number of agricultural wage and salary workers at 1.5 million in 2022 [BLS 2023b]. The
10 CPS estimates include self-employed and farmworkers, but only for the approximately 560
11 less detailed occupation categories included in the Census occupational codes scheme.

12 The USDA National Agricultural Statistics Service estimated the number of farmworkers at
13 776,000 in October 2023 [USDA 2023b]. Data from the BLS Quarterly Census of
14 Employment and Wages indicates that the average annual wage and salary employment in
15 crop production (NAICS 111) in 2022 was 574,292 [BLS, no date, a] and in animal
16 production and aquaculture (NAICS 112) was 264,023 [BLS, no date, a]. This is a total of
17 838,315 jobs. Therefore, farmworker employment is estimated to be between 800,000 and
18 1.5 million.

1 **Table 2–4. Major occupational groups with at least 25% employment in outdoor**
 2 **occupations**

Major occupational group	Examples of occupations	Employment (in thousands)	Major occupational group employment accounted for by employment in outdoor occupations (%) (likelihood) [†]	Percentage of all outdoor employment (burden) [†]
Farming, fishing, and forestry*	Farmworkers and laborers, crop, nursery, greenhouse; fishers and loggers; farmworkers, farm, ranch, and aquacultural animals; agricultural equipment operators.	918.2	94.0	4.6
Construction and extraction	Construction laborers; electricians; highway maintenance workers; operating engineers; and other construction equipment operators.	4,986.6	66.3	24.9
Installation, maintenance, and repair	General maintenance and repair workers; heating, air conditioning, and refrigeration mechanics and installers; bus and truck mechanics; telecommunications equipment installers.	3,369.9	51.5	16.9
Protective service	Police and sheriff’s patrol officers; firefighters.	1,548.9	43.3	7.7
Transportation and material moving	Heavy and tractor-trailer truck drivers; driver/sales workers; light truck drivers; refuse and recyclable material collectors.	4,756.4	31.5	23.8
Building and grounds cleaning and maintenance	Landscaping and groundskeeping workers; pest control workers; tree trimmers and pruners.	1,644.2	29.4	8.2

3 * The farming, fishing, and forestry population counts may be underreported.

4 † Percentages calculated from the raw employment data from the 2023 BLS Employment Projections
 5 program, <https://www.bls.gov/emp/tables/emp-by-detailed-occupation.htm>.

1 Table 2–4 accounts for approximately 86% of all outdoor workers. The table is not
2 exhaustive; it covers only major occupational groups. The occupational groups where
3 outdoor workers are present in smaller numbers include management (7.1%), production
4 (1.8%), personal care and service (2.6%), and office and administrative support (1.6%).

5 Although the farming, fishing, and forestry occupations represent nearly 5% of all outdoor
6 workers, 94% of workers in this industry are estimated to work outdoors [BLS 2023a]. The
7 farming workforce is a mixture of self-employed farm operators and their families and hired
8 farmworkers [USDA 2023a]. A hired farmworker is an individual who performs agricultural
9 work on farms or for farm labor contractors [Payne 2023; USDA 2023a]. A large proportion
10 (94.0%) of farmworkers are outdoor workers [BLS 2023a]. They are an at-risk population
11 primarily because they tend to be of lower socio-economic status [BLS 2021]. For a more
12 comprehensive discussion on the health equity of farmworkers, please refer to Section 2.4.
13 Farmworkers can be further divided into migrant farmworkers or seasonal farmworkers.
14 The Migrant Clinicians Network defines a migrant farmworker as “an individual who is
15 absent from a permanent place of residence for the purpose of seeking renumeralated
16 employment in agricultural work,” and seasonal farmworkers as “individuals who are
17 employed in temporary farmwork but do not move from their permanent residence to seek
18 farmwork” [Migrant Clinicians Network 2024].

19 Approximately 66% of workers in the construction and extraction industry work outdoors.
20 In the construction and extraction occupations laborers are the major group that work
21 outdoors. The installation, maintenance, and repair occupations include workers who install
22 and repair telecommunication equipment. Installers and repairers of telecommunication
23 lines, as well as electric power line installers and repairers are also included in this
24 occupational group. Unlike most workers who work at ground level, telecommunication
25 workers work part of the time from the top of antenna towers and utility poles, and thus
26 may experience a different exposure profile to wildland fire smoke than those who work at
27 ground level. More than half the workers in this occupation (51.5%) work outdoors. The
28 protective service occupations where outdoor work is most prevalent are police and
29 sheriff’s patrol officers. Approximately 43% of workers in this industry work outdoors.
30 Patrol officers spend part of their job inside vehicles and part of the time outside vehicles,
31 presumably during traffic stops and beat patrols. These workers, and others who drive for
32 work, are assumed to have some protection from wildland fire smoke while inside their
33 vehicles. When the vehicle’s windows are rolled up and the heating/cooling system is
34 filtering the air before it enters the cabin, the vehicle occupants are likely to experience less
35 inhalation exposure to wildland fire smoke than if they were not inside the vehicle.
36 However, if a vehicle’s air filtration system is unable to withstand prolonged exposure to
37 wildland fire smoke, the occupants will not be adequately protected from smoke inhalation.
38 The transportation and materials moving occupations where outdoor work is prevalent
39 include heavy and tractor-trailer truck drivers, light truck drivers, and driver/sales
40 workers. These workers spend part of their job inside vehicles and part of the time outside
41 vehicles presumably while loading or unloading merchandise. As described above, these
42 workers may have varied inhalation exposures to wildland fire smoke from spending part of
43 the workday inside vehicles. The building and grounds cleaning, and maintenance

1 occupations where outdoor work is most prevalent is landscaping and groundskeeping
2 workers. About 29% of workers in this industry work outdoors.

3 **2.2.4 Strengths and Limitations**

4 The data sources and methods used to estimate outdoor employment and its burden and
5 likelihood by major occupational group have strengths and limitations. The first strength of
6 this analysis is that it identified outdoor occupations using an empirical method based on
7 O*NET data from a sample that was scientifically designed to be representative of all SOC
8 detailed occupations in which U.S. workers are employed. Therefore, our methodology does
9 not simply make an assumption about who outdoor workers are.

10 However, using this data source also has two important limitations. One, like all similar
11 work setting variables in the work context module of the O*NET occupational
12 characteristics domain, the *frequency required to work outdoors exposed to weather* variable
13 measures the typical *frequency*, not the *duration*, of outdoor work for each detailed
14 occupation. Thus, in some cases, workers in occupations with lower standardized average
15 scores on the *frequency required to work outdoors exposed to weather* variable might spend
16 more time working outdoors during a specified period than workers in occupations with
17 higher scores.

18 To the extent that duration, not frequency, of outdoor work is the more relevant measure of
19 potential wildland fire smoke exposure, this could result in some exposure misclassification
20 bias. However, it is likely that the correlation between frequency and total weekly duration
21 of outdoor work is sufficiently positive and strong enough to minimize any actual
22 occurrence of such bias.

23 The other limitation is that O*NET detailed occupations, identified by the addition of a two-
24 decimal extension to the SOC code (.01, .02, etc.) are sometimes more specific than standard
25 SOC codes and, in such cases, have no corresponding SOC code. Of the 146 outdoor
26 occupations identified, 14 were O*NET detailed occupations with no directly corresponding
27 SOC code. Therefore, direct estimates of employment in these 14 detailed occupations were
28 not available from BLS, which only estimates employment at the 6-digit standard SOC
29 detailed occupation level. It should be clarified that the 14 O*NET detailed occupations with
30 no directly corresponding SOC code are subsets of categories that match SOC codes.

31 Another strength of this analysis is that it uses 2023 projections from the BLS Employment
32 Projections program to estimate employment by detailed occupation. This data source
33 provides estimates of total civilian, non-institutionalized employment for all 800+ detailed
34 occupations at the SOC 6-digit level. This has important advantages over other potential
35 data sources for civilian employment by occupation, such as the BLS [Occupational
36 Employment and Wage Statistics](#) (OEWS) program or the [Current Population Survey](#) (CPS).
37 The OEWS program estimates employment for all SOC 6-digit detailed occupations, but only
38 for nonfarm payroll jobs. It excludes the self-employed, owners and partners in
39 unincorporated firms, household workers, or unpaid family workers. These exclusions—

1 particularly excluding the self-employed—would substantially underestimate employment
2 in outdoor jobs.

3 The BLS Employment Projections data measures total employment as a count of jobs, not a
4 count of individual workers. As a measure of the magnitude of employment by occupation,
5 counts of jobs rather than individuals are advantageous. This captures the increased
6 potential for exposure contributed by workers holding more than one outdoor job and does
7 not overlook the exposure potential contributed by workers whose primary job is not an
8 outdoor job but who also hold one or more secondary outdoor jobs.

9 Finally, the definition used for an outdoor occupation was based on an arbitrary score of 75
10 or more on the *frequency required to work outdoors exposed to weather* variable. The
11 rationale for using this score is that it corresponds to the two highest values of the five-level
12 ordinal variable.

13 **2.3 Routes of Worker Exposure**

14 A route of exposure is the way that a contaminant enters the body. This occurs in three
15 primary ways: inhalation, dermal absorption, or ingestion. Inhalation exposure happens
16 when a worker breathes in the contaminant, and it enters the body through the respiratory
17 system. Dermal exposure occurs when contaminants contact and are absorbed into the
18 body through the skin. Ingestion may occur through hand-to-mouth or surface-to-mouth
19 contact after chemicals in the smoke settle on surfaces. However, this is not typically an
20 occupational route of exposure concern for wildland fire smoke. Take-home exposures can
21 occur when contaminants stick to the skin, hair, or clothes and get carried to the worker's
22 home or other shared environment, posing a subsequent potential path for exposure.
23 Indirect exposure of an embryo or fetus can occur if individuals are exposed during
24 pregnancy. Figure 2–8 summarizes the routes of exposure discussed in this section.

25 **2.3.1 Inhalation Exposure**

26 Occupational inhalation exposure to wildland fire smoke occurs when smoke is in a
27 worker's personal breathing zone (PBZ). The concentration of smoke in the worker's PBZ,
28 the duration of exposure, frequency of exposure, and physical exertion can affect the
29 worker's total level of inhalation exposure. Physical activity changes the breathing pattern,
30 leading to increased inhalation of air contaminants [Bigazzi and Figliozzi 2014].

31 Few studies assessing wildland smoke exposures to outdoor workers have been published,
32 even though the United States had 20.0 million outdoor jobs in 2023, which account for
33 11.8% of total civilian employment (see Section 2.2.3). Austin et al. [2021] looked at county-
34 level data across the state of Washington to estimate the burden of heat and PM_{2.5} exposures
35 for agricultural workers from 2010 to 2018. Peak PM_{2.5} exposures occurred when the heat
36 index was around 85°F and during the summer when wildland fires are most prevalent.
37 Washington state counties with the largest agricultural worker populations tended to have
38 the highest simultaneous heat and PM_{2.5} exposures. The authors also found that rural areas

1 often had limited access to air quality monitors, making identification and mitigation of
2 poor air quality episodes challenging [Austin et al. 2021].

3 Another occupational group that is subject to poor air quality exposures from wildland fires
4 includes outdoor construction workers. Construction workers may be at higher inhalation
5 exposure risk due to many factors including the following: (1) they may spend a
6 considerable amount of time outdoors; (2) they have a higher level of exertion leading to
7 higher respiration rates; and (3) with higher respiration rates, workers may do more mouth
8 breathing, which negates filtration mechanisms found within the nose [Zuidema et al.
9 2021].

10 Zuidema et al. [2021] assessed the potential impact of a Washington state emergency rule
11 meant to protect outdoor workers in the state from poor air quality due to wildland fires.
12 This rule has an “encouraged” threshold of 20.5 $\mu\text{g}/\text{m}^3$ for $\text{PM}_{2.5}$ (equivalent to an Air
13 Quality Index [AQI] of 69) and a “required” threshold of 150 $\mu\text{g}/\text{m}^3$ for $\text{PM}_{2.5}$ (or an AQI of
14 151). At these thresholds, employers are either encouraged or required to limit outdoor
15 workers’ exposure to smoke by reducing, rescheduling, or relocating work; providing
16 enclosed buildings or vehicles where air is filtered; or reducing work intensity. The study
17 estimated that the number of days of exposure exceeding the Washington state thresholds
18 was between 488 and 2,704 (depending on the threshold level) across all counties during
19 the study period (2011–2020). This study showed that large numbers of outdoor workers
20 in the construction industry are at risk for inhalation exposure to $\text{PM}_{2.5}$ levels above the
21 Washington state emergency rule threshold limits [Zuidema et al. 2021].

22 Among workers, wildland firefighters have been the primary subject of almost all smoke
23 exposure studies because they are a highly exposed cohort. Inhalation exposures have been
24 measured on wildland firefighters across a range of studies [Adetona et al. 2011; Cherry et
25 al. 2021; Cherry et al. 2023; Navarro et al. 2017; Navarro et al. 2021; Reinhardt and Ottmar
26 2004; Wu et al. 2021].

27 Smoke from wildland fires consists of a complex mixture of particulate and gaseous
28 compounds, including the criteria air pollutants (carbon monoxide, lead, nitrogen dioxide,
29 ozone, particulate matter, and sulfur dioxide) and many hazardous air pollutants such as
30 VOCs, PAHs, metals, inorganic acids, dioxins, and PCBs [EPA 2023b; NAS 2022]. These
31 constituents are further described in Section 2.1. The relative amount of the various
32 components varies based on combustion conditions (e.g., flaming versus smoldering), as
33 well as the fuel characteristics [Naehler et al. 2007; Wu et al. 2021]. $\text{PM}_{2.5}$ may serve as the
34 best marker of health effects among woodsmoke components and tends to be one of the
35 most elevated markers of ambient smoke components [Naehler et al. 2007].

36 From 2015 to 2017, the U.S. Forest Service collected respirable samples, particles with a
37 penetration of 50% at 4 μm diameter (see Figure 2–5), on wildland firefighting crews
38 performing various tasks [Navarro et al. 2021]. Inhalation exposures to the respirable
39 fraction measured by the U.S. Forest Service varied over the different types of crews and job
40 tasks with geometric means ranging from 0.15 to 0.65 mg/m^3 . The median respirable mass
41 concentration (0.79 mg/m^3) for wildland firefighters performing direct suppression

1 exceeded the recommended National Wildland Fire Coordinating Group occupational
2 exposure limit (OEL) of 0.7 mg/m³ [Navarro et al. 2021].

3 Navarro et al. [2017] also assessed exposure to polycyclic aromatic hydrocarbons (PAHs)
4 among wildland firefighters conducting fire suppression and prescribed burn activities.
5 Overall, 17 PAHs were detected in personal samples on firefighters, with naphthalene,
6 retene, and phenanthrene as consistently the highest measured among the PAHs for all
7 sampling sessions [Navarro et al. 2017].

8 Reinhardt and Ottmar [2004] also conducted several long-term exposure studies among
9 wildland firefighters in the 1990s. During 30 days of exposure monitoring at locations in
10 Washington, Oregon, California, and Montana, the data showed exposures to acrolein,
11 benzene, CO, carbon dioxide, formaldehyde, and respirable particulate (particles less than
12 3.5 µm in size, or PM_{3.5}). Further, they showed that the pollutants measured were highly
13 correlated, allowing one pollutant to be used to reasonably estimate others. This was
14 especially the case in nonurban areas where there was less confounding to urban sources of
15 air contaminants (e.g., PM and CO) like traffic [Reinhardt and Ottmar 2004].

16 Other researchers have assessed smoke exposures for other pollutants as well. Wu et al.
17 [2021] assessed exposures among wildland firefighters in the midwestern United States,
18 specifically looking at PM_{2.5}, CO, black carbon, and trace metals. The geometric means (GMs)
19 were 1.43 ± 0.13 mg/m³ for PM_{2.5}, 7.02 ± 0.69 ppm for CO, and 58.79 ± 5.46 µg/m³ for black
20 carbon. The concentrations of PM_{2.5} seen in this study were 1.8–3.2 times higher than those
21 seen in studies conducted in the western United States and 2.7–5.4 times higher than those
22 from prescribed burns in the southeastern United States [Wu et al. 2021]. The authors
23 hypothesized that the differences were likely due to the different types of vegetation in the
24 different regions of the country. And although trace metals were measured in the
25 particulate, the concentrations for all metals were well below any corresponding OELs.

26 **2.3.2 Dermal Exposure**

27 Dermal exposures are exposures that can be received through the largest organ of the
28 human body—the skin. In recent years, increasing attention has been placed on the
29 importance of dermal exposure to the entire worker exposure profile. Skin as an organ
30 differs from other organs in the body in terms of absorption, resistance, and transmission.
31 Dermal absorption can occur from direct contact from liquids, solids, vapors, and
32 suspensions.

33 Dermal exposures can be estimated by defining dermal contact area, dermal concentration,
34 dermal retention time, and dermal penetration potential [Guy and Hadgraft 2020]. Together
35 these factors can help assess the total dermal hazard and exposure. The chemical properties
36 of the contaminant affect the dermal penetration potential and dermal retention time [Guy
37 and Hadgraft 2020]. Dermal permeability is known to increase with temperature, which
38 may be an important factor to consider in occupations with heat exposure [Park et al. 2008].
39 The dermal contact area is the exposed surface area of the skin (such as the area not
40 covered by clothing or PPE), or the area in direct contact with the substance of concern,

1 typically defined in cm². Skin also varies in thickness throughout different parts of the body,
2 which may impact absorption or dermal penetration.

3 Like inhalation exposure, dermal exposure to wildland fire smoke has been studied
4 primarily in wildland firefighters, a highly exposed cohort. Sousa et al. [Sousa et al. 2022]
5 conducted a literature review that showed firefighters' skin can be contaminated with
6 polycyclic aromatic hydrocarbons (PAHs). The contamination was primarily on the neck,
7 wrists, face, and hands, which are areas less protected by clothing and personal protective
8 equipment (PPE) [Sousa et al. 2022]. Data have shown that PAHs can reach deep skin layers
9 because of diffusion or absorption into the epidermis, allowing for systemic distribution and
10 metabolism. PAHs with a smaller molecular weight tend to be more volatile and release into
11 the air. However, PAHs with a larger chemical structure tend to be absorbed into solid
12 surfaces, and their lipophilic properties may facilitate their transfer across biological
13 membranes, such as skin [ATSDR 1995]. Some examples of PAHs include benzo[a]pyrene,
14 fluorene, and naphthalene. VanRooij et al. [1993] found that 20%–56% of PAHs (as a low
15 dose of coal tar) on the skin will be absorbed within 6 hours, depending on the anatomical
16 site of the exposure. Many factors influence the dermal absorption and excretion rates
17 across different regions of the body, including skin thickness, hydration levels, and hair
18 follicle density [VanRooij et al. 1993].

19 A study of dermal exposures was conducted in firefighters located in 2 Canadian provinces
20 [Cherry et al. 2023]. Overall, 710 skin wipes were analyzed, 339 from the start of shift and
21 371 from end of shift on fire days. Of the 21 PAHs measured, only 3 (1-naphthalene,
22 phenanthrene, and pyrene) had concentrations above the limits of detection on 20% or
23 more of wipes. Rates of detection of the PAHs on the firefighters' skin were higher at the
24 end of the shift versus the start of the shift [Cherry et al. 2023].

25 Fent et al. [2014] evaluated firefighters' exposure to combustion products using air, dermal,
26 and biomarker sampling during and following a series of controlled structure burns. The
27 results suggested that despite wearing full protective ensembles, including respiratory
28 protection, the firefighters still absorbed combustion products into their bodies, likely
29 through their skin. The neck, being the least shielded by protective clothing among the
30 sampled dermal areas, experienced the highest levels of exposure.

31 **2.3.3 Combined Exposure to Wildland Fire Smoke,** 32 **Other Outdoor Air Contaminants, and Heat**

33 Outdoor workers near the WUI may also be exposed to other types of smoke (e.g., structural
34 and vehicle fire smoke) and a mixture of other anthropogenic air pollutants, such as traffic-
35 related air pollution, urban industry emissions, and power plant emissions, in addition to
36 wildland fire smoke [Hwang et al. 2023]. There is evidence that the smoke produced from
37 burning man-made materials differs in composition from wildland fire smoke and may be
38 more toxic [Fabian et al. 2014; Fent et al. 2018; Fent et al. 2020; Keir et al. 2020; O'Dell et al.
39 2020]. This is discussed in greater detail in Section 2.1.1. Work that involves operating
40 vehicles or heavy machinery, such as in construction or transportation occupations, may

1 contribute to additional exposure to air pollutants, such as diesel exhaust [Pronk et al.
2 2009]. Minimal information is available about outdoor workers' occupational exposure to
3 other air contaminants in combination with wildland fire smoke.

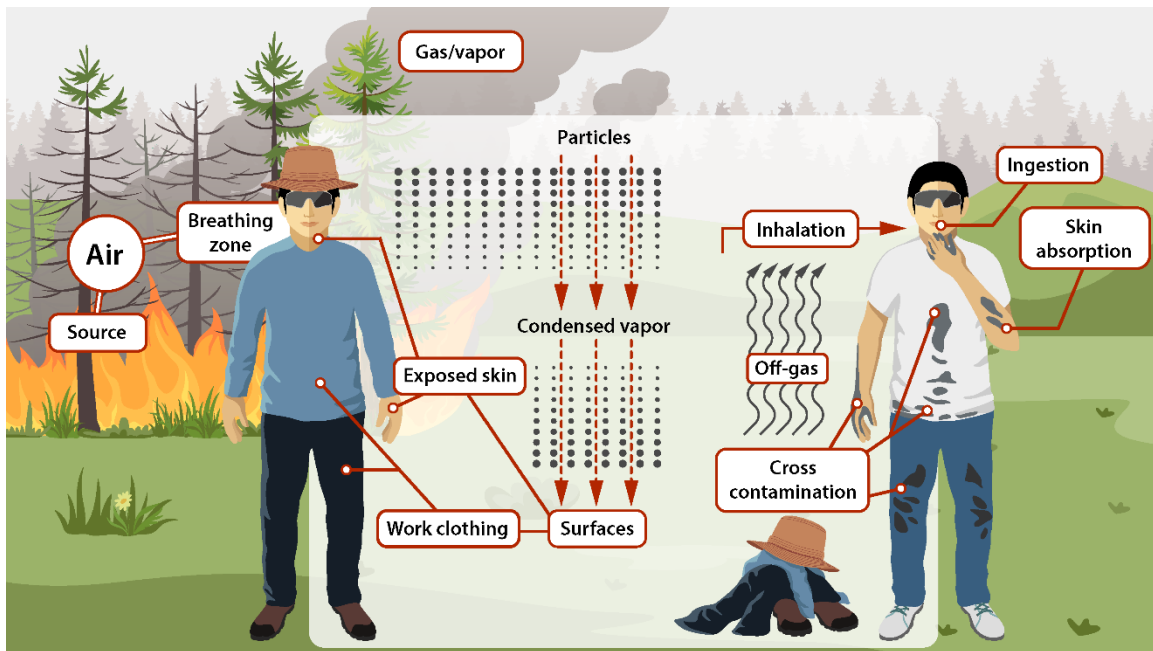
4 Heat exposure may be another combined occupational exposure concern. Pharmacological
5 research has shown that exposure to heat can increase the absorption and biological effects
6 of certain compounds [Sidhu et al. 2011; Gordon and Leon 2005; Vanakoski 1998]. A recent
7 study has shown that the concurrent exposures to extreme heat and wildfire smoke
8 resulted in increased cardiorespiratory hospitalizations in California from 2006 to 2019
9 when compared with hospitalizations from either hazard alone [Chen et al. 2024]. Other
10 studies have provided evidence of a relationship between increased mortality rates and the
11 combination of heat stress and exposure to air pollution [Rainham and Smoyer-Tomic 2003;
12 Katsouyanni et al. 1993].

13 **2.3.4 Take-Home Exposures**

14 Workers' exposures to wildland fire smoke may not be limited to their time on-duty. "Take-
15 home" exposures, or para-occupational exposures, occur when a worker inadvertently
16 carries contaminants from work via their clothes, hair, skin, PPE, personal items, and other
17 means, into the home or other shared environments [Kalweit et al. 2020]. The take-home
18 route of exposure is a concern for both continued exposure of the workers as well as
19 potential indirect exposure of others in workers' households. Well-known examples of take-
20 home exposures include agricultural pesticides and heavy metals [CDC 2015; Fenske et al.
21 2013; CDC 2012]. However, the potential for take-home exposures in other occupations,
22 including firefighting, has been documented [Brown et al. 2014; Easter et al. 2016; Fent et
23 al. 2017; Mayer et al. 2019; Shen et al. 2015; Siegel et al. 2023]. These studies
24 predominantly focus on structural firefighters, and as mentioned, the composition of
25 structural fire smoke and wildland fire smoke differs. Although take-home exposures
26 resulting from occupational exposure to wildland fire smoke in other outdoor workers have
27 not been specifically documented in the literature, studies that examine take-home
28 exposures in firefighting could inform the possible composition and characteristics of
29 potential take-home exposures from occupational exposure to wildland fire smoke in other
30 outdoor occupations.

31 The potential for cross-contamination and exposure from clothing and PPE in firefighters
32 has raised concerns. Fent et al. [2015] studied the off-gassing of firefighter turnout gear
33 (coat and trousers) following controlled structure burns. The sampling of the air
34 surrounding the gear in an enclosed case showed greater than fivefold increases in several
35 VOCs, including styrene, benzene, 1,4-dichlorobenzene, acetone, and cyclohexane, although
36 these levels were well below any applicable short-term OELs and would likely be fully
37 evaporated in a short time frame (within an hour) [Fent et al. 2015]. This could be an
38 exposure concern, particularly if the contaminated PPE is in an enclosed space with the
39 worker, such as a vehicle. The potential for exposure to smoke contamination from the
40 clothing of outdoor workers may provide another path to exposure, but this phenomenon
41 has not been documented in the published literature for outdoor workers.

1 Fent and other investigators have observed PAH contamination on the exterior of firefighter
2 clothing [Hwang et al. 2021; Mayer et al. 2022; Stec et al. 2018]. These studies indicate that
3 clothing exposed to smoke may serve as a source of PAH contamination and result in the
4 potential for dermal exposures following handling of the clothing. Wilkinson et al. [2023]
5 looked at the effectiveness of reducing PAH contamination by a wet wipe-down of clothing
6 on scene and laundering of firefighter clothing. The researchers found that both laundering
7 and wet soap wipe-down methods (post-fire) were effective in reducing surface
8 contamination and appear to prevent accumulation of contamination after repeated
9 exposures [Wilkinson et al. 2023]. However, semi-volatile PAHs deep within the fibers of
10 the protective clothing were not effectively reduced via either decontamination method,
11 permitting continued off-gassing of these compounds. The potential routes of worker
12 exposures through cross-contamination of skin and clothing from PPE, and the off-gassing
13 of PPE are pictured in Figure 2–8.



14

15 **Figure 2–8. Potential routes of exposure to wildland fire smoke**

16 Figure adapted from the UL Fire Safety Research Institute,
17 <https://training.fsri.org/course/108/comprehensive-cancer-prevention-strategies-for-the-fire-service>.

18 2.4 Health Equity

19 The scientific literature indicates that a disproportionate number of outdoor workers, such
20 as farmworkers and construction workers, can experience structural disadvantages and
21 may be more likely than other workers to be exposed to the risks of wildland fire smoke
22 [Benevolenza and DeRigne 2019; Cascio 2018; Chunga Pizarro 2024; Schulte et al. 2023].
23 Studies show that disadvantages related to socially constructed hierarchies [Flynn et al.
24 2015; Krieger et al. 2006], differential statutory protections [Ide 2021; Liebman et al. 2016],
25 and job characteristics [Foley et al. 2014; NIOSH 2022; Weil 2017] can contribute to a lack

1 of power in the workplace. These disadvantages can lead to preventable disparities in work-
2 related illness rates and fatalities, known as occupational health inequities [NIOSH 2024a;
3 CDC 2024; Fujishiro et al. 2022; Segule et al. 2022]. Structural disadvantages often overlap,
4 which can further limit access to worker protections and hinder the ability to cope with
5 adverse consequences of an injury or illness [Davis et al. 2023; Kalweit et al. 2020; NIOSH
6 2015; Parker et al. 2024; Peckham et al. 2017; WHO, no date].

7 Effective and comprehensive responses to wildland fire smoke (including planning,
8 preparedness, and prevention) can be enhanced by identifying structural and other
9 determinants that contribute to inequitable exposures and outcomes; understanding how
10 structural disadvantages can place some workers at increased risk from wildfire exposures
11 and limit their ability to access resources to mitigate them; and designing interventions that
12 address these disadvantages [Bush et al. 2014; Cunningham et al. 2018; Flynn et al. 2021a;
13 Quandt et al. 2020]. These factors should be considered in the development of a workplace
14 safety and health program, as discussed in Section 5.2.

15 The following sections describe how structural disadvantages can result in inequitable
16 exposure to and protection from wildland fire smoke for outdoor worker population groups
17 (e.g., foreign-born individuals, racial and ethnic minorities, women, younger and older
18 workers, low-income workers, workers in precarious jobs, and incarcerated workers).

19 **2.4.1 Occupational Segregation**

20 Occupational segregation occurs when a certain demographic group (e.g., gender, race,
21 ethnicity, immigration status) is over- or under-represented in a specific job category
22 [Zavoronkova et al. 2022]. Structural disadvantages can result in the overrepresentation of
23 certain worker populations in outdoor jobs. This can contribute to health inequities because
24 outdoor work conditions can increase exposure to wildland fire smoke. Workers
25 experiencing these inequities include those in agriculture, forestry, and fishing;
26 construction; outdoor services (e.g., lawncare, landscaping), and transportation workers
27 (e.g., truck drivers, refuse and recyclable materials collectors). Because many of these are
28 critical infrastructure sectors, the work may have a sense of urgency [CISA, no date]. For
29 example, during growing seasons, production pressures may require farmworkers to put in
30 long work hours. Isolation may be another factor since many outdoor workers may be
31 working alone or away from others. This can lead to a potential lack of access to assistance,
32 safety and health information, and PPE. Outdoor workers are also more likely to spend
33 more time inhaling ambient air and performing heavier, physically laborious tasks than the
34 public. This may increase inhalation rates and cause higher smoke doses per unit of smoke
35 inhaled [Schollaert et al. 2024]. For agricultural workers, peak crop production and
36 wildland fire seasons overlap, and the workers may have a greater proximity to fire-prone
37 landscapes, which could increase exposures to smoke [Austin et al. 2021; Parker et al. 2024;
38 Schollaert et al. 2024]. Agricultural workers often labor for hours or entire shifts, despite
39 exposures to ash, smoke, particulate matter, and other toxins [Pagán-Santana et al. 2023].

40 Men, foreign-born individuals, and racial and ethnic minorities may be overrepresented in
41 outdoor occupations and industries where workers are exposed to wildland fire smoke.

1 Alexander et al. [2021] reported that Hispanic workers were overrepresented in the
2 landscaping services industry, making up 42.7% of landscaping workers compared with
3 17.6% of the total workforce. In contrast, women made up only 10.5% of landscaping
4 services workers, but 47% of the total workforce [Alexander et al. 2021].

5 The Department of Labor’s National Agricultural Workers Survey provides economic and
6 demographic information about workers from farms, orchards, greenhouses, and nurseries.
7 According to the 2019–2020 survey, more than 60% of U.S. farmworkers were born in
8 Mexico and 5% were from Central America. Of U.S.-born farmworkers, one-third were
9 Hispanic [DOL 2022]. Most farmworkers were men (66%) [DOL 2022]. A total of 44% of
10 U.S. farmworkers lacked work authorization [DOL 2022]. Most farmworkers (62%)
11 reported they felt most comfortable speaking Spanish, while a few reported an indigenous
12 language. About 68% reported that they could speak English only “a little,” “somewhat,” or
13 “not at all.” For farmworkers, the average level of formal education was 9th grade. If
14 workers are provided training or written materials in a language or at a grade-level they
15 cannot read or understand, it may be difficult for them to implement safety protections such
16 as accessing and using PPE properly [O’Connor et al. 2014].

17 Like the occupations mentioned above, a disproportionate number of construction workers
18 can face additional structural disadvantages that place them at increased risk of exposure to
19 wildfire smoke or reduce their ability to cope with adverse consequences of that exposure
20 [Zuidema et al. 2021].

21 Bureau of Labor Statistics reports [BLS 2022, 2023c] on the construction industry present
22 the following information:

- 23 • Of all workers in construction, 25.3% were foreign-born [BLS 2022]. This is a
24 significantly higher proportion than workers in all industries (18%) [BLS 2023c].
- 25 • A high proportion of non-Hispanic Asian construction workers were foreign-born
26 (62.5%), slightly less than the proportion of foreign-born non-Hispanic Asian
27 workers in all industries (68.4%).
- 28 • Of Hispanic workers in construction, 67.7% were foreign-born. Although Hispanic
29 people are 17.6% of the U.S. workforce, they make up 30% of construction workers,
30 and their share of construction jobs increased 30% from 2003 to 2020.
- 31 • Hispanic workers were overrepresented in nonmanagerial jobs, such as laborers.
32 They accounted for 46.7% of construction laborers, considerably higher than their
33 share of those employed in the construction industry (30%).
- 34 • Relative to their share of the employed, Hispanic workers were underrepresented
35 among construction managers (14.3%). Non-Hispanic Whites accounted for 60.9%
36 of all those employed in the construction industry, with higher employment shares
37 among construction managers (78.9%). By contrast, 44.1% of construction laborers
38 were non-Hispanic White.
- 39 • In 2020, women accounted for only 10% of construction workers.

2.4.2 Social Hierarchies

Social hierarchies have been defined in the literature as informed, in part, by the social construction of identities along axes such as race, socioeconomic status, nativity, immigration status, language, and gender [Collins 2019; Crenshaw 1991; Homan et al. 2021]. Apart from the segmentation of some groups into more dangerous jobs, one's position within social hierarchies can influence a worker's relationship to employers and coworkers and can place them at increased risk of exposure to occupational hazards [Flynn et al. 2015; Krieger 2009; Liebman et al. 2013]. Social hierarchies can lead to differential treatment on the job and contribute to harassment, bullying, and discrimination [Davis et al. 2023; Okechukwu et al. 2014; Parker et al. 2024]. For example, young construction workers have reported that their older coworkers pressure them into ignoring safety regulations so the team can meet production demands [Flynn and Sampson 2012; Paap 2006]. Similarly, new workers have been reported to be given undesirable tasks and shifts that can increase their exposure to work-related risks and subsequent negative health outcomes [Breslin and Smith 2006].

Social position can also influence a worker's relationship with efforts to protect workers and communities from wildland fire smoke. Workers from structurally disadvantaged social groups—such as racial and ethnic minorities, foreign-born individuals, and women—have been shown to be frequently excluded or not properly represented in studies or data systems because of exclusionary study methodology [Eggerth and Flynn 2012], analytical approaches [Jones 2001; Kaufman et al. 1997], or data collection [Rodriguez-Lainz et al. 2018]. Research and governmental data collection efforts may inadequately capture sociodemographic data (e.g., country of birth, language, immigration status) and social determinants of health information (e.g., employment status, job characteristics, housing conditions, and transportation) [Rodriguez-Lainz et al. 2018; Silver et al. 2024]. This deficiency impedes the identification of workers at increased risk from wildland fire smoke exposure and the factors that influence these risks [Rodriguez-Lainz et al. 2018; Silver et al. 2024]. The result is that knowledge, programs, and PPE designed to protect workers from wildland fire smoke exposure may be less accessible or less effective for those who are at increased risk due to a disadvantaged social position [Cookson et al. 2021; Flynn et al. 2021b; Hart 1971; Hsiao et al. 2009; Katikireddi et al. 2021; Lorenc et al. 2013; White et al. 2009].

For example, PPE design can make it more effective and comfortable for some groups of workers over others. PPE was generally designed using anthropometric data of U.S. military recruits in the middle 1900s [Hsiao et al. 2009]. The increasing diversity of the workforce, compared with the populations represented in these anthropometric data, leads to greater challenges for women and ethnic minorities in finding properly fitting PPE. Several studies have identified poor-fitting PPE as an occupational hazard for women in construction, firefighting, and waste collection. This is because ill-fitting equipment protects workers less effectively and discourages use [Goldenhar et al. 1998; Goldenhar and Sweeney 1996; NIOSH 1999]. PPE designed for women has received limited promotion by manufacturers, making it difficult to find [Flynn et al. 2017]. PPE that fits younger farmworkers may be

1 needed because many states allow children as young as 12 to work, or even younger if they
2 work on their family farm [Liebman et al. 2013].

3 NIOSH is developing a *National Strategy for Equitable Personal Protective Equipment*
4 *Protections for All U.S. Workers* [Dempsey 2023]. These efforts will address the need for
5 human factors and ergonomics contributions to overcome current limitations and barriers
6 to equitable PPE protection for some workers, ensuring that all workers that require PPE
7 are adequately protected from hazards while minimizing negative consequences (e.g.,
8 discomfort, reduced perceptual capabilities, performance decrements).

9 **2.4.3 Differential Statutory Protections**

10 Labor and safety regulations can help protect workers from hazardous exposures such as
11 wildfire smoke. However, these protections can vary by industry sector. One example is the
12 agricultural sector, which has a history of being exempt from particular state and federal
13 labor policies and regulations [Diamond et al. 2022; Liebman et al. 2013; Siqueira et al.
14 2014]. As a result, some legal protections have historically either not applied to agricultural
15 workers or have been differentially applied to workers in this sector [Liebman et al. 2013].
16 For example, in some states children 12 and older can legally work on farms outside of
17 school hours, and children younger than 12 can work on their family’s farm [Liebman et al.
18 2013]. While some efforts to extend protections to agricultural workers have been
19 successful, workers in this sector (particularly those employed on farms with fewer than 11
20 employees) have less legal protection and regulatory coverage than their counterparts in
21 other industries [Liebman et al. 2013].

22 Incarcerated individuals engaged in outdoor labor constitute an important population for
23 consideration, as they perform various types of outdoor work comparable to non-
24 incarcerated workers [ACLU, GHRC 2022; Segule et al. 2022]. Protections under labor
25 regulations differ for this population because incarcerated workers are not “employees” as
26 defined under the Occupational Safety and Health Act, unless they are working for private
27 employers outside a prison [ACLU, GHRC 2022; Ide 2021; Segule et al. 2022]. Some states
28 with OSHA-approved state plans may classify incarcerated workers as employees. However,
29 when incarcerated workers in federal prisons are required to perform work similar to that
30 outside of prisons (e.g., farming, machine operations), applicable provisions apply [OSHA
31 1995]. Still, cases have been documented where enforcement of regulations was
32 insufficient. This resulted in incarcerated persons being exposed to hazards without
33 sufficient training and protective measures, as well as inadequate work practices and job
34 hazard assessment [ACLU, GHRC 2022; Ceballos et al. 2020; Ide 2021].

35 Foreign-born workers are another important demographic group as they constitute roughly
36 18% of the U.S. workforce and are overrepresented in outdoor jobs such as farmwork,
37 construction, forestry, landscaping, and disaster clean-up [BLS 2023c]. Like incarcerated
38 workers, foreign-born workers’ access to certain workplace protections and government
39 programs can be limited, especially for those with an undocumented status [Johnson 2001;
40 O’Donovan 2005]. Workers have reported that an undocumented status can discourage

1 them from raising safety concerns at work or accessing government protections and
2 programs [De Genova 2002; Flynn et al. 2015; Liebman et al. 2016].

3 **2.4.4 Job Characteristics**

4 How industries and jobs are organized can also place workers at increased risk for exposure
5 to wildland fire smoke. Many outdoor workers are employed or subcontracted by smaller
6 businesses. Subcontracting practices can serve to externalize risk of and responsibility for
7 occupational injuries from larger companies to smaller ones [Weil 2017]. Foreign-born
8 individuals and racial and ethnic minorities are overrepresented in small businesses, which
9 can further limit their access to safety resources, because these resources tend to be more
10 limited in smaller businesses compared with larger businesses [Buckley et al. 2008;
11 Cunningham et al. 2018; Hasle and Limborg 2006; Lentz and Wenzl 2006; Sinclair and
12 Cunningham 2014]. In addition, companies in outdoor industries such as construction and
13 agriculture increasingly rely on the practice of classifying workers as “independent
14 contractors,” which establishes a different relationship between the company and the
15 workers [Goldman and Weil 2021]. Specifically, this classification can lead to the company
16 externalizing the responsibility for safety (e.g., training, PPE, and workers compensation) to
17 the workers [Goldman and Weil 2021]. The legal distinction between independent
18 contractors and employees can result in significant disadvantages for workers, especially
19 when these classifications are done erroneously to reduce costs or avoid liability [Goldman
20 and Weil 2021; Weil 2017].

21 The use of piece-rates to compensate workers (often farmworkers) is another concern in
22 work organization. A piece-rate means a worker is paid per unit harvested, instead of an
23 hourly wage. Many farmworkers may prefer jobs that offer piece-rates because they can
24 make more money in less time [Wadsworth et al. 2019]. However, piece-rates may lead to
25 farmworkers working more quickly—despite the temperature, smoke levels, or other
26 environmental conditions—or working more hours to maximize pay. Piece-rates are most
27 often found among populations at a higher risk of exploitation, such as those working more
28 onerous tasks, indigenous workers, or undocumented workers [Reid and Schenker 2016].
29 Farmworkers paid with piece-rates may also face negative outcomes such as increased risky
30 behavior or risk of exhaustion leading to injuries. This can increase the risk for
31 musculoskeletal injuries, severe disabilities, stress, and mental health impacts [Johansson et
32 al. 2010; McCurdy et al. 2003].

33 **2.4.5 Compounding Factors**

34 For many workers, compounding factors outside of the workplace may increase their risk of
35 exposure to wildland fire smoke, such as residence location, living conditions, and
36 transportation [Scott et al. 2024]. Workers living in areas closer to or downwind from
37 wildland fires are more likely to have increased smoke exposures. This is often the case for
38 those working in rural areas. Many workers (including farmworkers, forestry, and
39 incarcerated workers) face limitations in where they can live. In addition to locale, living
40 conditions may also place some workers at additional risk [Davis et al. 2023]. Farmworker

1 housing varied from employee-provided (14%) and rentals (53%), to housing owned by
2 themselves or a family member (31%). Thirty percent of farmworkers lived in “crowded”
3 housing (>1 person per room).

4 Farmworkers, incarcerated workers, and other at-risk populations often lack access to
5 adequate air conditioning, proper insulation, and effective air filtration systems in their
6 living quarters [Arcury et al. 2015a; Huang et al. 2011; Pagán-Santana et al. 2023]. Smoke-
7 saturated clothing may also be difficult to sufficiently clean without access to washing
8 machines. If dryers are unavailable, workers may hang clothing outside in smoky
9 conditions, leading to additional exposures related to wildland fire smoke. Limited access to
10 showers may also restrict farmworkers and incarcerated workers from washing regularly
11 and as needed.

12 Rural areas can also have limited access to air quality monitors, making it challenging to
13 identify and inform at-risk worker populations and mitigate the poor air quality [Austin et
14 al. 2021]. Many of these workers also face limitations in transportation and commuting
15 options [Arcury et al. 2015b]. Those relying on vehicles (including public buses) with
16 inadequate air conditioning systems are likely to travel with windows open, increasing their
17 exposure to outdoor air pollutants. Workers may also ride to and around work locations in
18 the back of trucks [Arcury et al. 2015b], which likely increases their exposure to smoke.
19 Residence location may also affect workers’ access to medical care [Pagán-Santana et al.
20 2023].

21 For farmworkers in rural areas, there may be fewer healthcare facilities despite higher rates
22 of preexisting conditions [Schollaert et al. 2024]. In general, access to medical care may be
23 limited for a variety of reasons that include lack of insurance coverage and difficulties
24 affording payments [Siddiqui et al. 2009; DOL 2024a]. Immigrant workers may face
25 additional challenges because they are ineligible for subsidies, and immigrant family
26 incomes fall below poverty at a much greater rate than non-immigrant family incomes [DOL
27 2024b]. Incarcerated workers also have limited access to healthcare, especially specialized
28 care [Puglisi and Wang 2021].

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Chapter 3: Health Effects

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Key Chapter Takeaways

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- Weight-of-evidence authoritative reviews examining the effects of ambient air pollution show evidence of causal relationships between particulate matter (PM) exposures and several adverse health effects, including cardiovascular and respiratory diseases, nervous system effects, cancer, and non-injury mortality.
- Recent epidemiological studies on associations between wildland fire smoke exposure and physical health effects have corroborated the evidence from the authoritative reviews, primarily on cardiorespiratory diseases, which supports the analogy between hazards from ambient air pollution and wildland fire smoke exposure.
- Experimental studies provide mechanistic evidence of exposure to wildland fire smoke and health effects, such as inflammation and oxidative stress.
- Although research specific to wildland fire smoke hazards is rapidly growing, there is a general lack of research on health effects from occupational exposures related to wildland fires for outdoor workers.

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Chapter 3 is made up of four sections that collectively serve as the hazard identification portion of this hazard review. An objective statement was developed using a Population, Exposure, Comparator, and Outcomes (PECO) framework [Morgan et al. 2018] to guide the process for gathering evidence required for hazard identification:

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This hazard review will evaluate the impact of exposure to wildland fire smoke among farmworkers and other outdoor workers. Considering the evidence base, PM serves as a major

1 *pollutant of concern and as an indicator of exposure to wildland fire smoke as a complex*
2 *mixture. Health effects among exposed populations will be compared to populations with no or*
3 *low exposure to wildland fire smoke. Acute and chronic respiratory, cardiovascular, and other*
4 *conditions will be discussed.*

5 Section 3.1 summarizes the information gathered from previous authoritative reviews,
6 including identifying health effects, and assessing the weight of evidence for a causal
7 relationship between wildland fire smoke exposure and disease. Section 3.2 presents a
8 scoping review of recent epidemiological literature, supplementing the evidence in the
9 authoritative reviews. Section 3.3 describes key information from select toxicity studies.
10 And finally, Section 3.4 presents a summary and conclusions based on the information
11 presented in the previous sections of this chapter.

12 **3.1 Authoritative Reviews**

13 The first step in assessing occupational risk of adverse health effects is hazard
14 identification. Identifying hazards requires a careful assessment of the nature and strength
15 of the evidence on causation, hereafter referred to as the weight of evidence, between
16 wildland fire emissions and adverse health effects. To accomplish this, NIOSH guidance is to
17 first conduct a systematic literature search of relevant evidence streams under a general
18 framework comprising five key steps [NIOSH 2020]:

- 19 1. Define the causal questions of interest and develop criteria for study selection.
- 20 2. Develop a literature search protocol and conduct search.
- 21 3. Review, identify, and select relevant information from evidence streams.
- 22 4. Evaluate and integrate the evidence across studies.
- 23 5. Synthesize and interpret findings.

24 In general, causal inference draws from three primary sources: (1) human epidemiological
25 studies, (2) mechanistic data from in vitro studies, and (3) experimental animal (or in vivo)
26 studies. Ideally, direct evidence from epidemiological studies of workers is preferred for
27 assessing occupational risks, with supporting information from studies of other
28 populations, animal toxicology, and mechanistic data [NIOSH 2020]. However, except for
29 sparse information on wildland firefighters, essentially no information on adverse health
30 effects can be found for other workers exposed to wildland fire smoke. As such, the
31 information that is available is not sufficient to directly identify and assess all occupational
32 health risks.

33 In contrast, a wealth of information exists stemming from large, high-quality, population-
34 based studies examining the effects of ambient air pollution. These include studies on the
35 emissions from several large-scale wildland fires that have occurred worldwide. Given
36 similarities in exposures, this literature is believed highly relevant to assessing health
37 effects in working populations.

38 Health effects associated with air pollution, as well as occupational exposures among
39 firefighters, have been thoroughly reviewed by government agencies and other
40 authoritative bodies using the previously described principles of systematic review (i.e., the

1 five key steps). NIOSH often uses the hazard identification of other authoritative agencies to
2 improve efficiency and avoid a duplication of effort [NIOSH 2020]. As such, this hazard
3 review relies, in part, on the conclusions reached in previous authoritative reviews to
4 identify relevant malignant and nonmalignant health conditions [EPA 2019, 2022; IARC
5 2015, 2023]. These reviews are described next.

6 **3.1.1 Conclusions From Recent Authoritative Reviews**

7 **3.1.1.1 U.S. Environmental Protection Agency**

8 **Integrated Science Assessment for Particulate Matter**

9 In 2019, the U.S. Environmental Protection Agency (EPA) published its Integrated Science
10 Assessment for Particulate Matter (hereafter referred to as the PM ISA) [EPA 2019], which
11 provided the scientific foundation for the National Ambient Air Quality Standard (NAAQS)
12 for PM. This document builds upon several previous reports summarizing decades of health
13 effects research on ambient air pollution. Overall, the PM ISA provides a comprehensive
14 evaluation and synthesis of peer-reviewed studies characterizing PM exposures and
15 associated human health effects.

16 The PM ISA involved multiple scientific disciplines, including epidemiology, toxicology,
17 clinical, and exposure sciences. Although the PM ISA focuses on the effects of PM in ambient
18 air pollution from all sources, EPA acknowledged that fires, including wildfires, prescribed
19 fires, and agricultural fires, are among the greatest contributors to primary PM_{2.5} emissions,
20 accounting for roughly one-third of ambient PM_{2.5} in the United States [EPA 2019].

21 The EPA general framework for evaluating scientific information, including criteria for
22 assessing study quality and developing scientific conclusions, is available in a separate
23 document [EPA 2015]. EPA provided a detailed methodology for their literature search,
24 study selection strategy, and how individual study quality was assessed. These methods
25 consisted of (1) a multitiered, systematic literature search, (2) multiple levels of literature
26 screening (abstract and full text), and (3) peer, public, and scientific advisory committee ISA
27 review [EPA 2019].

28 The literature evaluated in the PM ISA, published from January 1, 2009, through March 31,
29 2017, provided a basis for or described the relationship between PM and health effects,
30 building on the conclusions presented in the 2009 PM ISA [EPA 2009]. The evidence base
31 included both experimental and observational epidemiological studies systematically
32 selected for review, using formal criteria meant to address key policy-relevant questions on
33 causal relationships between PM and adverse health. Individual study quality was evaluated
34 by considering the design, methods, conduct, and documentation of each study.

35 To assess the causal nature of relationships between PM exposure and health, the EPA used
36 a weight-of-evidence approach and made conclusions using a 5-level hierarchy:

- 37 • **Causal relationship**—The evidence is sufficient to conclude that there is a causal
38 relationship between the relevant PM exposure and the health effect of interest;
39 chance, confounding, and other biases could be ruled out with reasonable

1 confidence. Generally, the determination is based on multiple high-quality studies
2 conducted by multiple research groups.

- 3 • **Likely to be a causal relationship**—The evidence is sufficient to conclude that a
4 causal relationship between the relevant PM exposure and the health effect of
5 interest is likely to exist; chance, confounding, and other biases are minimized but
6 uncertainties remain. Generally, the determination is based on consistent findings
7 among multiple high-quality studies.
- 8 • **Suggestive of but not sufficient to infer a causal relationship**—The evidence is
9 suggestive of a causal relationship but is limited, and alternative explanations such
10 as chance, confounding, and other biases cannot be ruled out.
- 11 • **Inadequate to infer the presence or absence of a causal relationship**—The
12 available studies are of insufficient quality, consistency, or statistical power to make
13 any determination regarding the presence or absence of a causal relationship.
- 14 • **Not likely to be a causal relationship**—Several adequate (high-quality) studies
15 examining relationships with relevant exposures are consistent in failing to show an
16 effect at any level of exposure.

17 The categories of health effects assessed in the PM ISA included (1) respiratory effects, (2)
18 cardiovascular effects, (3) metabolic effects, (4) nervous system effects, (5) reproductive
19 and developmental effects, (6) cancer, and (7) total (nonaccidental) mortality (e.g.,
20 mortality excluding accidents, injuries, and other external causes, (see ICD 10 code A00–
21 RR99). Particulate matter exposures were classified within the PM ISA based on size
22 fractions. These are *fine* PM, with nominal mean aerodynamic diameter ≤ 2.5 micrometer
23 (μm) ($\text{PM}_{2.5}$); *thoracic coarse* PM with nominal mean aerodynamic diameter ≤ 10 μm and
24 > 2.5 μm ($\text{PM}_{10-2.5}$); and *ultrafine* particles (UFP) with nominal mean aerodynamic diameter
25 ≤ 0.1 μm [EPA 2019].

26 This hazard review has adopted the same terminology for ease of interpretation; however,
27 it is understood that $\text{PM}_{10-2.5}$ is not the thoracic fraction per se but represents a subfraction
28 of the thoracic fraction (see Section 2.1.2.1). As previously shown in Figure 2–5, the PM_{10}
29 penetration curve closely represents the thoracic fraction curve, although the curves
30 deviate beyond 20 μm particle diameters.

31 Among these categories, the breadth of the evidence on health effects is greatest for $\text{PM}_{2.5}$,
32 followed by $\text{PM}_{10-2.5}$ and UFP, respectively. Note that UFP fall within $\text{PM}_{2.5}$. EPA evaluated
33 the health effects of PM for both short-term (hours up to 1 month) and long-term exposures
34 (1 month to years).

35 The PM ISA focuses on examining the relationship between short- and long-term PM
36 exposure across size fractions and various health effects categories. Across the size
37 fractions examined, the most extensive evidence base exists for $\text{PM}_{2.5}$. This hazard review
38 also finds $\text{PM}_{2.5}$ to be the leading agent of concern from wildland fire smoke, based
39 primarily on the strong likelihood of exposure and strength of evidence of exposure-related
40 adverse health impacts described in the literature. Because $\text{PM}_{2.5}$ is a main component of
41 wildland fire smoke, the conclusions of the PM ISA with respect to $\text{PM}_{2.5}$ are informative in

1 the context of the potential health implications of wildland fire smoke on outdoor workers,
2 and as such are the focus of this section. Nonetheless, outdoor workers may be exposed to
3 other PM size fractions from wildland fire smoke, either as primary emissions or from the
4 formation of secondary particles in the atmosphere. Given this potential, this section also
5 includes brief discussion on the scientific evidence on PM_{10-2.5} and UFP, when available. A
6 brief description of the PM ISA causality determinations by health condition and exposure
7 categories, based on the EPA review of the evidence in the scientific literature, is provided
8 below and is summarized in Table 3–1. In line with the purpose of this hazard review, the
9 following general descriptions were limited to health effects observed primarily in age
10 groups corresponding to adult workers.

11 **Respiratory diseases:** The scientific evidence supports a likely causal relationship
12 between short-term PM_{2.5} exposure and respiratory effects. This determination was based
13 largely on consistent epidemiological evidence supporting exposure-related chronic
14 obstructive pulmonary disease (COPD) exacerbation, respiratory infection, and respiratory
15 mortality. Some evidence was found of an association between short-term PM_{2.5} exposure
16 and lung function decrements and pulmonary inflammation in controlled human exposure
17 studies. Recent epidemiological studies considering the potential for confounding of the
18 PM_{2.5} association by gaseous and particulate copollutants (e.g., nitrogen dioxide [NO₂],
19 nitrogen oxides [NO_x], ozone [O₃], and carbon monoxide [CO]) showed that the presence of
20 other agents had very little effect on associations between PM and asthma exacerbation,
21 combined respiratory-related diseases, or respiratory mortality.

22 The relationship between short-term PM_{10-2.5} and UFP and respiratory effects was
23 suggestive of but not sufficient to infer a causal relationship, based on some epidemiological
24 evidence of respiratory infection and other respiratory effects, excluding COPD.

25 The evidence supports a likely causal relationship between long-term PM_{2.5} exposure and
26 respiratory effects. This determination was based largely on consistent evidence from
27 multiple epidemiological studies reporting positive associations between exposure and
28 respiratory mortality, COPD, and respiratory infection and coherence with other cause-
29 specific respiratory mortality. Among noted limitations, few studies looked at the effects of
30 co-pollutant confounding on respiratory illnesses. However, animal toxicological studies
31 provided supporting evidence of inflammation and airway morphological changes and PM_{2.5}
32 -induced oxidative stress. The evidence of a causal relationship between long-term PM_{10-2.5}
33 and UFP and respiratory effects was inadequate.

34 **Cardiovascular diseases:** The evidence supports that a causal relationship exists between
35 short-term PM_{2.5} exposure and cardiovascular effects. Epidemiological studies provided
36 strong evidence of consistent positive associations between short-term PM_{2.5} exposures and
37 cardiovascular-related mortality, hospital admissions, and emergency department visits.
38 Coherence in biological plausibility was seen in epidemiology, experimental animals, and
39 mechanistic data. Consistent links were shown connecting short-term PM_{2.5} exposures to
40 ischemic heart disease, heart failure, arrhythmias, and thrombosis. There was little
41 evidence of strong confounding by copollutants. The relationship between short-term

1 PM_{10-2.5} and UFP exposures and cardiovascular effects were suggestive of but not sufficient
2 to infer a causal relationship.

3 The evidence supports that a causal relationship exists between long-term PM_{2.5} exposure
4 and cardiovascular effects, based largely on consistent, positive associations of recent U.S.-
5 based cohort epidemiological studies. Studies on cardiovascular morbidity provided
6 additional evidence of association by considering specific demographic populations and
7 evaluating for confounders such as socioeconomic status. As in short-term PM_{2.5} exposure,
8 adjusted copollutant models for long-term PM_{2.5} did not indicate a potential for strong
9 confounding of the association by copollutants. The relationship between long-term PM_{10-2.5}
10 and cardiovascular effects were suggestive of but not sufficient to infer a causal
11 relationship, and inadequate for any determination on UFP.

12 **Metabolic diseases:** The evidence of a causal relationship between short-term PM_{2.5} and
13 metabolic effects is suggestive of but not sufficient to infer a causal relationship. In general,
14 there was limited evidence from a small number of epidemiological and toxicological
15 studies mostly reporting effects on glucose and insulin homeostasis and other indicators of
16 metabolic function such as inflammation in the visceral adipose tissue and liver. The
17 evidence of a causal relationship between short term PM_{10-2.5} and UFP was inadequate.

18 The evidence of a causal relationship between long-term PM_{2.5} or PM_{10-2.5} and metabolic
19 effects was suggestive of but not sufficient to infer a causal relationship. The determination
20 was based primarily on epidemiological studies reporting positive associations between
21 long-term PM_{2.5} exposure and diabetes-related mortality, as well as long-term PM_{10-2.5}
22 exposure and incident diabetes and cross-sectional studies of glucose and insulin
23 homeostasis in European cohorts. The evidence on a causal relationship between long-term
24 UFP and metabolic effects was inadequate.

25 **Nervous system disorders:** The evidence supports a likely causal relationship between
26 long-term PM_{2.5} exposure and nervous system effects based largely on animal toxicological
27 and epidemiological studies. These studies provide evidence of associations between long-
28 term PM_{2.5} exposures and brain morphology changes, cognitive decline, and dementia. EPA
29 also found limited epidemiological evidence of neurodevelopmental effects with support by
30 animal studies showing PM_{2.5} -induced inflammatory and morphological changes in specific
31 areas of the brain. The relationship between long-term PM_{10-2.5} and UFP with nervous
32 system effects were suggestive of but not sufficient to infer a causal relationship. Regarding
33 short-term exposure, the evidence on PM_{2.5} and UFP was suggestive of but not sufficient to
34 infer a causal relationship, and inadequate for any determination on short-term PM_{10-2.5}.

35 **Reproductive and developmental effects:** The PM ISA reported separately on (1) male
36 and female fertility and reproduction, and (2) pregnancy and birth outcomes, because these
37 groups have differing etiologies and relevant exposure periods within life stages. The
38 evidence of causal relationships between PM_{2.5} and pregnancy, birth outcomes,
39 reproductive, and fertility effects was suggestive of but not sufficient to infer a causal
40 relationship between exposure and any outcome investigated. In general, there was limited
41 evidence from studies on low birth weight and other developmental outcomes observed

1 during the post-neonatal period. There was also limited evidence in epidemiological studies
2 suggesting exposure-related decreases in sperm motility, in vitro fertilization success, and
3 fecundability; however, questions remained concerning copollutant confounding and other
4 sources of uncertainty. The evidence of a causal relationship between PM_{10-2.5} or UFP and
5 reproductive and developmental effects was inadequate.

6 **Cancer:** The evidence supports a likely causal relationship between long-term PM_{2.5}
7 exposure and cancer. This determination is based largely on the support of biologic
8 plausibility from animal experiments; mechanistic information showing that PM_{2.5} is
9 genotoxic and induces oxidative stress, electrophilicity, and epigenetic alterations, and
10 consistent evidence from epidemiological studies showing positive associations in the risk
11 of lung cancer incidence and mortality. The latter suggests a linear, no-threshold exposure-
12 response relationship. The PM ISA noted positive associations between PM_{2.5} and other
13 cancers (e.g., cancers of the breast, liver, brain, and leukemia); however, fewer studies were
14 available. The relationship between PM_{10-2.5} and cancer was suggestive of but not sufficient
15 to infer a causal relationship, and for UFP evidence was inadequate for any determination.

16 **Nonaccidental mortality:** The evidence supports a causal relationship between short-term
17 PM_{2.5} exposure and nonaccidental mortality. This determination was based primarily on
18 recent epidemiological evidence from multicity studies conducted in the United States,
19 Canada, Europe, and Asia, in combination with single-and multicity studies previously
20 evaluated in the 2009 PM ISA. These studies consistently found positive associations
21 between PM_{2.5} exposures and nonaccidental mortality. There was consistent and coherent
22 evidence of exposure-related cardiovascular morbidity, along with lesser evidence of
23 respiratory morbidity, that supported the biologic plausibility of increased total mortality
24 through cardiorespiratory mortality. The relationship between short-term PM_{10-2.5} and total
25 mortality were suggestive of but not sufficient to infer a causal relationship, and inadequate
26 for UFP.

27 Both the 2009 and 2019 PM ISAs concluded the evidence supported a causal relationship
28 between long-term PM_{2.5} exposure and nonaccidental mortality. The 2019 PM ISA
29 determination was based on new cohort studies and reanalysis of the previous large cohort
30 studies, which when taken together consistently demonstrated positive associations for
31 cause-specific mortality. This includes mortality from lung cancer and cardiorespiratory
32 effects. There was evidence of cardiovascular and respiratory morbidity that contributed to
33 the biological plausibility for total mortality from long-term PM_{2.5} exposure. Studies also
34 showed decreases in long-term PM_{2.5} exposures resulted in increased life expectancy. The
35 evidence is suggestive of, but not sufficient, to infer a causal relationship between long-term
36 PM_{10-2.5} and total mortality. UFP evidence was inadequate for any determination.

37 In 2022, as part of the reconsideration of the 2020 PM National Ambient Air Quality
38 Standard, EPA published a supplement to the 2019 PM ISA, which consisted of a targeted
39 assessment of studies published since the literature cutoff date of the 2019 PM ISA [EPA
40 2022]. While the 2022 supplement did not comprehensively review new literature, it
41 evaluated relevant studies conducted in the United States and Canada since the 2019 PM
42 ISA. These evaluations judged whether new evidence supported (was consistent with),

1 supported and extended (was consistent with and improved internal validity), or did not
 2 support (was not consistent with) the causality determinations in the 2019 PM ISA. Overall,
 3 the supplement provided further support and, in some cases, extended the support for the
 4 previous causal determinations on cardiovascular effects and mortality. The supplement
 5 also extended the evidence base that showed disparities in PM-related health effects among
 6 minority populations and persons of lower socioeconomic status. However, the supplement
 7 does not describe a full multidisciplinary evaluation of evidence that results in new weight-
 8 of-evidence conclusions (i.e., causality determinations).

9 While the PM ISA provides information on the potential health effects of exposure to PM at
 10 the population-level, much of the information is also relevant to potential health effects
 11 among outdoor workers exposed to wildfire smoke. The consideration of exposure duration
 12 and PM size fraction is equally relevant to workers as it is to community members when
 13 evaluating health effect outcomes.

14 **Table 3-1. Summary of EPA causality determinations for PM exposure and health**
 15 **outcomes. Adapted from the Integrated Science Assessment for Particulate Matter**
 16 **[EPA 2019]**

Health Effect	Exposure duration*	Particle size†	Causality determination
Respiratory	Short-term	PM _{2.5}	Likely to be causal
Respiratory	Short-term	PM _{10-2.5}	Suggestive of, but not sufficient to infer
Respiratory	Short-term	UFP	Suggestive of, but not sufficient to infer
Respiratory	Long-term	PM _{2.5}	Likely to be causal
Respiratory	Long-term	PM _{10-2.5}	Inadequate
Respiratory	Long-term	UFP	Inadequate
Cardiovascular	Short-term	PM _{2.5}	Causal
Cardiovascular	Short-term	PM _{10-2.5}	Suggestive of, but not sufficient to infer
Cardiovascular	Short-term	UFP	Suggestive of, but not sufficient to infer
Cardiovascular	Long-term	PM _{2.5}	Causal
Cardiovascular	Long-term	PM _{10-2.5}	Suggestive of, but not sufficient to infer
Cardiovascular	Long-term	UFP	Inadequate
Metabolic	Short-term	PM _{2.5}	Suggestive of, but not sufficient to infer
Metabolic	Short-term	PM _{10-2.5}	Inadequate
Metabolic	Short-term	UFP	Inadequate
Metabolic	Long-term	PM _{2.5}	Suggestive of, but not sufficient to infer
Metabolic	Long-term	PM _{10-2.5}	Suggestive of, but not sufficient to infer
Metabolic	Long-term	UFP	Inadequate
Nervous system	Short-term	PM _{2.5}	Suggestive of, but not sufficient to infer
Nervous system	Short-term	PM _{10-2.5}	Inadequate
Nervous system	Short-term	UFP	Suggestive of, but not sufficient to infer
Nervous system	Long-term	PM _{2.5}	Likely to be causal
Nervous system	Long-term	PM _{10-2.5}	Suggestive of, but not sufficient to infer

Health Effect	Exposure duration*	Particle size†	Causality determination
Nervous system	Long-term	UFP	Suggestive of, but not sufficient to infer
Fertility and reproduction	NA	PM _{2.5}	Suggestive of, but not sufficient to infer
Fertility and reproduction	NA	PM _{10-2.5}	Inadequate
Fertility and reproduction	NA	UFP	Inadequate
Pregnancy and birth outcomes	NA	PM _{2.5}	Suggestive of, but not sufficient to infer
Pregnancy and birth outcomes	NA	PM _{10-2.5}	Inadequate
Pregnancy and birth outcomes	NA	UFP	Inadequate
Cancer	Long-term	PM _{2.5}	Likely to be causal
Cancer	Long-term	PM _{10-2.5}	Suggestive of, but not sufficient to infer
Cancer	Long-term	UFP	Inadequate
Nonaccidental mortality	Short-term	PM _{2.5}	Causal
Nonaccidental mortality	Short-term	PM _{10-2.5}	Suggestive of, but not sufficient to infer
Nonaccidental mortality	Short-term	UFP	Inadequate
Nonaccidental mortality	Long-term	PM _{2.5}	Causal
Nonaccidental mortality	Long-term	PM _{10-2.5}	Suggestive of, but not sufficient to infer
Nonaccidental mortality	Long-term	UFP	Inadequate

1 * Short-term exposure occurs for a period of hours up to 1 month. Long-term exposure occurs for a
2 period lasting from 1 month to years.

3 † PM_{2.5}, particulate matter with a nominal mean aerodynamic diameter less than or equal to 2.5
4 micrometers (µm); PM_{10-2.5}, particulate matter with a nominal mean aerodynamic diameter greater than
5 2.5 µm and less than or equal to 10 µm; UFP, ultrafine particulate matter.

6 **3.1.1.2 Comparative Assessment of the Impacts of Prescribed Fire** 7 **Versus Wildfire (CAIF): A Case Study in the Western United States**

8 In 2021, EPA published its comparative assessment of the impacts of prescribed fire versus
9 wildfire in the Comparative Assessment of the Impacts of Prescribed Fire Versus Wildfire
10 (CAIF). The assessment included a limited synthesis of the health effects associated with
11 wildland fire, both prescribed and wildfire [EPA 2021]. It also pointed to emerging evidence
12 on health effects from wildfire smoke that appeared consistent with the assessment of
13 effects from ambient air pollution described previously in EPA's PM ISA. Within this
14 assessment, EPA noted that most evidence regarding the health effects of wildfire smoke

1 exposure was from studies examining short-term exposures over single-day or multiday
2 lags ranging from 0 to 5 days.

3 The CAIF assessment noted a lack of data on (1) health effects due to repeated wildfire
4 smoke exposures over days, weeks, or months; (2) the long-term late onset or persistent
5 health effects from a single wildfire event; and (3) health effects due to protracted wildfire
6 exposures lasting over many months and multiple fire seasons. Finally, EPA noted limited
7 information from existing epidemiologic studies on the wildfire smoke exposure
8 concentration-response relationship; therefore, the quantitative assessment relied
9 primarily on exposure-response functions from studies of ambient PM_{2.5} to estimate the
10 health burden from wildland fire emissions. This decision points to uncertainty in
11 quantifying health risks from wildland fires, which is an important area of investigation for
12 future risk assessment.

13 **3.1.1.3 Monographs of the International Agency for Research on Cancer**

14 The International Agency for Research on Cancer (IARC) of the World Health Organization
15 (WHO) conducts comprehensive scientific assessments of the literature pertaining to
16 potential human carcinogens. The methods and findings from these assessments are
17 published in its series: *IARC Monographs on the Identification of Carcinogenic Hazards to*
18 *Humans* (formerly *Monographs on the Evaluation of Carcinogenic Risks in Humans*). These
19 monographs serve as hazard identification, which is the first step in support of future risk
20 assessments. However, they do not quantify risks from carcinogenic exposures.

21 In each monograph, the framework for the evaluation is documented in its preamble.
22 Methods and classifications have differed slightly between monographs over the years, but
23 recent publications have generally adhered to the process briefly described here. First,
24 agents are selected for critical review by a multidisciplinary Working Group of subject
25 matter experts, provided that some evidence of human exposure and carcinogenicity exists.
26 The literature is then systematically searched to identify all pertinent epidemiological,
27 toxicological, and exposure studies with information on the potential causal relationship of
28 interest. Another Working Group reviews the identified studies by applying the principles of
29 systematic review in screening, synthesis, and evaluation of evidence, including study
30 quality. The Working Group assesses the weight of evidence, with an emphasis on exposure
31 characterization, cancer in humans, cancer in experimental animals, and supportive
32 mechanistic data. The Working Group then integrates all evidence streams, following a
33 prescribed rationale, and selects one of the following weight-of-evidence classifications:

- 34 • The agent is carcinogenic to humans (Group 1).
- 35 • The agent is probably carcinogenic to humans (Group 2A).
- 36 • The agent is possibly carcinogenic to humans (Group 2B).
- 37 • The agent is not classifiable as to its carcinogenicity to humans (Group 3).

38 Group 1 human carcinogens are those agents with the greatest evidence of a causal
39 relationship between exposure to the agent and cancer. A Group 1 classification means
40 there is sufficient evidence of a causal relationship in human epidemiological studies such
41 that alternative explanations (e.g., chance, bias, confounding) can be ruled out with

1 reasonable confidence. Alternatively, agents are classified Group 1 with a combination of
2 limited or inadequate evidence in human epidemiological studies, sufficient evidence in
3 experimental animals, and strong mechanistic evidence from studies of exposed humans.

4 In 2015, IARC published its monograph on outdoor air pollution [IARC 2015]. In its
5 assessment, the Working Group examined substantial literature on a wide variety of air
6 pollution sources, including PM. It was clear that the large variability in outdoor air
7 pollution mixtures hinders carcinogenic classification. However, the Working Group
8 concluded that many carcinogenic mechanisms are shared among pollutants, of which PM
9 appeared to dominate most sources. Therefore, the Working Group determined that
10 sufficient evidence exists to conclude that outdoor air pollution and PM are carcinogenic to
11 humans (Group 1), based on sufficient evidence in humans and experimental animals, as
12 well as strong mechanistic evidence. Regarding human studies, there was sufficient
13 epidemiological evidence of lung carcinogenicity and some evidence of positive associations
14 with bladder cancer.

15 In 2023, IARC published its monograph on occupational exposure as a firefighter [IARC
16 2023]. The Working Group determined evidence sufficient in humans for the
17 carcinogenicity of occupational exposure as a firefighter (Group 1). The Working Group
18 concluded this because they found sufficient evidence of a causal relationship between
19 firefighter exposure and mesothelioma and bladder cancer.

20 Consistent positive associations were also observed between firefighter exposure and
21 cancers of the colon, prostate, and testis, malignant melanoma of the skin, and non-Hodgkin
22 lymphoma. Strong mechanistic evidence indicated that firefighter exposures are genotoxic;
23 induce epigenetic alterations, oxidative stress, and chronic inflammation; and modulate
24 receptor-mediated effects. These are all considered key characteristics of carcinogens in
25 exposed humans [IARC 2023].

26 Notably, the evidence base was not broad enough to differentiate the hazard classifications
27 between types of firefighters or fires, such as structural, wildland, industrial, aircraft, and
28 marine. The degree to which the IARC monograph [2023] accurately reflects the effects of
29 occupational exposures to wildland fire emissions is uncertain, as the available evidence
30 comes mainly from large epidemiological studies of structural firefighters. However,
31 because smoke exposure is a primary concern related to cancer among all firefighters, for
32 the purposes of the current hazard review, the monograph on occupation as a firefighter
33 was deemed relevant when considering health effects from occupational exposure to
34 wildland fire smoke.

35 **3.1.1.4 World Health Organization Global Air Quality Guidelines**

36 In 2021, the WHO published its most recent health-based authoritative recommendations
37 on air quality guidelines [WHO 2021]. The guidelines were developed, in part, from
38 information from multiple systematic reviews and meta-analyses. These studies looked at
39 the health effect estimates of short- and long-term exposures to different sources of
40 ambient air pollution. The evaluation included thorough assessments of study quality that
41 enabled researchers to estimate the level of uncertainty in cause-and-effect associations.

1 Associations with high confidence were considered most informative for air quality
2 recommendations. Although wildfire emissions were acknowledged as a potential source,
3 no studies of the health effects associated with wildland fires appear to have been evaluated
4 for the WHO recommendations.

5 The WHO-sponsored systematic reviews are published in an appendix to its report and
6 separately in the peer-reviewed literature [Chen and Hoek 2020; Huangfu and Atkinson
7 2020; Lee et al. 2020; Orellano et al. 2020; Orellano et al. 2021; Perez Velasco and
8 Jarosinska 2022; WHO 2021; Zheng et al. 2021]. The report's conclusions about PM
9 exposures were generally aligned with those found in EPA's 2019 PM ISA. This is expected
10 given the large overlap in the review of available literature.

11 Briefly, the WHO studies found clear evidence that long-term exposure (defined as months
12 to years) to PM_{2.5} and PM₁₀ was associated with increased mortality from all causes,
13 cardiovascular disease, respiratory disease, and lung cancer [Chen and Hoek 2020]. For
14 short-term exposures (defined as 1 hour to days), the evidence supported associations
15 between PM_{2.5} and PM₁₀ exposures for all-cause mortality and cardiovascular, respiratory,
16 and cerebrovascular mortality [Orellano et al. 2020].

17 **3.2 NIOSH Scoping Review**

18 **3.2.1 Approach to Evidence Synthesis**

19 This section presents results from the NIOSH scoping review of the relevant epidemiological
20 literature to supplement evidence in the authoritative reviews described in Section 3.1.
21 Previous authoritative reviews from other agencies addressed slightly different topics than
22 those in the scope of this hazard review, which relates to wildland fire smoke exposure
23 among outdoor workers. For example, the EPA's Integrated Science Assessment for
24 Particulate Matter [EPA 2019, 2022] and the IARC's evaluation of Outdoor Air Pollution
25 [IARC 2015] included all sources of PM, not just from wildland fire smoke. Recent work by
26 IARC evaluated occupational exposures, including a range of smoke byproducts, among
27 firefighters [IARC 2023].

28 This scoping review supplements existing information from authoritative reviews by
29 identifying the recent epidemiological literature relevant to wildland fire smoke exposure
30 and adverse health effects. It pertains mostly to how findings can potentially be generalized
31 to occupational populations exposed to wildland fire emissions.

32 For this scoping review, a large and varied body of research was characterized thematically,
33 rather than to answer specific research questions [Munn et al. 2022]. This scoping review
34 characterized the following:

- 35 • The populations of outdoor workers that have been studied in the relevant
36 literature.
- 37 • The range of health effects studied in the relevant literature and how it compares
38 with those evaluated in previous authoritative reviews.

- The hazards in wildland fire smoke (including PM and other measures) and how they are characterized in the relevant literature, in light of wildland fire smoke containing a mixture of contaminants.
- The research gaps that remain in understanding the health effects of exposure to wildland fire smoke, including occupational exposure.

Studies in this scoping review were synthesized according to the search strategy and described in terms of the characteristics (including populations evaluated, exposure metrics of wildland fire smoke ascertained, and associated health conditions studied) needed to address the objectives mentioned above.

3.2.1.1 Search Strategy

To systematically gather epidemiological studies examining the health impacts of wildland fire smoke exposure, a comprehensive search was conducted of the scientific literature, including articles, reviews, and errata. The scope of this review was confined to works published from January 1, 2017, through February 8, 2024. The start date was chosen to ensure capture of literature following the EPA’s comprehensive, systematic literature review on the health effects of exposure to PM for its Integrated Science Assessment [EPA 2019].

Because a large portion of studies on wildland fire smoke use PM to measure exposure [Adetona et al. 2016], and because wildland fire is one of the major contributors to ambient PM in the United States [EPA 2019], NIOSH concluded that EPA’s PM ISA review sufficiently characterized the hazards posed by exposure to PM from wildland fire smoke available in the literature prior to 2017. Additionally, the availability of relevant health effects research is increasing in step with a growing wildland fire threat; therefore, literature published after the PM ISA review has a greater potential to further the understanding of health conditions related to wildland fires.

For this scoping review, the reference databases Scopus, PubMed, and Embase were searched for publications containing specified terms in title or abstract fields. Language restrictions were not implemented during the database search, but rather during article review. The specified search terms were chosen to capture a broad selection of potentially-relevant epidemiological studies and included the following:

[[Exposure* OR exposed OR assessment* OR emission* OR safety OR health OR “particulate matter” OR “pm 2.5” OR “pm2.5” OR “pm 10” OR “pm10” OR smoke OR Occupation OR occupations OR occupational OR occupationally OR workplace OR workplaces OR worksite OR worksites OR worker OR workers OR workforce OR employee OR employees OR “at work” OR job OR jobs OR “job related” OR “work related” OR “occupational exposure” OR “occupational exposures” OR “occupationally exposed” OR “workplace exposure” OR “workplace exposures” OR fire OR personnel OR “fire management”] AND [Wildland OR wildfire* OR “prescribed fire*” OR “prescribed burn*” OR “controlled fire*” OR “controlled burn*” OR “forest fire*” OR “wildland urban interface” OR “WUI” OR “brush fire*” OR brushfire OR “bush fire*” OR bushfire OR “vegetation fire”]].

1 The resulting set of publications was imported and de-duplicated using the Covidence and
2 EndNote computer programs [Clarivate 2023; Veritas Health Innovation 2023]. A panel of
3 NIOSH occupational health professionals conducted three rounds of review to determine
4 each study's eligibility for inclusion in the scoping review based on specified eligibility
5 criteria. The three rounds of review included (1) title and abstract screening (two reviewers
6 per article), (2) full-text review (two reviewers per article), and (3) data extraction, which
7 involved an additional round of full-text review and an extraction of study characteristics
8 for discussion in the scoping review (one extractor per article). An additional reviewer
9 provided resolution for discordant ratings and group conferrals occurred as needed.

10 Study eligibility was based on the following inclusion criteria and presented in Table 3–2:

11 Eligible studies were English language, peer-reviewed human analytic studies that provide
12 information from a statistical test or tests of the association between a measure or proxy of
13 wildland fire smoke and acute or chronic physical disease or its manifestations in adults of
14 working ages.

15 The following definitions applied:

- 16 • **Peer-reviewed**—This criterion excludes commentaries, letters, news articles,
17 editorials, websites, and technical reports.
- 18 • **Human analytic studies**—Epidemiological studies excluding descriptive studies (case
19 series, case reports, routine surveillance studies), animal studies, and in vitro studies.
20 Cross-sectional studies that provided information on the exposure-response association
21 were considered eligible for inclusion. Human analytic studies may include original
22 experimental, quasi-experimental, or observational designs, as well as meta-analyses
23 reporting summary measures of association.
- 24 • **A measure of association between exposure and health effect**—Studies must
25 include measures of both the exposure and a physical health condition or conditions in
26 the population under study, which are then related in a statistical analysis to estimate
27 an exposure-response.
- 28 • **Acute or chronic physical disease or its manifestations**—Physical diseases exclude
29 injury and disorders related to psychological, emotional, and mental health. Eligible
30 outcome measures include (1) self-reported conditions, (2) clinical or overt measures
31 such as disease diagnosis or mortality, and (3) measures considered subclinical, such as
32 biomechanistic or cellular changes, symptomology, lung function, or metabolic
33 outcomes. The physical health restriction was made in efforts to focus the review on
34 conditions aligned with the PM ISA and should not be construed as a lack of evidence of
35 other health conditions that may be related to wildland fire smoke. For example,
36 growing evidence suggests trauma-related declines to mental health and well-being
37 among exposed persons [Eisenman and Galway 2022].
- 38 • **Adults of working ages**—These terms exclude studies that primarily study children
39 under the age of 18 or adults 65 years or older. The exception are studies of prenatal or
40 *in utero* exposure, where the exposure of interest is to adults of working ages. The age
41 criterion is intended to focus the review on working populations per NIOSH guidance

1 [NIOSH 2020]; however, it is acknowledged that the affected workforce includes a small
2 proportion of workers outside the age range of 18–65 years.

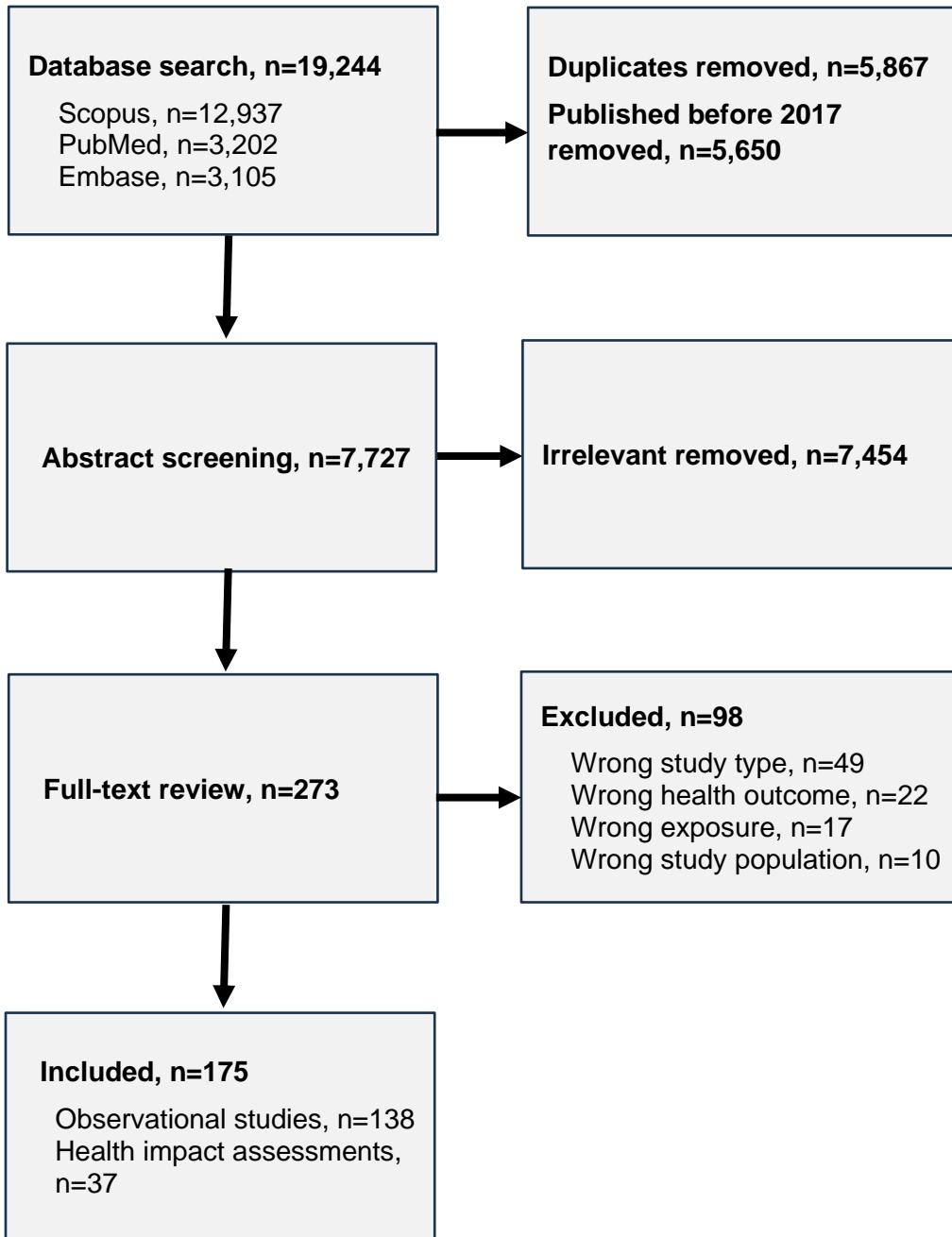
3 Studies were not restricted to outdoor worker populations or non-firefighting outdoor
4 workers because (1) few studies were anticipated to be available on wildland fire smoke
5 exposure on non-firefighting outdoor workers [Koopmans et al. 2022; Navarro 2020]; (2)
6 non-firefighting outdoor workers are exposed at frequencies, durations, and doses that
7 likely span between general population exposures and heavy exposures among wildland
8 firefighters [Adetona et al. 2016; Navarro 2020; Zuidema 2022]; (3) studies on general
9 population exposures do not necessarily exclude workers occupationally exposed within
10 the community; and (4) outdoor workers' exposure might continue even off duty if they
11 remain within the exposed community or via take-home exposure, discussed in Section
12 2.3.4.

1

Table 3-2. Study eligibility criteria for inclusion in the scoping review

Component	Inclusion	Exclusion
Population	<p>Adult (aged 18–65 years) human studies, including workers or residents.</p> <p>Studies of infants or children exposed prenatally or in utero, where the exposure of interest is presumed to be adults of working ages.</p>	<p>Studies of only nonworking-aged participants, such as children exposed postnatally or adults 65 years of age or older.</p> <p>Nonhuman study populations, including plants, fungi, wildlife, livestock, viruses, bacteria, experimental animals, and fire characteristics.</p> <p>Toxicology studies of human cells or tissue, such as in vitro studies.</p>
Exposure	<p>Measures or estimates of exposure to wildland fire smoke from sources such as wildfires, prescribed fires, wildland-urban interface fires, forest fires, and bushfires. This includes exposure to particulate matter or other constituents of smoke, occupational classifications, or other proxies of exposure.</p>	<p>No measure, estimate, or surrogate of exposure from a wildland fire smoke source.</p>
Outcome	<p>Measures of acute or chronic physical disease or its manifestations, including self-reported, clinical, or overt conditions such as disease diagnosis and mortality. This also includes subclinical conditions such as biomechanistic or cellular changes, symptomology, lung function, and metabolic outcomes.</p>	<p>No measured health effect.</p> <p>Conditions of abnormal psychological, emotional, and mental status.</p> <p>Safety, traumatic injury, and musculoskeletal outcomes.</p> <p>Health outcomes with no body system or condition identified.</p>
Study type	<p>Peer-reviewed studies including original experimental, quasi-experimental, or observational designs and meta-analyses reporting summary measures of association.</p> <p>Studies that statistically test the association or associations between health effects and exposure to wildland fire smoke.</p>	<p>Full text not in English.</p> <p>Non-peer-reviewed literature.</p> <p>descriptive studies (e.g., case or case-series)</p>

2



1

2 **Figure 3–1. Flow diagram of studies included in the scoping review**

3

3.2.2 Epidemiological Evidence

The number of studies included and excluded in each round of review are displayed in the PRISMA diagram in Figure 3–1. After removing duplicates, the search yielded 13,377 articles. Nearly 60% of these were potentially eligible articles published in 2017 or after (n=7,727) (Figure 3–1), spanning topics related to epidemiology, environmental science, ecology, biology, fire management, toxicology, and others. Title and abstract screening reduced the pool of potential studies to 273 for full text screening after removal of 7,454 irrelevant studies, i.e., those studies easily identified as lacking information aligned with criteria for this scoping review. Additional exclusions following full-text review resulted in 175 studies eligible for synthesis (a full list of references included in the scoping review can be found in Appendix A). Among eligible studies, nearly 75% were published since 2021 (n=127), indicating the increasing concern and importance of health conditions associated with wildland fires. The emergence of relevant research is likely the consequence of recent large-scale fires, particularly those occurring within the wildland-urban interface, which are growing more frequent, intense, and expansive (discussed further in Chapter 1).

Most studies were cross-sectional or quasi-experimental designs, such as time-series, case-crossover designs (n=111). These studies are generally better suited to examine acute health effects under short-term transient exposure conditions, such as effects observed within hours up to a month following exposure. Seventeen longitudinal cohort studies and four case-control studies were identified, which are preferred for evaluating long-term effects. Six meta-analyses were reviewed. This set of 138 studies are hereafter referred to as the relevant set of “observational studies,” which were the focus for outcome-specific synthesis of the information on the association between wildland fire emissions and physical health.

An additional 37 health impact assessments (HIAs) predicted health and economic impacts from wildland fires in models using previously published exposure-response functions. By design, HIAs do not add to the body of evidence on a causal relationship between wildland fire smoke and adverse health conditions; however, they can provide information that may benefit future decision-making regarding risk mitigation. Therefore, due to the unique nature of HIAs, they were described as a separate group in this scoping review.

Among observational studies, few (15%) examined working populations. Twenty studies evaluated exposure-related health effects among firefighters, focusing primarily on wildland firefighters but also including some structural firefighters who responded to incidents related to wildland fires. Only one study examined a non-firefighting occupational group (i.e., police officers) [Moitra et al. 2021]. Subgroups of interest within general population studies (n=117) varied, comprising mostly community residents (n=90). Some studies examined potentially vulnerable populations, such as patient populations (n=4) and pregnant individuals or offspring of exposed adults (n=21).

Over 90% of observational studies evaluated the effects of wildfire smoke or unspecified wildland fire sources of smoke (n=128); fewer evaluated prescribed fires (n=10). Half of the studies on prescribed fire sources of smoke evaluated firefighters [Adetona et al. 2017;

1 Adetona et al. 2019; Wu et al. 2020; Wu et al. 2021a; Wu et al. 2021b], while the other half
2 evaluated nonoccupational populations [Jones and Berrens 2021; Kondo et al. 2022; Lai et
3 al. 2022; Lankaputhra et al. 2023; Pennington et al. 2023].

4 Studies employed various exposure definitions, with 67% reporting at least one
5 quantitative estimate of the airborne concentration of PM, primarily PM_{2.5} and PM₁₀, or
6 chemicals from wildfire emissions, most commonly carbon monoxide (CO) or ozone (O₃).
7 Among quantitative exposure data, estimates of PM_{2.5} concentrations appeared most often,
8 with some information available in over 60% of studies reviewed. Quantitative exposure
9 estimates were used in statistical analyses to estimate risks per unit exposure in about 49%
10 of studies.

11 The remainder examined exposure-response associations using metrics derived from
12 emission quantities, such as ordinal PM_{2.5} exposure categories, or exposed-days based on a
13 set PM_{2.5} threshold, or used surrogate measures of smoke exposure, including burn area,
14 smoke density, and occupation, among others. The complexity of exposure metrics varied
15 markedly among studies, ranging from describing exposure potential using occupational
16 status (e.g., wildland firefighter, yes/no) to deriving latent variables using principal
17 components analysis of multiple combinations of fire pollutant and meteorological data.

18 Some study designs used methods to assess exposure that enabled targeted estimates of the
19 effects attributable to wildfire smoke specifically, while others did not distinguish between
20 anthropogenic and wildfire sources beyond restriction of exposure to a period
21 corresponding with a wildfire. Few studies obtained individual-level measurements of
22 exposure via personal sampling or biomonitoring; however, this level of information was
23 more common among smaller studies of wildland firefighters than in other populations.

24 Observational studies evaluated various health outcomes:

- 25 • 51% (n=70) respiratory health conditions
- 26 • 36% (n=50) cardiovascular conditions
- 27 • 17% (n=24) reproductive and developmental conditions
- 28 • 10% (n=14) infectious diseases
- 29 • 8% (n=11) sub-clinical changes
- 30 • 4% (n=6) neurological conditions
- 31 • 4% (n=6) metabolic conditions
- 32 • 2% (n=4) cancer
- 33 • 2% (n=3) all-cause mortality only
- 34 • 3% (n=5) other health outcomes

35 Studies evaluated health outcomes including both morbidity (n=115) and mortality (n=28)
36 endpoints. Among studies evaluating mortality, all but three assessed cause-specific
37 outcomes in addition to total (all-cause) mortality. For this review, mortality studies that
38 assessed associations with more detailed causes of death were described by the specific
39 health outcomes they assessed, such as respiratory deaths or cardiovascular deaths. Studies
40 with specific and aggregate measures of morbidity, such as emergency department visits or

1 hospital admissions, were treated similarly. Three studies reporting only on total mortality
2 were described separately in Section 3.2.2.9.

3 Table 3–3 presents aggregate information related to the specific health conditions,
4 populations, and exposure metrics evaluated, as well as the presence of any evidence for
5 risk associations. Note that the interpretation and criteria for statistical significance may
6 vary across studies, as critical values for significance tests and confidence intervals can
7 differ between studies. However, a significance level, α , equal to 0.05, was used most often
8 in the studies reviewed. For consistency, this review adopted $\alpha=0.05$ ($P<0.05$) for
9 describing the evidence of associations described in Table 3–3. Likewise, where studies
10 presented only confidence intervals for measures of effect, associations were included in
11 the aggregation of results in Table 3–3, where the confidence intervals did not include the
12 null value.

13 Below are descriptions of study characteristics by the major health outcome categories
14 evaluated in the set of observational studies.

15 **3.2.2.1 Respiratory Health**

16 In this scoping review, 70 studies evaluated respiratory health effects associated with
17 wildland fire smoke. Among the studies, a majority ($n=45$) investigated a composite of
18 respiratory diseases and symptoms. Many of these studies also separately evaluated specific
19 health effects, including asthma, COPD, and individual respiratory symptoms such as cough,
20 sore throat, and difficulty breathing.

21 Studies commonly measured hospitalizations ($n=21$) and emergency department visits
22 ($n=27$). Most studies evaluated exposure to wildfire smoke using $PM_{2.5}$. Studies evaluating
23 asthma ($n=30$) and COPD or other chronic lower airway disease ($n=17$) frequently showed
24 a significant increase associated with wildland fire smoke exposure. Specifically, 87% of
25 studies on asthma and 71% of studies on COPD or other chronic lower airway diseases
26 reported significant increases in these outcomes.

27 Other respiratory health effects frequently evaluated included respiratory infections
28 ($n=13$), lung function ($n=12$), pneumonia ($n=11$), respiratory symptoms ($n=8$), and
29 cardiorespiratory events ($n=4$). Results from these outcomes were largely mixed. For
30 example, significant increases following wildfire smoke exposure were reported by some
31 studies for acute respiratory infections ($n=6$), respiratory symptoms ($n=4$), and pneumonia
32 ($n=2$).

33 Eleven studies evaluated respiratory mortality attributable to wildland fire smoke. Of these,
34 55% ($n=6$) reported increased mortality related to respiratory disease during smoke days
35 or periods of smoke [Augusto et al. 2020; de Souza Fernandes Duarte et al. 2023; Jie 2017;
36 Martenies et al. 2023; Schwarz et al. 2023; Tarín-Carrasco et al. 2021]. Jie [2017] evaluated
37 mortality, combining respiratory and cardiovascular mortalities due to low numbers,
38 finding no significant association between mortality and pollutants associated with
39 wildland fire smoke. Other studies that evaluated cardiorespiratory events [Augusto et al.
40 2020; McBrien et al. 2023; Schwarz et al. 2023] reported mixed results on hospitalizations

1 and emergency department visits, with a significant finding reported by McBrien et al.
2 [2023].

3 Most respiratory studies identified in this scoping review measured exposure to wildland
4 fire smoke using PM_{2.5} (n=48), and 10 studies measured exposure using PM₁₀. A few studies
5 evaluated exposure using other smoke components, such as CO [de Souza Fernandes Duarte
6 et al. 2023; Machin et al. 2019; Moitra et al. 2021], NO₂ [de Souza Fernandes Duarte et al.
7 2023; Moitra et al. 2021, Niyatiwatchanchai et al. 2023], or O₃ [de Souza Fernandes Duarte
8 et al. 2023, Moitra et al. 2021; Niyatiwatchanchai et al. 2023; Reid et al. 2019]. Overall, these
9 studies reported increased incidence of emergency department visits for several
10 respiratory conditions. For example, de Souza Fernandes Duarte et al. [2023] reported
11 significant increases of respiratory disease and pneumonia for PM₁₀, PM_{2.5}, CO, and ozone.
12 de Souza Fernandes Duarte et al. [2023] and Reid et al. [2019] reported significant
13 increases of COPD for ozone exposure. Niyatiwatchanchai et al. [2023] evaluated the health
14 effects from wildland fire smoke but did not evaluate the smoke components separately;
15 Moitra et al. [2021] noted no significant findings.

16 Seven studies evaluated respiratory health effects in firefighters [Barbosa et al. 2022;
17 Cherry et al. 2021; Ferguson et al. 2017; Gianniou et al. 2018; Nelson et al. 2020;
18 Niyatiwatchanchai et al. 2023; Ramos and Minghelli 2022]. All these studies evaluated lung
19 function. The different lung function parameters in these studies included forced expiratory
20 volume in one second (FEV₁), forced vital capacity (FVC), FEV₁/FVC, and peak expiratory
21 flow (PEF). Significance varied between lung function parameters within the same study.

22 For example, Gianniou et al. [2018] reported statistically significant decreases in lung
23 function among wildland firefighters when evaluating forced expiratory flow at 25%–75%
24 predicted ($P=0.026$), FEV₁/FVC ($P=0.024$), total lung capacity % predicted ($P=0.029$), and
25 the carbon monoxide transfer coefficient (KCO)% predicted ($P=0.039$), while other
26 parameters such as FEV₁ and FVC % predicted were not significant. Nelson et al. [2020]
27 noted significantly lower PEF post-exposure ($P<0.01$) in wildland firefighters; other lung
28 function metrics were also decreased but not statistically significant.

29 Niyatiwatchanchai et al. [2023] found significantly higher post-bronchodilator area of
30 reactance values from impulse oscillometry testing (a measure of small airways function
31 and distal airways heterogeneity) and significant improvements in pre-bronchodilator FEV₁
32 and FVC and post-bronchodilator FVC in wildland firefighters following exposure. Cherry et
33 al. [2021] noted that firefighters had an increased risk of asthma consultation post-fire.

34 Several studies employing spirometry showed decreased lung function parameters with
35 increasing exposure, of which FEV and FVC were significant [Barbosa et al. 2022; Ferguson
36 et al. 2017]. Ramos and Minghelli [2022] did not report significant decrease in lung function
37 in firefighters following exposure to wildland fire smoke.

38 Only one other study evaluated an occupation other than firefighters [Moitra et al. 2021].
39 That cross-sectional study evaluated lung function parameters in a group of Canadian Royal
40 Mounted Police officers (n=218), including total lung capacity, FEV₁, FVC, and FEV₁/FVC.
41 Among predominantly male participants (71%), Moitra et al. [2021] reported a decline in

1 forced expiratory volume in one second (FEV₁) at 76.5 (SD=5.9) percent predicted and
2 residual volume 80.1 (SD=19.5) percent predicted, though neither decline was statistically
3 significant at an $\alpha=0.05$ threshold. The authors concluded that short-term exposure to
4 wildfire-associated air pollutants may impose subtle but “clinically important deleterious
5 respiratory effects, particularly in the peripheral airways.”

6 In summary, respiratory disease was the most extensively studied health effect attributable
7 to wildland fire smoke. Among respiratory conditions examined, asthma and COPD
8 consistently showed significant effects following exposure to wildland fire smoke. Most of
9 these studies used PM_{2.5} to measure exposure. Studies on other respiratory health
10 conditions were fewer in number and showed inconsistent findings.

11 Little information was reported on other contaminants, such as CO, NO₂, SO₂, and O₃.
12 Nevertheless, the available studies indicated an increased incidence of emergency
13 department visits, particularly associated with O₃ exposure.

14 Few studies evaluated the health effects of wildland fire smoke on worker populations.
15 Change in lung function was the most common health effect evaluated in these workers, and
16 firefighters represented most workers evaluated. Overall, declines in lung function among
17 workers post-season or post-fire varied in significance. Potential respiratory effects in other
18 worker populations are unknown, and additional research would provide insight in
19 determining any differences between occupations as well as the general population.

20 **3.2.2.2 Cardiovascular Health**

21 Cardiovascular disease (CVD) is a broad term encompassing a diverse group of health
22 conditions affecting the heart and blood vessels. Historically, the definition of CVD has
23 varied markedly, including a wide range of outcomes classified under the International
24 Classification of Diseases (ICD-10) codes:

- 25 • Rheumatic fever/rheumatic heart disease (I00–I09)
- 26 • Hypertensive diseases (I10–I15)
- 27 • Ischemic (coronary) heart disease (I20–I25)
- 28 • Pulmonary heart disease and diseases of pulmonary circulation (I26–I28)
- 29 • Other forms of heart disease (I30–I52)
- 30 • Cerebrovascular diseases (CeVD, I60–I69) such as stroke and transient ischemic
31 attacks (TIAs)
- 32 • Atherosclerosis (I70)
- 33 • Other diseases of arteries, arterioles, and capillaries (I71–I79)
- 34 • Diseases of veins, lymphatics, and lymph nodes not classified elsewhere (I80–I89)
- 35 • Congenital cardiovascular defects (Q20–Q28)
- 36 • Other unspecified disorders of the circulatory system (I95–I99) [Go et al. 2013; Virani
37 et al. 2021]

38 This scoping review included 50 studies reporting information on the relationship between
39 wildland fire emissions and CVD risk. Most studies (88%) evaluated associations between
40 short-term exposures and immediate CVD outcomes. Outcome definitions varied across

1 studies and included aggregate measures (92%), such as all CVD (n=42), and all
2 cardiorespiratory conditions (n=2), and specific outcomes (37%), such as ischemic heart
3 disease (IHD) (29%), myocardial infarction (MI) (20%), and cerebrovascular disease
4 (CeVD) (25%), as well as symptoms and risk factors, such as angina, hypertension, and
5 dyslipidemia.

6 Case ascertainment used emergency department visits or hospital admissions most often
7 (57% of studies); however, endpoints included other measures of morbidity (n=13 studies)
8 and mortality (n=17 studies). All but four studies examined general population groups; the
9 remainder evaluated CVD risk in groups of wildland firefighters. No other working
10 populations were evaluated. Most study designs (90%) were cross-sectional or quasi-
11 experimental; however, three retrospective cohort studies examined long-term effects.

12 The most frequent exposure measure was PM_{2.5}, which was reported in 69% of studies.
13 Other emission quantities reported included PM₁₀, CO, O₃, and NO₂. Exposure proxies were
14 used in 15 studies.

15 Findings among studies were largely mixed, with more than half (57%) reporting at least
16 one statistically significant positive association between a measure of wildland fire
17 emissions and a CVD outcome. Slightly less than half (44%) of the risk measures for
18 aggregate CVD outcomes (e.g., cardiorespiratory conditions, or all-CVD) included at least
19 one significant positive association.

20 Among specific CVD outcomes studied in five or more studies, the proportion of positive
21 associations was greatest for hypertension (80%), followed by IHD, including MI (53%).
22 The proportion was smallest for CeVD outcomes (17%).

23 Health conditions with less than five studies reporting included peripheral vascular disease
24 (n=2), sudden cardiac arrest (n=1), and TIA (n=3). Among these smaller groups of studies,
25 no evidence was presented of a positive association between wildland fire smoke and
26 increased frequency of TIA or peripheral vascular disease; however, in a large case-
27 crossover study (n=5,336), Jones et al. [2020] reported significant excess risk of out-of-
28 hospital cardiac arrest from smoke exposures during the 2015–2017 California wildfires.

29 Three longitudinal studies examined long-term CVD effects [Gao et al. 2023a; Glass et al.
30 2019; Zeigler et al. 2022]. The findings among these studies were mixed, with little evidence
31 of effect.

32 Glass et al. [2019] conducted a cohort study examining mortality in female Australian
33 firefighters (n=16,903) followed during 1980–2011. They found no evidence of an
34 association between the number of landscape fire incidents attended and circulatory
35 disease mortality. Similarly, no evidence was found of increasing CVD mortality with
36 wildfire-related PM_{2.5} exposures in a cohort study of nearly 500,000 participants enrolled in
37 the UK Biobank Study during 2004–2010 [Gao et al. 2023a].

38 Zeigler et al. [2022] examined associations between wildland firefighting and indicators of
39 cardiovascular disease in two groups of firefighters: one for effects following multiple active
40 firefighting seasons (n=28) and a second for examining pre- and post-firefighting season

1 differences within a single season (n=18). That study reported evidence suggesting
2 persistent wildland firefighting may adversely impact arterial stiffness. The period of
3 observation was not reported. The small sample size was an important study limitation.

4 Of 12 studies reporting on CeVD risk, two reported at least one significantly positive
5 association. One study reported modest increases in the relative risk of all-cause CeVD
6 emergency department visits associated with wildfire smoke days, particularly among those
7 aged 65 years or older, in a population-based study of the effects from the 2015 California
8 wildfires [Wettstein et al. 2018]. An important study limitation was the lack of controlling
9 for potential modifying factors, such as medical history, race, and sex. Another study
10 provided some evidence of an association between periods (days) of high bushfire smoke
11 and hospital admissions for ischemic stroke during large-scale fires in Australia from
12 October 2019 through February 2020 [Hasnain et al. 2024]. However, that study also
13 reported an absence of significant changes in total daily admissions for CeVD, acute stroke,
14 and acute ischemic stroke when risk was examined over the entire bushfire period.

15 In summary, CVD was second to respiratory disease in the number of studies reporting on
16 adverse physical health conditions attributable to wildland fire emissions. Within this
17 group, most studies examined immediate CVD effects from PM resulting from acute
18 exposures during large-scale fires. These studies largely supported previous conclusions on
19 CVD and ambient air pollution that point to a likely causal relationship between particulate
20 exposure and acute CVD effects, consisting primarily of increased risk of IHD, heart failure,
21 and arrhythmia among exposed persons.

22 However, less is known about long-term risks from air pollution, and few longitudinal
23 studies—and no prospective studies—examined long-term CVD or CeVD risk. Therefore,
24 additional research is needed to clarify associations between wildfire emissions and latent
25 or persistent CVD effects, including interrelationships with other chronic illnesses (e.g.,
26 COPD and asthma). Moreover, few studies examined risks in working populations, and
27 those that did were restricted to wildland firefighters. Therefore, CVD risks in other
28 workers who are exposed to wildland fire smoke during work remains largely unknown.

29 Most research has focused on outcomes related to heart disease (e.g., IHD, MI, angina, and
30 arrhythmia), with relatively few studies examining other conditions, such as peripheral
31 artery disease (PAD) or CeVD risks. Furthermore, evidence on exposure-related CeVD
32 effects is inconsistent. Additional etiologic research focusing on the relationship between
33 wildland fire smoke and understudied CVD outcomes, such as PAD, ischemic and
34 hemorrhagic stroke, and TIAs, is needed.

35 **3.2.2.3 Reproductive and Developmental Health**

36 Twenty-four studies evaluated reproductive or developmental health outcomes associated
37 with prenatal or preconception exposure to wildland fire smoke (Table 3–3). These studies
38 focused on various outcomes, including the following:

- 39 • Decreasing/low birthweight or small for gestational age (14 studies) [Abdo et al.
40 2019; Brew et al. 2022; Fernández et al. 2023; Foo et al. 2024; Jones and Berrens
41 2021; Jones and McDermott 2022; Jung et al. 2023b; Li et al. 2021; McCoy and Zhao

- 1 2021; Requia et al. 2022a; Rosales-Rueda and Triyana 2019; Zhang et al. 2023c;
2 Zhang et al. 2023d; Zheng 2023]
- 3 • Preterm birth or gestational age (9 studies) [Abdo et al. 2019; Brew et al. 2022;
4 Heft-Neal et al. 2022; Jones and Berrens 2021; Jones and McDermott 2022; Jung et
5 al. 2023a; Requia et al. 2022c; Zhang et al. 2023d; Zheng 2023]
 - 6 • Pregnancy loss (i.e., stillbirth and miscarriage) (3 studies) [Brew et al. 2022; Jung et
7 al. 2021; Xue et al. 2023]
 - 8 • Birth defects and congenital abnormalities (3 studies) [Park et al. 2022; Requia et al.
9 2022b; Zheng 2023]
 - 10 • Other birth or developmental outcomes (7 studies), such as height [Rosales-Rueda
11 and Triyana 2019; Singh and Dey 2021], increased birthweight/large for gestational
12 age [Brew et al. 2022; Fernández et al. 2023; Zheng 2023]; admission for neonatal
13 intensive care following birth [Abdo et al. 2019], respiratory conditions [Abdo et al.
14 2019; Dhingra et al. 2023; Rosales-Rueda and Triyana 2019], and cognitive function
15 [Rosales-Rueda and Triyana 2019]
 - 16 • Other pregnancy-related conditions (4 studies), including gestational diabetes,
17 gestational hypertension, or preeclampsia [Abdo et al. 2019; Brew et al. 2022], in
18 vitro fertilization outcomes [Kornfield et al. 2024], and other conditions of
19 pregnancy or delivery [Brew et al. 2022; Rosales-Rueda and Triyana 2019]

20 All studies evaluated reproductive or developmental health effects associated with prenatal
21 environmental exposure to wildland fire smoke among a general, nonoccupational,
22 population except for three, which assessed preconception effects of environmental
23 exposure on embryos from women undergoing in vitro fertilization [Kornfield et al. 2024]
24 and adverse birth outcomes associated with maternal occupation as a firefighter (including
25 wildland firefighters) [Jung et al. 2021; Jung et al. 2023a].

26 Although the only two occupational studies found no increased risk among offspring of
27 wildland firefighters compared with a general population, the authors discuss potential
28 reproductive hazards of firefighting beyond just wildland fire smoke, including other
29 fireground exposures, physical exertion and physiological strain, and psychosocial stress.

30 Several studies used additional proxies of exposure, such as location or time, which might
31 have limited specificity for classifying wildland fire smoke exposure [Brew et al. 2022; Jones
32 and Berrens 2021; Jones and McDermott 2022; Jung et al. 2023b; McCoy and Zhao 2021;
33 Park et al. 2022; Requia et al. 2022a; Requia et al. 2022b; Singh and Dey 2021; Zheng 2023].

34 A study by Zheng [2023] conducted various sensitivity analyses that classified exposure in
35 multiple ways using proxies of smoke (i.e., employing differing thresholds for distance to
36 and size of fires) in combination with multiple methods of outcome ascertainment. Analyses
37 yielded varying estimates and only the results from the study's primary models were
38 aggregated for the current review. Nonetheless, the analyses demonstrate the dependence
39 of findings on method of exposure ascertainment/classification, particularly when proxies
40 for exposure that were not derived from smoke are used.

1 On the other hand, aggregate quantitative measures of smoke-related contaminants—
2 namely PM_{2.5}—were incorporated into exposure ascertainment, at least to some extent, by
3 10 studies, likely improving the specificity of exposure classification [Abdo et al. 2019;
4 Dhingra et al. 2023; Fernández et al. 2023; Foo et al. 2024; Heft-Neal et al. 2022; Li et al.
5 2021; Requia et al. 2022c; Xue et al. 2023; Zhang et al. 2023c; Zhang et al. 2023d]. A
6 composite rating of ambient air pollution, such as an Air Quality Index (AQI), was used to
7 define exposure by two studies [Kornfield et al. 2024; Rosales-Rueda and Triyana 2019].

8 Effect modification by period of pregnancy (e.g., trimester) was studied in a majority of
9 studies on prenatal exposure, demonstrating a priority in research for identifying periods of
10 vulnerability to wildland fire smoke exposure for various health conditions. Additional
11 effect modification was explored by several studies. For example, while the studies of
12 firefighters found no main effect of wildland firefighting, wildland firefighting was found to
13 be an effect modifier for the association between volunteer firefighting and birth outcomes
14 [Jung et al. 2021; Jung et al. 2023a]. Another study found the effects of environmental
15 exposure to a wildfire in Australia on multiple birth and pregnancy outcomes varied based
16 on overlap in timing with the COVID-19 pandemic [Brew et al. 2022]. Studies of
17 environmental exposure in Brazil stratified by geographic region to account for
18 heterogenous landscapes [Requia et al. 2022a,b, 2022c].

19 The presence of associations varied across health conditions and studies. However, for the
20 most-studied topics, a majority of the analyses presented statistically significant
21 associations between exposure and adverse outcomes: 71% for birthweight and 78% for
22 gestational age. Designs varied for these studies, with cross-sectional and quasi-
23 experimental designs characterizing the largest portions. Two meta-analyses calculated
24 summary estimates on the association between wildland fire smoke exposure and
25 birthweight, yielding mixed results [Foo et al. 2024; Zhang et al. 2023c].

26 Findings from studies on other conditions (e.g., pregnancy loss, birth defects, and other
27 outcomes) were mixed, and many associative findings were small. These topics were less
28 studied in the recent literature on wildland fire smoke exposure, likely due in part to the
29 relative infrequency of some outcomes in the general population (e.g., certain birth defects,
30 stillbirth) [CDC 2008; Gregory et al. 2022] and subsequent challenges for researching them.
31 Although most studies focused on the effects on offspring of prenatal exposure, more
32 evidence on fertility-related outcomes and preconception exposures, among both women
33 and men, is warranted. Similarly, the potential for take-home exposure and associated
34 health effects among exposed workers' family members (e.g., pregnant partners) can be
35 investigated.

36 About half of the reproductive health studies incorporated measured constituents of smoke
37 in their exposure classification. Additional research employing such methods will likely
38 provide more robust exposure assessment for use in exposure-response and dose-response
39 analyses. Along with PM_{2.5}, the effects of other wildland fire smoke-related exposures can be
40 explored in relation to reproductive and developmental health.

1 No reproductive health studies on occupational populations exposed to wildland fire smoke
2 were available in the synthesized literature other than several on firefighters. Additional
3 research on various occupational populations might clarify the burden of reproductive and
4 developmental health outcomes among exposed workers. This could help differentiate the
5 exposure-response relationships compared with the general population and between
6 different occupational groups.

7 **3.2.2.4 Cancer**

8 This scoping review found sparse information on the association between exposure to
9 wildland fire smoke and cancer. Four epidemiological studies met the eligibility criteria
10 [Gao et al. 2023a; Glass et al. 2019; Korsiak et al. 2022; Yu et al. 2022]. Three examined
11 wildfire-related PM_{2.5} exposure in general populations in the United Kingdom (UK), Brazil,
12 and Canada. One investigated cancer mortality among Australian firefighters attending
13 landscape fires. Three of the studies were retrospective cohort designs [Gao et al. 2023a;
14 Glass et al. 2019; Korsiak et al. 2022], while the remaining study was quasi-experimental
15 [Yu et al. 2022].

16 Gao et al. [2023a] conducted a study using the national UK Biobank cohort, which included
17 over 5 million person-years of data with an average follow-up of 11.2 years. They found a
18 5% increase in the risk of mortality from all neoplasms (ICD 10 C00–C97, D00–D48) per 10
19 micrograms per cubic meter ($\mu\text{g}/\text{m}^3$) increase in wildfire-related PM_{2.5} concentration. Long-
20 term exposure was defined as a 3-year cumulative concentration of wildfire-related PM_{2.5}
21 within a 10-km radius of each exposed person’s residential address.

22 In another large cohort study with 34 million person-years of observation, Korsiak et al.
23 [2022] found that wildfire exposure, as defined by the burned area near residential
24 locations, was associated with increased incidence of cancers of the lung and brain, but not
25 hematopoietic cancers.

26 Among female volunteer firefighters, Glass et al. [2019] found a modest, but nonsignificant
27 positive trend ($P=0.08$) in cancer mortality increasing with the number of attendances at
28 landscape fires. That study reported a significant positive association among those in the
29 highest tertile of attendance compared with those with no incidents reported.

30 Yu et al. [2022] examined mortality from several cancers among the general population of
31 Brazil, reporting modest increases in the relative risk per $\mu\text{g}/\text{m}^3$ wildfire-related PM_{2.5}
32 concentration for cancers of the nasopharynx, esophagus, stomach, colon and rectum,
33 larynx, skin, female breast, prostate, and testis. That study also reported an attributable risk
34 for all cancers combined of 37 per 10⁵ persons for 2-year average wildfire-related PM_{2.5}
35 exposure ($2.38 \mu\text{g}/\text{m}^3$).

36 Information elucidating the shape of the exposure-response, especially at low exposures,
37 was not available in any study.

38 Overall, these studies contributed evidence suggesting that wildland fire smoke is
39 associated with increased cancer risk. However, as is common in most observational
40 studies, study designs were not sufficient to rule out many potential sources of bias (e.g.,

1 confounding, measurement error), and risk estimates were exceptionally vulnerable to bias
2 given small effect sizes observed in all studies.

3 Another important limitation is an overall lack of studies examining late and chronic health
4 effects, such as cancer, from acute or protracted exposures to wildland fire smoke. Only
5 three longitudinal studies were available, with only one study of a working population
6 (firefighters).

7 Workers in farming, forestry, and construction who predominantly work outdoors are likely
8 to experience vastly different exposures compared with nearby residents. Assessment of
9 temporal modification was lacking, which is an important area of etiologic research of latent
10 diseases, such as cancer. In general, further research is needed to examine the relationship
11 between wildland fire smoke and cancer, with increased attention given to methods for
12 improved internal validity, temporality, and increased generalizability to working
13 populations who are believed most at risk.

14 **3.2.2.5 Metabolic Conditions**

15 For this review, metabolic effects include characteristics of diabetes mellitus, such as
16 hyperglycemia and insulin resistance, and other risk factors associated with metabolic
17 syndrome, such as dyslipidemia, or obesity, excluding hypertension. Hypertension was
18 considered in the review of cardiovascular disease.

19 The scoping review found six studies reporting information on metabolic effects from
20 wildland fire smoke [Chen et al. 2023; Kondo et al. 2022, Mahsin et al. 2022; Malig et al.
21 2021, Rosales et al. 2022; Yao et al. 2020]. All studies used quasi-experimental designs, with
22 five studies examining diabetes mellitus in Canadian and U.S. populations and one study
23 examining dyslipidemia in U.S. wildland firefighters [Rosales et al. 2022]. All but one study
24 examined the exposure-response for metabolic effects, while one study examined exposure-
25 related cardiorespiratory effects among persons previously diagnosed with diabetes
26 mellitus [Mahsin et al. 2022]. Four studies estimated PM_{2.5} exposure concentrations [Chen
27 et al. 2023; Mahsin et al. 2022; Malig et al. 2021, Yao et al. 2020]; while two studies used
28 exposure proxies [Kondo et al. 2022; Rosales et al. 2022].

29 Of four studies examining exposure-response associations between short-term PM_{2.5}
30 exposure and diabetes mellitus, three found modest, but statistically significant, positive
31 associations [Kondo et al. 2022; Malig et al. 2021; Yao et al. 2020]. Chen et al. [2023]
32 reported a positive association between smoke events and diabetic emergency department
33 visits; however, the relative risk estimate was not statistically significant. In a study of pre-
34 to post-season biologic changes among wildland firefighters, Rosales et al. [2022] reported
35 maladaptive serum lipids and body mass alterations. Mahsin et al. [2022] found evidence
36 suggesting persons with diabetes mellitus might be at greater risk of PM_{2.5}-related adverse
37 cardiorespiratory health effects.

38 Overall, four of the six studies contributed evidence supporting a weak association between
39 short-term exposure to wildland fire smoke and adverse metabolic effects, primarily
40 diabetes mellitus. Little discussion focused on potential mechanisms, and the authors
41 acknowledged that differences in demographic variables, as well as comorbid conditions

1 might have influenced estimates. Other limitations common to observational studies were
2 noted by researchers, which should be explored in future research. Effect sizes were small;
3 therefore, estimates were particularly susceptible to bias. Except for a single study of
4 wildland firefighters, no new information was presented on wildland fire smoke-related
5 metabolic effects in working populations. Increased attention to the risk of adverse
6 metabolic effects in working populations is needed.

7 **3.2.2.6 Neurological Conditions**

8 This scoping review found six studies that investigated the effects of wildfire smoke
9 exposure on neurological conditions. These conditions included cognitive function,
10 headache, cerebral hemodynamics, and neurological-related mortality [Cleland et al. 2022;
11 Elser et al. 2023; Gao et al. 2023a; Hasnain et al. 2024; Kondo et al. 2022; Lai et al. 2022;
12 Tan et al. 2019; Zhang et al. 2023a]. All studies examined effects on the general population.
13 Most studies (five of the six) used quantitative estimates of PM_{2.5} as an exposure metric,
14 with one of the studies also using the quantitative estimate of PM_{2.5} to further categorize
15 days of exposure based on smoke density (e.g., “high” versus “low” smoke days) [Cleland et
16 al. 2022]. The remaining study used measurements of a pollutant standard index (PSI) that
17 included wildfire smoke as a proxy for smoke [Tan et al. 2019].

18 All three studies examining the effects of exposure to PM_{2.5} on cognitive function found
19 significant risk associations with both short-term (e.g., cognitive performance and memory
20 challenges) and long-term (e.g., dementia) cognitive abilities [Cleland et al. 2022; Lai et al.
21 2022; Zhang et al. 2023a]. Elser et al. [2023] also found an association between general
22 short-term PM_{2.5} exposure and emergency department visits related to tension headaches.
23 However, when considering wildfire-specific PM_{2.5} exposure, no significant associations
24 were found across headache subcategories [Elser et al. 2023]. Tan et al. [2019] found an
25 association between increased haze exposure (measured in PSI, which includes smoke from
26 wildfires) and changes in the cerebral hemodynamics. Lastly, no increased risk association
27 was found between exposure to wildfire smoke and neurologic-related mortality [Gao et al.
28 2023a].

29 The largest knowledge gap pertains to the neurological effects of wildfire smoke in working
30 populations. Additionally, many of the studies were subject to small effect sizes and found
31 “trends” of associations that were not statistically significant, indicating that further
32 research is needed. Furthermore, of the statistically significant associations related to
33 neurologic effects, many were found from all-source PM_{2.5} or other pollutant exposure
34 rather than wildfire-specific smoke exposure.

35 **3.2.2.7 Infectious Disease**

36 An infectious disease is an illness that is caused by a pathogen or its toxic product, which
37 arises through transmission from an infected person, an infected animal, or a contaminated
38 inanimate object to a susceptible host [van Seventer and Hochberg 2017, pp. 22–39].
39 Environmental factors, such as exposures to wildland fire emissions, may act to increase the
40 risk of exposure to infectious agents, increase the vulnerability of the host, or both.

1 Fourteen studies examined associations between exposures to wildland fire smoke and
2 infectious disease. U.S. populations exposed to large-scale wildfires were examined most
3 (n=11), followed by South American (n=2) and Australian (n=1) populations. No worker
4 studies were available. Except for one longitudinal cohort study of systemic fungal diseases,
5 all studies used quasi-experimental designs to assess exposure-response.

6 Eleven studies reported on PM_{2.5} or PM₁₀ exposures, two studies each reported on CO, O₃,
7 and NO₂ concentrations, and five studies examined other exposure measures and proxies.
8 Most studies (71%) examined the relationship between wildland fire emissions and SARS-
9 CoV-2 test positivity or the occurrence of respiratory coronavirus disease 2019 (COVID-19);
10 however, four studies investigated other outcomes, such as influenza, tuberculosis, fungal
11 infections, and infection-related mortality. The latter examined mortality patterns in a
12 group of U.S. hemodialysis patients and found no evidence of an association between
13 wildland fire emissions and infectious disease mortality [Xi et al. 2020]. The remaining
14 studies reported at least one statistically significant positive association between wildland
15 fire exposure and infectious disease risk. Examples of positive studies are briefly described
16 below.

17 Positive associations between wildfire emissions and COVID-19 were observed in multiple
18 populations. For example, Meo et al. [2020] found that COVID-19 cases and deaths in 10
19 different counties in California were associated with wildfire PM_{2.5}, CO, and O₃. Similar
20 results were observed in a study in other west coast U.S. residents [Sannigrahi et al. 2022].

21 In a study of the COVID-19 outbreak in Australia, Cortes-Ramirez et al. [2022] found a
22 statistically significant positive association between higher percentages of wildfire burned
23 area and increased COVID-19 incidence. No evidence was found of an association between
24 COVID-19 and average PM₁₀ levels.

25 Some evidence suggested increasing pathogenicity from wildland fire exposure. For
26 example, in a study of the SARS-CoV-2 test positivity rate at a large regional hospital in
27 Nevada, Kiser et al. [2021] found a 6.3% relative increase in the positivity rate per 10 µg/m³
28 increase in PM_{2.5} concentration during wildland fires.

29 Although positive associations were observed in several studies, inconsistencies were
30 observed between and within studies. For example, Zhou et al. [2021] reported positive
31 associations between PM_{2.5} levels and COVID-19 cases and deaths when pooling data from
32 92 western U.S. counties under investigation. Results varied widely between counties,
33 including some counties that reported protective effects from exposure. The heterogeneity
34 among counties points to differences in within-county covariates that could not be
35 accounted for by study design. These differences may lead to bias in risk estimates.

36 Evidence was found that supports exposure-related risk of other infectious diseases. For
37 example, Landguth et al. [2020] found that an increase of 1 µg/m³ in average daily summer
38 PM_{2.5} concentration during the wildfire season was associated with a 16%–22% increase in
39 influenza cases in the following winter influenza season. In a population-based,
40 retrospective study of California hospital admissions, Mulliken et al. [2023] reported

1 evidence of an increase in diagnosis of coccidioidomycosis (valley fever), but not
2 aspergillosis, in the months following large scale wildland fires.

3 Overall, these studies contributed evidence suggesting increased onset and exacerbation of
4 multiple infectious diseases from wildfire emissions. However, the underlying mechanisms
5 remain unclear. Several studies, such as Cortes-Ramirez et al. [2022], Kiser et al. [2021], and
6 Meo et al. [2021], have posited that exposure to PM_{2.5} and other agents released in wildfires
7 may modify immune responses and facilitate transport into the lungs. More research is
8 needed to clarify possible mechanisms.

9 Regarding infectious disease risk, an important limitation was the absence of worker
10 studies; therefore, the representativeness of the findings for occupational hazards remains
11 uncertain. For the purposes of causal inference, this review focused on the direct effects of
12 wildfire emissions on infectious disease. Indirect effects caused by impacts to healthcare
13 and social services resulting from large scale wildfires, especially during the COVID-19
14 pandemic, were not considered.

15 **3.2.2.8 Subclinical Changes**

16 This scoping review found 11 studies that included investigation of subclinical markers of
17 early effects from exposure to wildland fire smoke. Some of the subclinical effects studied
18 included oxidative stress, inflammatory biomarkers, deoxyribonucleic acid (DNA) markers,
19 and urinary mutagenicity [Abreu et al. 2017; Adetona et al. 2017; Adetona et al. 2019;
20 Aguilera et al. 2023; Kim et al. 2017; Main et al. 2020; Niyatiwatchanchai et al. 2023;
21 O'Dwyer et al. 2021; Wu et al. 2020; Wu et al. 2021b; Xu et al. 2023].

22 Seven studies examined the effects specifically in firefighters responding to wildfires [Abreu
23 et al. 2017; Adetona et al. 2017; Adetona et al. 2019; Main et al. 2020; Niyatiwatchanchai et
24 al. 2023; Wu et al. 2020; Wu et al. 2021b]. The remaining four studies were conducted on
25 the general population [Aguilera et al. 2020; Kim et al. 2017; O'Dwyer et al. 2021; Xu et al.
26 2023].

27 All studies on wildland firefighters used their occupation as a proxy for smoke exposure. To
28 examine any potential effects that might occur from differences in exposure at a single fire
29 site, three studies further classified individuals based on their job task at the fire, such as
30 holding the fire line versus lighting fires [Adetona et al. 2017; Adetona et al. 2019; Wu et al.
31 2021b].

32 Of the four studies that examined effects in the general population, two studies used an
33 individual's location as a proxy for smoke exposure [Aguilera et al. 2023; Kim et al. 2017]
34 and the remaining two studies obtained quantitative estimates of PM_{2.5} to assess smoke
35 exposure.

36 Four studies on firefighters examined the potential association between smoke exposure
37 and markers of oxidative stress [Abreu et al. 2017; Adetona et al. 2019; Wu et al. 2020; Wu
38 et al. 2021b]. However, only Wu et al. [2021b] found a significant increase in oxidized
39 guanine species (Ox-GS) from pre-shift to the next morning following wildland fire smoke
40 exposure on burn days.

1 Additionally, Wu et al. [2021b] found a significant difference in cross-shift changes (pre-
2 post shift) in Ox-GS and 8-isoprostane between burn and non-burn days. Five studies
3 included assays on inflammatory responses to smoke exposure [Adetona et al. 2017; Main
4 et al. 2020; Niyatiwatchanchai et al. 2023; O'Dwyer et al. 2021; Wu et al. 2020]. Four of
5 those five studies were conducted on firefighters, two of which found associations between
6 smoke exposure and both pro- and anti-inflammatory cytokine expression [Adetona et al.
7 2017; Main et al. 2020].

8 Xu et al. [2023] studied the effect of smoke exposure on the DNA methylation patterns of
9 the general population and found an association between increased PM_{2.5} and differential
10 DNA methylation patterns related to inflammatory regulation and platelet activation.
11 However, when looking at overall DNA damage, Abreu et al. [2017] found no significant
12 difference in basal DNA damage in firefighters compared with controls.

13 Two studies examined the association between smoke exposure and urinary mutagenicity
14 in firefighters [Adetona et al. 2019; Wu et al. 2021b]. Neither study found an association
15 with cross-shift changes in urinary mutagenicity. However, Wu et al. [2021b] observed a
16 difference in urinary mutagenicity when comparing pre-shift to next-morning levels among
17 firefighters in different job positions at a prescribed fire. Firefighters in the "lighting"
18 position, who use drip torches to ignite fires in predesignated areas, generally experience
19 greater smoke exposure and exhibited increased urinary mutagenicity compared with those
20 in the "holding" position, who patrol and maintain fires within planned burn areas.

21 Aguilera et al. [2023] conducted a proteomic assessment on the general population after
22 wildland fire smoke exposure and found significant changes in expression for several
23 immune-related proteins.

24 Overall, most of the studies (7 of 11) showed an association between smoke exposure and a
25 subclinical outcome, with oxidative stress and inflammation as the most frequently studied.
26 However, these studies exhibit several limitations, including variability in the assays used to
27 evaluate health endpoints and the origins of the samples collected, such as blood versus
28 urine. Additionally, in studies involving firefighters, where their occupation served as a
29 proxy for smoke exposure, researchers acknowledged that multiple stressors beyond
30 smoke exposure could influence these endpoints.

31 **3.2.2.9 Total Mortality and Health Effects Not Categorized**

32 Eighteen studies evaluated the association between wildland fire smoke and total (i.e., all-
33 cause) mortality. Outcomes definitions varied, with 72% evaluating all or unspecified
34 causes and the remainder examining total mortality, omitting accidents, injuries, and other
35 external causes (i.e., ICD 10 code A00).

36 Given the etiologic heterogeneity, detailed information on a specific adverse condition is
37 generally preferred for exposure-response analyses rather than aggregate measures.
38 However, total mortality often primarily reflects cardiorespiratory deaths, which are the
39 most common in human experience and are believed to be most closely associated with
40 exposure to wildland fire smoke. For this reason, these studies might prove informative in
41 future risk assessment.

1 Many of the all-cause mortality studies have been described in other sections of the report
2 because they also evaluated cause-specific mortality. Because of this, they are not discussed
3 further in this section. Three studies did not investigate any specific death cause outlined in
4 the scoping review, so they are summarized in Table 3–3 within their own category. These
5 studies examined mortality in exposed populations from Spain [Linares et al. 2018],
6 Australia [Jegasothy et al. 2023], and the United States [Zhang et al. 2023b]. No studies of
7 working populations were among this group.

8 Two quasi-experimental studies directly assessed mortality, defined as “all-cause” in one
9 study [Jegasothy et al. 2023] and nonaccidental mortality in the other [Linares et al. 2018].
10 The remaining study was a longitudinal study examining survival in the interval between
11 age at hospital discharge and age at death from any cause, last contact, or study end, among
12 recovering lung cancer surgery patients experiencing wildland fire exposures [Zhang et al.
13 2023b].

14 One study had quantitative PM_{2.5} estimates [Jegasothy et al. 2023], and one study used PM₁₀
15 estimates [Linares et al. 2018]. One study used a proxy for exposure derived from wildfire
16 occurrences (ever/never) within time windows post-discharge and zip codes of the
17 patient’s residence [Zhang et al. 2023b].

18 Each study contributed some evidence of modestly increasing mortality (or decreasing
19 survival) with increasing short-term exposure to wildland fire smoke. An important
20 limitation in the group of studies was the lack of information on working populations. For
21 example, lung cancer surgery patients are not representative of healthy workers, and the
22 generalizability of the remaining two studies is uncertain.

23 Five articles reported on physical health conditions outside of defined categories [Beyene et
24 al. 2022; Fadadu et al. 2022; Fadadu et al. 2021; Sheldon and Sankaran 2017; Syam et al.
25 2017]. Of these, three studies assessed several self-reported signs and symptoms associated
26 with upper respiratory symptoms, eye irritation, and chest tightness [Beyene et al. 2022;
27 Sheldon and Sankaran 2017; Syam et al. 2017].

28 In general, these studies demonstrated associations between signs and symptoms and acute
29 wildfire smoke exposure that were aligned with findings from more robust cause-specific
30 research. The remaining articles reported evidence of a modest positive association
31 between short-term exposure to wildfire-associated air pollution and increased clinic visits
32 by California patients for treatment of atopic dermatitis and skin itch [Fadadu et al. 2021,
33 2022]. Among adults, this association appeared restricted to persons aged 65 years or older,
34 which may poorly reflect risks in working populations [Fadadu et al. 2022].

35 **3.2.2.10 Health Impact Assessments**

36 This scoping review included 37 health impact assessments (HIAs) that reported
37 population-based, cause-specific, estimates of the health burden from wildland and
38 prescribed fire smoke. The health burden was predicted using information on exposures,
39 the affected population, and the exposure-response function, or concentration-response
40 function (CRF), that related the estimated exposure to the risk of an adverse event. The
41 CRFs were abstracted from previous etiologic research, primarily on ambient air pollution.

1 As a result, findings from HIAs, although informative on potential health burden
2 attributable to wildland fire smoke, do not contribute evidence on the exposure-response
3 necessary for hazard identification.

4 Most burden measures were generally derived assuming a loglinear exposure-response,
5 although other model forms were used in some HIAs. The loglinear exposure-response
6 model follows the general form:

$$\Delta y = y_0 \times (1 - e^{-\beta_i \times \Delta X}) \times P$$

7
8 for change in burden Δy , baseline hazard rate y_0 , in exposed population P . The dose-
9 response parameter, β , represents the change in the effect at unit exposure concentration, X ,
10 and ΔX signifies the change in concentration that is attributable to wildland fire in most
11 cases. In general, exposure concentrations were estimated in spatial fields using
12 atmospheric models that incorporated measurements of air quality and meteorological
13 conditions, when available.

14 CRF parameters, such as β , were derived from empirical data in source epidemiological
15 studies under assumptions on the shape of the exposure-response (e.g., linear, loglinear,
16 and power models). Their use in HIAs is based on similarities between the source
17 epidemiological studies and the outcome and exposure scenario of interest. Burden
18 estimates were calculated using CRFs abstracted from a single epidemiological study,
19 multiple studies, or by pooling estimates from several epidemiological studies (e.g., meta-
20 analysis). For the latter, inverse variance weighted random-effects models were used most
21 often.

22 In most HIAs, baseline rates were derived from registries and medical records (e.g., CDC
23 Wonder, hospital visit records), and population densities were taken from census
24 information. Most HIAs retrospectively estimated burdens attributable to the incidence of
25 actual large-scale wildfires, prescribed fires, or both. Some investigations used simulations
26 to project risk under assumed conditions, such as studies examining the effects from
27 proposed forest management interventions, changing settlement patterns in the wildland-
28 urban interface, or variation in future patterns related to climate (e.g., temperature and
29 precipitation). These included Neumann et al. [2021], Ravi et al. [2018], and Schollaert et al.
30 [2023].

31 Given differences in population size, exposure characteristics, and study aims, burden
32 endpoints varied markedly among health impact assessments. Common endpoints were all-
33 cause and cause-specific mortality (n=26) and cardiorespiratory hospital admissions
34 (n=16) or emergency department visits (n=13). Some studies described health burdens by
35 measures of workdays lost, years of life lost, or disability-adjusted life years (n=6).

36 Consistent with evidence on the association between wildland fire smoke and adverse
37 physical health, health conditions frequently assessed were upper and lower respiratory
38 diseases (n=23); CVD (n=14); and cancer (n=6). Other health conditions examined included
39 neurological and developmental outcomes [Cromar et al. 2024]. Most burden estimates
40 were attributable to major wildfires. However, a few studies examined the effects from

1 prescribed fires [Afrin and Garcia-Menendez 2021; Huang et al. 2019; Ravi et al. 2018] or
2 both prescribed and wildfires [Punsompong et al. 2021; Roberts and Wooster 2021]. Some
3 HIAs examined the combined effects of anthropogenic and wildfire sources, such as Bruni
4 Zani et al. [2020], Graham et al. [2021], and Oliveri Conti et al. [2017]. Nearly all studies
5 estimated health burdens in large populations (e.g., state-level, country-level, or larger);
6 however, there were two studies that predicted health risks in smaller groups of wildland
7 firefighters [Navarro et al. 2019; Teixeira et al. 2024].

8 Methods among HIAs varied in complexity, ranging from a simple assessment of a single
9 endpoint to examining multiple endpoints under differing scenarios. In most HIAs, exposure
10 estimates were derived using sophisticated temporal-spatial emission models, resulting in
11 sector-based (e.g., county- or zip code-level) concentration estimates. Most exposure
12 assessments used data from ground-level monitoring stations across the areas of interest,
13 either integral to modeling or as a means of model validation.

14 Among recent studies, several conducted multiple analyses of several health endpoints
15 using freely available software tools, such as the EPA's Environmental Benefits Mapping and
16 Analysis Program (BenMAP-CE) [Sacks et al. 2018] or the World Health Organization's
17 AirQ+ software [Oliveri Conti et al. 2017]. BenMAP-CE was used most often (n=6) and
18 includes data (e.g., CRFs, population files, and health and economic data) and methods (e.g.,
19 modeling, data pooling, treatment of estimate uncertainty) for estimating health and
20 economic impacts to populations exposed to wildland fire smoke.

21 HIAs have several limitations specific to their methodology, in addition to the general
22 limitations inherent to an ecologic study design and exposure-response modeling. One of
23 the primary concerns is the inevitable differences between the scenario under investigation
24 and the conditions under which the models were derived. This discrepancy leads to
25 uncertainty regarding the accuracy of the true exposures and exposure-response portrayed
26 by the fitted models [Cleland et al. 2021].

27 Some HIAs have shown that burden estimates can be very sensitive to exposure and
28 epidemiological inputs [Cleland et al. 2021; Jiang and Enki Yoo 2019; Johnson and Garcia-
29 Menendez 2022]. Uncertainty analyses are sparse and have focused primarily on the
30 accuracy of exposure estimates [Jiang and Enki Yoo 2019], although evidence suggests that
31 errors in the exposure-response function may be more impactful in some scenarios
32 [Johnson and Garcia-Menendez 2022].

33 Another important limitation of HIAs is the reliance on model estimates of PM_{2.5} as the
34 primary exposure, assuming no difference exists in toxicity between PM_{2.5} as a general
35 pollutant and PM_{2.5} specifically from wildland fires. However, hazardous contaminant
36 mixtures can largely differ by source, meteorology, and exposure conditions (e.g., episodic
37 versus continuous exposure). Of note, evidence from some epidemiological studies indicates
38 that PM_{2.5} from wildfires may be more toxic than that from ambient sources [Aguilera et al.
39 2021].

40 These studies have mostly estimated the health burden in a general population, which may
41 poorly reflect the actual burden in a working population. Workers may have greater

1 exposures to wildfire smoke and have different health statuses due to factors such as
2 lifestyle and healthcare access. For example, census and health information used in HIAs
3 may not adequately represent migrant workers, who may be overrepresented in outdoor
4 working populations.

5 In this scoping review, information from HIAs restricted to workers was limited to two
6 studies involving small groups of wildland firefighters [Navarro et al. 2019; Teixeira et al.
7 2024]. More work is needed to derive fire-specific exposure-response functions and
8 population characteristics that are better suited for use in risk assessment involving
9 occupational exposures to hazardous agents from wildland fires.

10 Overall, HIAs have demonstrated a serious health burden in populations exposed to
11 wildland fire smoke. Among agents examined, PM_{2.5} inhalation contributed most to total
12 attributable risk, mainly consisting of exposure-related cardiorespiratory morbidity and
13 mortality. These studies provided a wealth of information on exposures from major fire
14 events and have contributed to exposure and risk assessment methods that may benefit
15 future assessments.

16 However, HIAs have not adequately addressed important sources of uncertainty in their
17 burden estimates. Further consideration of uncertainty is needed to improve assessments
18 of smoke-related health impacts. Moreover, few HIAs have directly examined risk in
19 working populations, and a large uncertainty exists in transporting population risks to
20 groups of workers. Finally, although meant to inform decision-making processes (e.g., risk
21 characterization), HIAs do not add to the body of evidence on causal relationships between
22 wildland fire smoke exposures and adverse health conditions. For these reasons, these
23 studies are of limited value in this hazard review.

1 **Table 3–3 Frequency (n) of studies included in the scoping review by evaluated health conditions, populations, exposure metrics,**
 2 **and associative findings**

Health conditions*	Total	Firefighter populations	Other worker populations	General population	PM _{2.5} exposures*	Other PM exposures*	Other exposures*†	Proxy for exposure*‡	Risk association§
Respiratory									
Total	70	7	1	62	48	10	5	22	60
Composite measures, such as combined respiratory endpoints	45	0	0	45	34	6	3	9	36
Cardio-respiratory	4	0	0	4	2	1	0	3	3
Lung function	12	7	1	4	4	0	2	6	8
Asthma	30	1	0	29	26	3	2	5	26
COPD	17	0	0	17	15	2	2	2	12
Respiratory symptoms, such as cough or wheeze	5	1	0	4	4	0	0	1	4
Respiratory infection	13	0	0	13	12	0	1	2	6
Pneumonia	11	0	0	11	10	2	2	1	2
Other	3	0	0	3	1	1	0	1	2
Cardiovascular									
Total	50	4	0	46	35	7	1	15	28
Composite measures, such as combined CVD endpoints	45	2	0	43	33	6	1	11	20
IHD	14	0	0	14	13	1	0	1	5
MI	10	0	0	10	10	0	0	0	4

Health conditions*	Total	Firefighter populations	Other worker populations	General population	PM _{2.5} exposures*	Other PM exposures*	Other exposures*†	Proxy for exposure**‡	Risk association [§]
Arrhythmia	7	0	0	7	7	0	0	0	1
Hypertension	5	2	0	3	2	0	0	3	4
Peripheral vascular disease	2	0	0	2	2	0	0	0	0
Heart failure	6	0	0	6	6	0	0	0	1
Cardiac arrest	1	0	0	1	1	0	0	1	1
CeVD, such as stroke, TIA	12	0	0	12	11	0	0	2	2
Reproductive and developmental									
Total	24	2	0	22	10	0	1	14	20
Birthweight and small for gestational age	14	0	0	14	6	0	0	8	10
Preterm birth and gestational age	9	1	0	8	4	0	1	5	7
Pregnancy loss	3	1	0	2	1	0	0	2	1
Birth defects and congenital abnormalities	3	0	0	3	0	0	0	3	2
Other birth and developmental outcomes	7	0	0	7	3	0	0	4	4
Other pregnancy-related outcomes	4	0	0	4	1	0	0	3	3
Cancer									
Total	4	1	0	3	3	0	0	1	4
Lung	2	0	0	2	2	0	0	0	1
Brain	2	0	0	2	2	0	0	0	1

1

2

Health conditions*	Total	Firefighter populations	Other worker populations	General population	PM _{2.5} exposures*	Other PM exposures*	Other exposures*†	Proxy for exposure**‡	Risk association [§]
Hematopoietic sites	1	0	0	1	1	0	0	0	0
Other sites	1	0	0	1	1	0	0	0	1
Neurological									
Total	6	0	0	6	5	0	0	1	5
Cognitive	3	0	0	3	3	0	0	0	3
Headache	1	0	0	1	1	0	0	0	1
Other	2	0	0	2	1	0	0	1	1
Metabolic									
Total	6	1	0	5	4	0	0	2	4
Diabetes mellitus	5	0	0	5	4	0	0	1	3
Metabolic syndrome	1	1	0	0	0	0	0	1	1
Infectious disease									
Total	14	0	0	14	10	2	2	5	13
COVID-19	10	0	0	10	7	0	0	4	10
Other	4	0	0	4	3	0	0	1	4
Sub-clinical changes									
Total	11	7	0	4	2	0	0	9	7
Oxidative Stress	4	4	0	0	0	0	0	4	1
Inflammation	5	4	0	1	1	0	0	4	3

Health conditions*	Total	Firefighter populations	Other worker populations	General population	PM _{2.5} exposures*	Other PM exposures*	Other exposures*†	Proxy for exposure*‡	Risk association§
DNA markers	2	1	0	1	1	0	0	1	1
Urinary mutagenicity	2	2	0	0	0	0	0	2	1
Other	3	0	0	3	1	0	0	2	2
Other health conditions¶									
Total	8	0	0	8	4	1	0	4	8
All-cause mortality	3	0	0	3	1	1	0	1	3
Other	5	0	0	5	3	0	0	3	5

* Health condition and exposure groups are not mutually exclusive; studies may fall into multiple groups.

† Including carbon monoxide (CO), ozone (O₃), nitrogen dioxide (NO₂), sulfur dioxide (SO₂), benzene, benzo(a)pyrene (BaP), or other measured constituents of smoke not including particulate matter.

‡ Examples of proxies included exposure assignment based on occupation, location, time period, smoke or fire density, and Air Quality Index ratings.

§ The number of studies presenting any evidence for at least one risk association based on a critical value of $\alpha=0.05$ or an absence of the null value within 95% confidence intervals.

¶ Other health conditions include total mortality, atopic dermatitis, conjunctivitis, and other signs and symptoms not previously classified.

Abbreviations: PM, particulate matter; COPD, chronic obstruction pulmonary disease; CVD, cardiovascular disease; IHD, ischemic heart disease; MI, myocardial infarction; CeVD, cerebrovascular disease; TIA, transient ischemic attack.

3.2.3 Discussion

3.2.3.1 Summary, Strengths, and Limitations of Epidemiological Studies

The literature search identified nearly 200 peer-reviewed relevant articles published from January 2017 through February 2024. Most were published after 2021, which demonstrates an acceleration in health effects research commensurate with an increasing global threat of wildland fire that is likely to continue well into the foreseeable future. The group of eligible studies have evaluated wildland fire exposure-related risks in several populations worldwide, though information on working populations was sparse.

Among affected workers, the available research is almost exclusive to wildland firefighters, which may poorly reflect risks in other workers in outdoor environments. No studies focused on farm, forestry, or construction workers. Similarly, save for populations investigated in studies of reproductive and developmental health, other potentially vulnerable populations appear to be understudied, as few studies reviewed examined risk differences by sex, race, ethnicity, or socioeconomic status. Section 2.4 on Health Equity discusses the importance of these sociodemographic characteristics in addressing outdoor workers' unique occupational health needs.

Despite notable differences in available data and study designs, including variation in exposure durations and the timing of health effects, findings across studies were remarkably consistent. Although the evidence base for wildland fire effects was smaller compared with that of ambient air pollution, the wildland fire literature encompassed information on a wide range of physical health effects that aligned with the existing knowledge from decades of research on the impacts of ambient air pollution [IARC 2016; EPA 2019, 2022]. For example, there was considerable evidence of cardiorespiratory effects from wildland fire smoke.

Respiratory and cardiovascular health topics were studied most frequently in the literature. A notable portion of studies focused on reproductive and developmental outcomes among offspring of exposed adults. With the emergence of COVID-19, considerable attention was given to the effects of wildland fire smoke exposure on infectious disease.

Recent biomechanistic studies have also sought to identify cellular changes induced by exposure that can help explain the development of overt disease. These changes include genotoxicity, oxidative stress, DNA alterations, and inflammation, which are potential mechanisms for the development of various health conditions such as cancer, cardiovascular disease, metabolic disorders, respiratory illnesses, and neurological disorders.

Studies evaluating associations between exposure to wildland fire smoke and cancer, neurological outcomes, and metabolic conditions were less frequent in the included set of epidemiological literature. In general, the employed study designs allowed for more comprehensive study of acute conditions, such as hospital admissions for cardiorespiratory events, rather than chronic conditions that include the development of cancer or COPD.

1 While most studies used levels of PM_{2.5} or proxies for smoke (e.g., occupation, time period,
2 location, AQI) to define exposure, limited information was available on the direct health
3 effects of other hazardous agents related to wildland fires, such as ozone. Furthermore, few
4 studies differentiated between exposure to wildfire versus prescribed fire, let alone
5 identified differences in effects between the two sources of smoke. Finally, the effects of
6 exposures over multiple fire seasons remain largely unknown, as does the extent
7 (frequency and severity) of latent or persistent effects that may result from past exposures.

8 Many studies had notable strengths. First, most studies were population-based designs that
9 overall appeared adequately powered given large numbers of persons under observation,
10 particularly for common outcomes, such as respiratory and cardiovascular disease.

11 Second, a wealth of information was available for use in exposure assessment, including
12 satellite imagery, continuous area monitoring data, and meteorologic measurements. These
13 data were fit to exposure-response models using multiple techniques that not only
14 supported unbiased estimation of exposure, but also provided means to explore the
15 potential effects of measurement error, such as in Jiang et al. [2023].

16 Third, for most outcome categories, several studies were available, and within each
17 category, findings had remarkable consistency across a diverse set of studies. Collectively,
18 these findings show estimates that were resilient to differences in study design or selected
19 populations of interest.

20 Notable limitations were common in most studies reviewed. Limitations affect all
21 observational epidemiology study designs to some degree. A comprehensive evaluation of
22 study limitations is beyond the aims of this scoping review; however, some important
23 general limitations are described below.

- 24 • First, observational studies, on average, are more prone to bias compared with
25 randomized experimental designs.
- 26 • Second, individual-level data were not available in population-based studies that
27 dominated this review. Risk estimates from these ecologic or partially ecologic
28 designs were vulnerable to bias that may arise from the inherent inability to
29 characterize within-level variability in some or all variables (i.e., exposure, disease,
30 and covariates) used in exposure-response analysis. Similarly, incomplete
31 information on potential risk factors is unavoidable in most observational studies;
32 therefore, bias from residual confounding is a general concern.
- 33 • Third, analyses included varying levels of complexity. For example, many but not all
34 studies included some level of confounding adjustment, and only a handful
35 considered effect modification by important characteristics. Few studies corrected
36 for multiple comparisons to reduce error rate, and information was lacking on the
37 shape of the exposure-response among outcomes examined.
- 38 • Fourth, most observed effect sizes among outcomes were relatively small, leading to
39 increased vulnerability to all sources of bias. Additionally, effect size and
40 directionality might be influenced by the definition of exposure (i.e., exposure
41 misclassification or misspecification), particularly in studies without individual-

1 level exposure estimates or using proxies for exposure. Further, many effect
2 estimates for uncommon health conditions were imprecise in the face of small
3 sample sizes in some studies.

- 4 • Fifth, few studies provided information on temporality of the cause-and-effect
5 association. For example, it was not always clear whether the observed increase in
6 risk from wildland fire exposure reflected onset of a new condition, exacerbation of
7 a preexisting condition, or both. Similarly, it was not always clear whether the
8 increased risk immediately following exposure persisted long after exposure or
9 whether late onset effects could occur.

10 **3.2.3.2 Considerations for the Scoping Review Approach**

11 The approach taken in this scoping review has several limitations. First, the search strategy
12 has the potential for missed studies, either due to restrictive search terms, limited
13 databases, or misclassifications during review. Although these studies may have been
14 informative to the field, this misclassification likely did not make a difference to any specific
15 topics. Therefore, the conclusions would likely remain unchanged.

16 Second, study quality (e.g., confounding and bias) was not formally or quantitatively
17 evaluated for individual studies, as this is not a typical step of a scoping review given their
18 exploratory nature [Munn et al. 2018]. However, the EPA and IARC literature reviews [EPA
19 2019, 2022; IARC 2016, 2023] provided a robust quality assessment of relevant literature.
20 Nevertheless, studies included in the current scoping review were likely of varying levels of
21 quality. The extent to which imperfections in study design and incomplete study data
22 affected study findings is largely unknown.

23 Third, the current review did not include several topics in the wildland fire-related public
24 health literature. For example, studies discussing mental health and psychosocial outcomes
25 were excluded because the review was restricted to physical health effects. Occupational
26 mental health is a burgeoning field and NIOSH is involved in several initiatives to address
27 mental health issues among workers [NIOSH 2024; Schulte et al. 2024].

28 Studies on traumatic injury associated with wildland fire smoke were also excluded from
29 this review. EPA previously concluded that a causal relationship exists between PM and
30 visibility impairment [EPA 2009, 2019, 2022], which can present safety concerns and
31 increase the risk of injury.

32 The literature search also revealed 19 review articles, published 2017–2024, that
33 summarized the available evidence on associations between exposures to wildland fire
34 emissions and adverse physical health effects. These studies were excluded from data
35 extraction and synthesis because they lacked summary estimates from meta-analysis;
36 however, they are briefly described below as a potential reference source for future
37 research and in acknowledgement of their potential importance in the hierarchy of evidence
38 [Murad et al. 2016].

39 Among reviews of general findings, one study described short-term (acute) health impacts
40 [Barros et al. 2023], two studies reviewed long-term health effects [Gao et al. 2023b; Grant

1 and Runkle 2022] and four studies summarized health effects among workers [Groot et al.
2 2019; Hwang et al. 2023; Koopmans et al. 2022; Koopmans et al. 2020].

3 Reviews that focused on specific health effects included the following:

- 4 • Six on respiratory effects [Balmes and Holm 2023; Barros et al. 2023; Jiao et al.
5 2024; Noah et al. 2023; Stawovy and Balakrishnan 2022; Yu et al. 2023]
- 6 • Three reviewing birth outcomes [Amjad et al. 2021; Basilio et al. 2022; Evans et al.
7 2022]
- 8 • One each for CVD [Chen et al. 2021], neurological [Harris 2023], and ocular effects
9 [Jaiswal et al. 2022]

10 Given their direct relevance to this hazard review, findings from the four reviews of
11 occupational exposure are briefly described below.

12 Among reviews on occupational exposures, wildland firefighters comprised the largest
13 group under study and dominated the literature summarized in each review [Balmes and
14 Holm 2023; Noah et al. 2023; Stawovy and Balakrishnan 2022]. Along with wildland
15 firefighters, one review included support personnel, such as crews from the forest industry,
16 equipment operators, and related personnel at fire bases [Koopmans et al. 2020]. However,
17 none of the reviews included information on other potentially exposed workers who were
18 not directly involved in firefighting, such as farmworkers or construction workers.

19 A common theme among these studies was that they contained limited information on
20 workers; however, the available evidence of exposure-related acute cardiorespiratory
21 effects was generally consistent with that from larger population-based studies that have
22 generally shown evidence of a causal relationship. In contrast, the evidence on long-term
23 effects, such as cancer and chronic lung disease, was inadequate to make a causal
24 determination, primarily because of a paucity of relevant well-designed studies.

25 **3.2.3.3 Knowledge Gaps in Epidemiological Evidence**

26 This scoping review identified several areas for future research on health effects from
27 occupational exposure to wildland fire smoke. Perhaps of primary importance is the lack of
28 research investigating exposure-related effects among the various affected occupational
29 groups. In this scoping review, information on workers is sparse, with only a few studies on
30 wildland firefighters and one study of mounted police officers. These data, as well as studies
31 on general populations that comprise the bulk of the available literature, may poorly reflect
32 risks in other worker populations, such as agriculture, forestry, and construction.

33 Future research to expand the study of relationships between wildland fire smoke and a
34 wider array of potential health effects would be beneficial. More than half of the studies in
35 the scoping review focused on respiratory and cardiovascular effects, with the primary
36 outcomes assessed being acute effects, such as hospitalizations and emergency department
37 visits.

38 Studies evaluating associations between exposure to wildland fire smoke and cancer,
39 neurological effects, metabolic conditions, and uncommon reproductive and developmental

1 outcomes were far less frequent. For example, persistent and late-onset diseases (e.g.,
2 cancer, nervous system disorders, COPD) appear understudied. Valuable information on
3 exposure-response relationships and associated temporality, as well as the cumulative
4 effect of exposure to smoke from multiple wildland fires over time, could be obtained from
5 prospective or longitudinal studies that evaluate these effects.

6 Additionally, the body of research on COVID-19 and other infectious diseases is emerging
7 and could benefit from additional study.

8 Elucidating interrelationships between main health effects, comorbidities, and
9 sociodemographic factors following exposure to wildland fire smoke is another important
10 area for future research. For example, the lines between exacerbation of existing conditions
11 and new disease are often blurred in the studies reviewed. There is limited information on
12 how comorbid conditions affect the severity of health outcomes following exposure to
13 wildland fire smoke and whether interactions between chronic diseases may impose a
14 greater risk of adverse health outcomes. Likewise, health inequities (e.g., in access to care)
15 and social determinants of health may further impact the health of exposed workers,
16 possibly interacting with or modifying the effects of wildland fire smoke exposure.

17 Methods to improve exposure assessment are also needed. Personal sampling, along with
18 currently used methods, could be used to assess exposures by fire type and occupation.
19 Information on exposure routes other than inhalation, like dermal exposure and take-home
20 exposure of workers' families, is needed.

21 The different types of wildland fire smoke (e.g., wildfires, prescribed fires, wildland urban
22 interface fires) are complex and contain varied mixtures of harmful agents [EPA 2021].
23 Although considerable work has been completed for ambient air pollution, additional
24 research could help elucidate exposure-response functions for wildland fire emissions.
25 Differences in the toxicity of wildland fire smoke and ambient air pollution have not been
26 fully explored, as acknowledged in many HIAs in this scoping review.

27 Finally, more information is needed on main effects of and interactions between the various
28 toxic components within wildland fire smoke and other hazards in the occupational
29 environment, such as heat. It is not clear whether exposures to multiple hazards
30 encountered in outdoor and wildland fire environments act additively or synergistically on
31 the risks of adverse health conditions.

32

3.3 In Vitro and In Vivo Mechanistic Evaluation of Wildland Fire Health Effects

Studies examining biologic mechanisms of a causal relationship have a fundamental role in hazard identification. Mechanistic data stem primarily from experimental studies involving in vitro (e.g., cell culture systems, biologic molecules, tissue slices) and in vivo animal models (e.g., murine models). These data are generally used to support the biologic plausibility of a causal relationship between the agent of interest (e.g., wildland fire smoke) and human disease. This section describes select literature on relevant in vitro and in vivo studies. Comprehensive reviews of the literature have been conducted previously in support of the authoritative reviews described in Section 3.1. For this hazard review, NIOSH experts briefly described findings from key publications, including some literature published since the authoritative reviews, that stem mainly from a broad area of interest in wildland fire smoke and wood smoke. These include NIOSH investigations of the effects on wildland firefighters [Gaughan et al. 2014a,b] and the composition and toxicity of wildland fire smoke components [Leonard et al. 2000, 2007], as well as guidance on conducting evaluations [Gaughan et al. 2022].

Section 3.3 begins with a brief introduction of constituents in wildland fire smoke (Section 3.3.1), followed by an overview of the relevant toxicity mechanisms (Section 3.3.2). Sections 3.3.3 and 3.3.4 briefly describe salient findings selected from a limited review. Section 3.3.3 covers in vitro studies, while Section 3.3.4 covers in vivo animal studies. Both sections elucidate relevant modes of action.

3.3.1 Introduction

Wildland fire smoke is difficult to define as an exposure element. The smoke is made up of many different materials and may be in various states of biological reactivity. The chemical composition, oxidation state, size, and solubility can fundamentally affect the toxicological response. For example, wildland fire smoke from a dry grassland differs greatly from that of a deciduous forest or coniferous forest [Ma et al. 2022]. The soil where the fire is burning can also influence the material found in the smoke. Further, hot burning fires produce a different type of smoke than a smoldering fire or a peat fire.

Research shows that organic aerosols, dilution-driven evaporation, and related oxidation can be different in daytime wildland fire plumes and nighttime plumes [Liu-Kang et al. 2024; Palm et al. 2020]. The natural environment's vegetation, weather, and geography can also affect the complexity of the smoke generated. Further, the type of smoke and route of exposure needs to be taken into account while discussing biological reactions after exposure.

3.3.1.1 Combustion

Combustion of wildland material can happen in these phases, which are described below: distillation, drying, pyrolysis, char oxidation, and flaming combustion. All of these phases can produce airborne PM and gases.

1 Distillation involves volatilization of compounds in a liquid state as the vegetation heats up.
2 [Benkoussas et al. 2007; Morvan and Dupuy 2001]. Pyrolysis burns between 200°C and
3 250°C, depending on the plant material, and leads to the production of volatile gases and a
4 reactive char (char being the solid residue or carbonaceous material that remains) [Rowell
5 and LeVan-Green 2005]. Oxidation of the char results in smoldering or glowing combustion.

6 The pyrolysis and oxidation generate flammable gases that form the flame. This process
7 produces highly oxidized compounds (CO₂, NO_x, SO₂) [Lobert et al. 1991]. Flaming
8 combustion results in more glowing, smoldering combustion from the residual char
9 [Yokelson et al. 1999].

10 During a wildland fire situation, these events occur simultaneously and in close proximity.
11 The convective updraft of the wildland fire carries the materials into a smoke plume. This
12 plume, depending on atmospheric dynamics, has the greatest potential to have effects
13 beyond the local area. These volatile gases and highly oxidized compounds carried in the
14 particular matter are highly reactive, therefore making them materials of interest in human
15 exposure biological effects.

16 **3.3.1.2 Wildland Fire Smoke Composition**

17 Details on wildland fire smoke composition were provided in Chapter 2. Briefly in review,
18 wildland fire smoke has a complex chemical and physical composition determined by the
19 combustion conditions and fuel type [Cascio 2018; Kim et al. 2018]. Wildland fire smoke, in
20 general, is made up of coarse and fine PM, volatile organic compounds (VOCs), polycyclic
21 aromatic hydrocarbons (PAHs), gases (CO, CH₄, SO₂, NO_x, NO₂), and metals [Urbanski et al.
22 2009]. Air quality impacts are due to the primary pollutants (e.g., PM, CO, NO_x) and the
23 secondary pollutants (e.g., O₃, secondary organic aerosol) generated [Urbanski et al. 2009].
24 See Section 2.1 for a further discussion on chemical and physical properties of smoke.

25 PM consists of a mixture of the combusted compounds, particles high in carbon (both
26 elemental and organic), and metallic compounds [Urbanski et al. 2009]. PAHs are mainly
27 made up of naphthalene, retene, and phenanthrene [Navarro et al. 2017]. These materials
28 are carried into the atmosphere where they can further react and generate secondary
29 organic aerosols [Gilardoni et al. 2016; Liu-Kang et al. 2024; Palm et al. 2017]. The air
30 quality impact of wildland fires on biological systems depends on several variables: the
31 amount and chemical makeup of the smoke, weather, smoke plume dynamics, and the
32 atmosphere into which the smoke is dispersed [Urbanski et al. 2009].

33 **3.3.1.3 Generation and Composition Conclusions**

34 One of the biggest challenges in defining wildland fire smoke is determining what makes it
35 up. Because smoke composition is highly variable, and the specific composition dictates the
36 biological impact of exposure, it is difficult to generalize the data available. More research
37 examining the variability of the composition of wildland fire smoke and the resulting
38 difference in potential adverse health effects is needed. For example, knowledge gaps may
39 be reduced from more thorough and well-defined smoke elemental analysis. In one
40 approach, smoke could be generated from different source materials and temperatures, and
41 then collected at different times after the initial burn. These samples could be used to define

1 chemical and elemental makeup and then developed into a “smoke type” library. Other
2 conditions to consider include downwind temperature, atmospheric elevation, sunlight
3 exposure, and other factors that could affect the smoke’s reactivity. A “smoke library” could
4 be used when a fire breaks out to help determine the type and severity of exposure.

5 **3.3.2 Toxicity Mechanisms in Wildland Fire Smoke**

6 The main route of exposure to wildland fire smoke is by inhalation. The reviewed research
7 literature has several interpretations of the mechanisms behind wildland fire smoke
8 toxicity. The research on biological and toxicological effects supports three modes of
9 possible action: (1) oxidative stress and systemic inflammation, (2) neural receptor
10 reactions, and (3) translocation [Newby et al. 2015; Stone et al. 2017].

11 **3.3.2.1 Oxidative Stress and Systemic Inflammation**

12 Wildland fire smoke can generate oxidative stress and cause inflammation in exposure sites
13 and activate downstream signaling between cells. A review of cardiovascular actions
14 demonstrated that oxidative imbalance may be the key contributor to wildland fire smoke
15 toxicity [Miller 2020]. This imbalance can cause lipid peroxidation (the oxidative process of
16 lipid degradation by reactive oxygen species), activate platelets, inflammation of the
17 vascular endothelia, and changes in how blood vessels function.

18 Chemical characterization studies from both in vitro and in vivo research have found metals
19 in wildland fire smoke particles are known to induce oxidative stress via redox (reduction
20 and oxidation) mechanisms [Ghio et al. 2012a; Ghio et al. 2012b; Ghio et al. 2020; Leonard
21 et al. 2000; Leonard et al. 2007; Samet et al. 2020]. A study using human volunteers found
22 that wood smoke induced oxidative stress that can disrupt the cell cycle and initiate cellular
23 apoptosis [Muala et al. 2015].

24 The particle component of wildland fire smoke has been shown to generate an
25 inflammatory response in the lung once it is inhaled. Human, animal, and cellular studies
26 have found that, depending on variables such as dose, particle reactivity, and inhibited
27 clearance of particles, exposure may lead to systemic inflammation [Schwartz et al. 2020;
28 Stone et al. 2017]. Evidence of oxidative stress and systemic inflammation was revealed
29 during human, tissue, and cellular studies by demonstrating elevated blood markers of
30 inflammation, interleukin (IL)-1 β , IL-6, IL-8, TNF- α , and Clara cell protein (now referred to
31 as club cell secretory protein) [Forchhammer et al. 2012; Ghio et al. 2012a; Grilli et al.
32 2019].

33 **3.3.2.2 Neural Receptor Reactions**

34 Wildland fire smoke can react with neural receptors in the respiratory system, causing the
35 autonomic nervous system to affect blood pressure and heart rhythm. Cardiovascular and
36 heart rate studies in human volunteers have shown that wood smoke exposure can
37 significantly decrease heart rate variability, which is a measure of autonomic nervous
38 system function [Andersen et al. 2017; Unosson et al. 2013]. Wildland fire smoke can also
39 affect pulmonary receptors, baroreceptors, and chemoreceptors, altering cardiovascular
40 function [Perez et al. 2015]. The activated sensor nerves can send signals that lead to the

1 modulation of the baroreceptor that controls normal cardiovascular function and blood
2 pressure [Crabbe 2012]. The sensor nerves can also signal chemoreceptors in the carotid
3 body that maintain homeostasis of O₂, CO₂, pH, blood pressure, and temperature [Lopez-
4 Barneo et al. 2008]. Several investigations have shown significant changes in blood pressure
5 following wildfire smoke exposure [Baumgartner et al. 2011; Clark et al. 2013; Fedak et al.
6 2019].

7 **3.3.2.3 Translocation**

8 Studies on humans have shown that nanoparticles, gases, and other small molecules can
9 translocate through the alveolar membranes [Cascio 2018; Miller et al. 2012; Perez et al.
10 2015]. Both animal and human nanoparticle studies focusing on cell-cell junction and
11 permeability have shown the ability of some nanoparticles to translocate across the
12 alveolar-capillary barrier into the blood circulatory system [Furuyama et al. 2009; Schmid
13 et al. 2009]. Soluble materials, such as PAHs and metals, attached to these nanoparticles can
14 then diffuse into the bloodstream [Gerde et al. 2001].

15 Gaseous fractions of wildland fire smoke, such as SO₂, ammonia, NO_x, and CO, were found to
16 be important factors in vascular response in mice [Seilkop et al. 2012]. Therefore, studies
17 investigating a number of pathways and endpoints show the impact on human tissue can be
18 from several sources: (1) the gases dissolving into interstitial fluid, (2) the original exposure
19 material translocating as nanoparticles, (3) solubilized elements entering the blood stream,
20 and (4) the activation of signaling pathways within the organism.

21 **3.3.2.4 Other Types of Exposures**

22 Although lung toxicity is the focus of most investigations with wildland fire exposures, other
23 types of related reactions, such as the human ocular surface and epithelial layer, have been
24 examined. Evidence shows that wildland fire smoke can increase allergic responses and
25 upper airway disease. Mechanisms behind the disruption are disturbances of the epithelial
26 layer and the activation of an inflammatory or immune responses. While this evidence is not
27 consistent, it is an area of active investigation [Noah et al. 2023].

28 Wildland fire smoke exposure has also been implicated in functional changes in immune
29 cells and associated proteins in the bloodstream [Aguilera et al. 2023]. The ocular surface
30 has been investigated relating to wildland fire exposures and results demonstrated that
31 acute episodes could generate areas of ocular surface exposure that lead to an increase in
32 eye irritation [Berra et al. 2015].

33 Wildland fire smoke is being investigated for possible links to the onset and increases in
34 Alzheimer's disease because of the changes seen in DNA methylation, inflammatory
35 cascades, oxidative stress, and immune response effects [Schuller and Montrose 2020].
36 Preliminary evidence suggests that wildland fire smoke can accelerate neurological aging in
37 humans and affect learning capabilities. Wildland fire smoke and its derivative toxicants
38 interact with these aging mechanisms through telomere damage, cell senescence, epigenetic
39 effects, and mitochondrial disruption [Scieszka et al. 2023a].

3.3.2.5 Toxicity Mechanisms Conclusions

Toxicity studies have shown several possible mechanisms through which wildland fire smoke can affect the cardiovascular system. These mechanisms include (1) oxidative stress through systematic inflammation, (2) autonomic nervous system imbalance, (3) translocation of smoke elements entering the bloodstream, and (4) release of mediators into circulation. Further studies have investigated chemical grouping as a measure on toxicity response. Among groups that contain methoxyphenols versus those that do not, the methoxyphenol group showed highly significant negative relationships with cytokine activation and lung injury markers [Rager et al. 2021].

Investigations like these can help reveal the driving reactions that cause wildland fire smoke toxicity. Studies that further define wildland fire smoke composition, toxic effects, and activation mechanisms may help reduce associated adverse health effects. This research can inform occupational exposure limits, requisite personal protective equipment, health monitoring types needed, and potential treatment options.

3.3.3 Human In Vitro Investigations of Wildfire Smoke

Human in vitro cell models have been done to further examine how wildland fire smoke may impact human health. These exposures have included a number of smoke sources, target cells, and cell exposure models. These models have become more sophisticated over time, therefore improving their reflection of real-world exposures. Some (but not all) of these studies are described below to better illustrate important methods and findings.

3.3.3.1 Respiratory Tract Cells

Submerged Cell Cultures

One study exposed human monocytic cells (THP-1) to wood smoke extract. Results demonstrated a reduction in cell proliferation due to the effects on the S/G2 phase of the cell cycle. These same cells also showed increased membrane damage by measuring lactate dehydrogenase release [Bolling et al. 2012].

Smoke from bushfires, a common name for a wildfire involving low-growing plants such as scrub and brush, was used in a study that compared its effects with that of cigarette smoke. Bushfire smoke extract was prepared and exposed to phorbol myristate acetate (PMA) transformed THP-1 cells, which are human macrophage-like cells. Results showed effects upon the cell's pro-inflammatory pathways and viability. The results indicate that bushfire smoke extract impairs macrophage function similar to that of cigarette smoke [Hamon et al. 2018].

Wildland fire smoke extracts were exposed to human bronchial cells (BEAS-2B) to assess cell viability and genotoxicity, as well as different strains of *Salmonella typhimurium* (TA98, TA100) to assess mutagenicity. The extracts induced significant mutagenicity; however, no cytotoxicity or DNA damage was observed at the concentrations used. The results confirm that wildland fire smoke can be mutagenic [Gea et al. 2021].

1 In another study, human alveolar basal epithelial cells (A549) were exposed to a wood
2 smoke infused solution to examine the effects on alveolar epithelial barrier, cell migration,
3 and survival. The wood smoke was found to activate the p44/42, but not the p38 MAPK
4 pathway. This indicates that wood smoke may cause the breakdown of alveolar function
5 and structure through the p44/42 pathway, possibly leading to respiratory damage upon
6 chronic exposure [Zeglinski et al. 2019].

7 **Air-Liquid Interface Cell Cultures**

8 To investigate wildfire smoke exposures in an Australian brushland, one study used
9 submerged cultures on small airway epithelial cells and air-liquid interface (ALI)
10 differentiated primary bronchial airway epithelial cells. Wildfire smoke was found to inhibit
11 autophagic flux and cause barrier inhibition in airway epithelium. Because autophagy is
12 important in viability, cell repair, and inflammatory regulation, inhibiting this process may
13 lead to aggravation of respiratory conditions [Roscioli et al. 2018].

14 An air-liquid interface exposure system was used to expose immortalized human
15 tracheobronchial epithelial cells to measure diagnostic biomarkers. Authors used wildfire
16 smoke from two unique sources. The origin of the wildfire smoke and materials that made
17 up the smoke was found to influence the resulting long noncoding ribonucleic acid (RNA)
18 expression in exposed lung cells. These RNA molecules have proven to be important in
19 many biological processes and are becoming prominent diagnostic biomarkers for human
20 disease. These results demonstrate that wildfire smoke sources and type can impact its
21 toxic effects [Nguyen et al. 2023].

22 A recent paper used ALI to expose human airway epithelial cells to wood smoke. Authors
23 found that oxidative stress was not related to carbon monoxide. The use of the air-liquid
24 interface allowed direct exposure to the wood smoke, avoiding the alterations caused by
25 adding media or residue concentration. The results demonstrate the advantages of an
26 improved model of human airway exposures to smoke and other effectors [Abzhanova et al.
27 2024]. This study can serve as a model for comparisons between two or several different
28 types of smoke and their effects on exposed cells.

29 **3.3.3.2 Non-Respiratory System Cell Cultures**

30 Mitroo et al. [2024] exposed ocular (ARPE-19) cells to soot generated in a laboratory to
31 mimic materials found in wildland fire smoke sources. Using a range of combustion
32 properties relating to temperature, researchers generated and exposed oxygen content and
33 fuel type soot to the cells. The changes observed in these properties were found to affect
34 ARPE-19 cell toxicity [Mitroo et al. 2024].

35 In another study, ash sample extracts from a wildfire were used to examine bioassay
36 responses in several cell types including human breast carcinoma (VM7luc4E2 and
37 T47DLucARE) cell lines. Researchers measured nuclear, estrogen, and androgen receptors
38 along with markers of stress IL-8 and cyclooxygenase-2. The receptors were generally
39 unaffected while the markers of stress were found to be significantly higher [Young et al.
40 2021].

1 Liu et al. [2005] exposed cultured human pulmonary artery endothelial cells to condensed
2 wood smoke. Results showed increases in reactive oxygen species (ROS) generation,
3 mitochondria membrane disruption, DNA fragmentation, and increased mRNA expression
4 for superoxide dismutase and heme oxygenase, all of which are effects in free radical
5 production imbalance.

6 **3.3.3.3 Conclusions from In Vitro Studies**

7 Results from the limited in vitro studies available showed that wildfire smoke can affect
8 cells in a variety of ways. Studies found (1) cell membrane damage, (2) structural effects, (3)
9 activation of inflammatory pathways, (4) DNA and RNA damage/alteration, and (5)
10 cytokine activation. However, literature searches revealed a deficiency of human-based
11 cellular research using wildfire smoke.

12 Most of the studies cited used smoke extract with concentrates or soot as a source for
13 cellular exposure. These forms do a poor job of modeling real-world exposures. This is
14 because the smoke material potentially reacts with the diluent and the cell media before it
15 reacts with the cell surface. More valid models of smoke exposure in humans can be
16 explored because of advanced ALI methods and better varieties of human respiratory tract
17 cells (i.e., immortalized, normal, and primary) available for research. With the growing
18 number, duration, and severity of wildfires, all research areas on human exposure to
19 wildfire smoke should be pursued. Developing a smoke library, better defining smoke
20 characteristics, and employing advanced in vitro methods would yield the most impactful
21 results in discovering mechanisms of action. These methods include air-liquid interface
22 (ALI) cultures, lung-on-chip microphysiological systems, and precision-cut lung slices.

23 In vitro work can be a relatively fast and inexpensive method to provide extensive
24 information on cell type differences and the varied reactions to different smoke types. The
25 current drawback of in vitro studies is their validation to human disease due to use of
26 immortalized cells from healthy tissue as normal cells while they are not normal because of
27 their altered DNA for continuous proliferation and use of cancer cell lines as a substitute of
28 primary healthy cell. As more in vitro studies are performed, and better models are
29 developed, this validation gap needs to be addressed. In vitro research can help define
30 initial mechanisms of cellular reactions including inflammatory markers and downstream
31 signaling. In vitro studies can also help researchers measure cell viability, DNA damage, and
32 mutagenicity. Currently, data from human in vitro work representing respiratory tract,
33 bloodstream, dermal, and ocular model systems, along with allergic reactions, are very
34 limited.

35 **3.3.4 In Vivo Studies**

36 While human exposure to wildland fire smoke has become more common in recent years,
37 much less in vivo animal model research specific to this exposure has taken place compared
38 with other widely studied particulate exposures. Decades of air pollution/particulate matter
39 and engineered nanomaterial research using animal models have established general
40 mechanisms of pulmonary toxicity following inhalation exposure.

1 Research has shown that toxicity is not confined to the lung, toxicity can cause adverse
2 systemic effects as well. These can involve cardiovascular, immune, altered gut microbiome,
3 pregnancy, and neurological issues. The systemic adverse effects can occur by (1) acute
4 autonomic effects at the time of exposure, (2) direct effects of translocation with toxicants
5 leaving the lung and interacting with systemic tissues, and/or (3) a change in the circulating
6 mediators consistent with the effects of the pulmonary exposure.

7 The next section examines *in vivo* pulmonary exposures, because inhalation is the primary
8 route of exposure to the outdoor worker. It is not meant to be inclusive of all published
9 studies. Illustrative studies are discussed to highlight the potential for toxicity associated
10 with wildland fire exposure. Few studies specifically address wildland fire smoke exposure
11 *in vivo*, and most use a surrogate wood smoke generated exposure.

12 **3.3.4.1 Pulmonary**

13 **General**

14 A series of studies investigated the toxicity of PM_{10-2.5} (thoracic coarse particle fraction) and
15 PM_{2.5} (fine fraction). The PM studied was collected from the June 2008 wildfires in central
16 and northern California. The wildfire-associated PM was compared with ambient PM
17 collected during June 2007 conditions without wildfire contribution. Male BALB/c mice
18 were exposed to 0–100 µg of the different PM by intratracheal instillation.

19 The wildfire PM caused inflammatory cell influx, lung injury, and inflammatory cytokine
20 production. It also reduced the antioxidant capacity of the lung, increased macrophage
21 cytotoxicity, increased isoprostane concentrations as a biomarker of lipid peroxidation, and
22 decreased intracellular Clara cell (club cell) secretory protein [Wegesser et al. 2009;
23 Wegesser et al. 2010; Williams et al. 2013].

24 Heat-treated wildfire PM reduced the inflammatory and oxidative stress response,
25 suggesting organic components contributed to the toxicity [Wegesser et al. 2010]. The
26 wildfire PM was approximately 10-fold more damaging to the lung compared with
27 representative ambient PM lacking wildfire contribution. Of note, the toxicity comparison of
28 PM was done on an equal mass basis. This means that ongoing wildfires could lead to a 2- to
29 4-fold increase in PM compared with normal ambient PM levels, further exacerbating the
30 potential toxicity in humans [Wegesser et al. 2009; Wegesser et al. 2010; Williams et al.
31 2013].

32 In a similar manner, toxicity was evaluated for coarse, fine, and ultrafine PM fractions
33 derived from the 2008 North Carolina peat fire. The samples were collected during the
34 active/smoldering phase or during the nearly extinguished phase, which was more
35 representative of ambient PM. Female CD-1 mice were exposed by oropharyngeal
36 aspiration to 100 µg of peat fire PM. At 4 and 24 hr post-exposure, the coarse PM fraction
37 collected from the active smoldering fire was the most inflammatory to the lung. The
38 authors noted that this fraction contained the most lipopolysaccharide content given the
39 peat composition and the lower smoldering temperatures compared with an active wildfire
40 [Kim et al. 2014].

1 The EPA Environmental Public Health Division, along with collaborators, conducted a series
2 of studies evaluating the toxicity of surrogate wood smoke/biomass aerosols. Five biomass
3 fuels, northern red oak, peat, ponderosa pine needles, lodgepole pine, and eucalyptus, were
4 used. Smoke condensate was collected from flaming and smoldering conditions. The
5 samples were then used to expose female CD-1 mice at 100 μg by oropharyngeal aspiration.
6 In general, the flaming samples were more toxic to the lungs by inducing greater
7 inflammatory cell influx, cytokine production, and lung injury compared with the
8 smoldering samples with peat and eucalyptus being the most toxic [Kim et al. 2018].

9 Eucalyptus, northern red oak, and peat were used to expose female BALB/c mice by
10 inhalation to either flaming or smoldering conditions. The mice were exposed to 4
11 milligrams per cubic meter (mg/m^3) of flaming and 40 mg/m^3 of smoldering PM for 1 hr/d
12 for 2 days, with pulmonary evaluation at 4 hr and 24 hr post-exposure. Factored for
13 estimated deposited PM mass, the flaming peat and eucalyptus biomass were the most toxic
14 to the lung compared with smoldering, while the oak biomass had minimal effects. [Kim et
15 al. 2019].

16 Using a similar approach, female CD-1 mice were exposed to 100 μg of flaming or
17 smoldering peat or red oak by oropharyngeal aspiration. As seen in the previous studies,
18 the flaming samples had the greatest toxicity for the lung [Carberry et al. 2022].
19 PM composition and combustion phase were the driving factors for toxicity outcomes.

20 Research studies done by the Lovelace Respiratory Research Institute evaluated hardwood
21 smoke exposures. Male and female CDF(F-344)/CrIBR rats were exposed to hardwood
22 smoke (60% black and 40% white oak) at 30, 100, 300, and 1,000 $\mu\text{g}/\text{m}^3$, 7 days per week
23 (d/wk) for 6 months. No overtly toxic responses to the lung were indicated. The results
24 showed that many of the effects were not linear, with males more affected than females.
25 Hardwood smoke induced greater oxidative stress than diesel exhaust [Seagrave et al.
26 2005].

27 A second study of similar design with hardwood inhalation exposure from black and white
28 oak found only mild effects following a subchronic 6-month exposure time frame. Only
29 macrophage accumulation was noted in terms of histopathology [Reed et al. 2006]. The use
30 of oak as the hardwood could be responsible for the mild to minimal pulmonary effects
31 similar to the comparative studies of Kim et al. [2018, 2019].

32 In other studies, male C57BL/6J mice were exposed by inhalation to aerosols of pine or
33 spruce smoke for 4 hr/d for three consecutive days. The total suspended particle
34 concentration was 6–10 mg/m^3 , on average, with estimated lung deposition of 32.1
35 nanograms per square centimeter (ng/cm^2) per day. In general, the pulmonary
36 inflammatory response was mild with more inflammatory protein production observed for
37 spruce aerosols [Ihantola et al. 2020].

38 In another study, Danielsen et al. [2010] exposed male Fisher-344 rats to wood smoke PM
39 at 0.64 milligrams per kilogram (mg/kg) by intratracheal instillation. The PM samples
40 included particulates from four sources: a rural area, a wood stove-rich area, a wood stove
41 with normal oxygen supply, and a wood stove with low oxygen supply. The authors noted

1 neutrophilic inflammation and inflammatory gene expression. The particulate from a wood
2 stove with low oxygen supply tended to have the greatest effect. Using wood smoke
3 particles derived from *Pinus edulis* to mimic Native American communities in New Mexico,
4 Brown Norway rats were exposed by inhalation up to 12 weeks at 10 mg/m³. Inflammation
5 and histopathologic changes were noted but overall were considered mild [Tesfaigzi et al.
6 2002].

7 Emerging burn pit exposure studies offer an additional complex mixture surrogate to
8 consider. As wildland fires increase in number, the wildland-urban interface will continually
9 be affected. A study by Kim et al. [2021] evaluated smoke emission condensates (flaming or
10 smoldering conditions) from various combinations of plywood, cardboard, and plastic.
11 Female CD-1 mice were exposed to 100 µg of various mixtures by oropharyngeal aspiration.
12 Greater pulmonary toxicity was in general found for aerosol generated under flaming
13 conditions and for those containing plastic [Kim et al. 2021].

14 **Airway Reactivity**

15 A series of studies were performed to examine airway reactivity using the guinea pig as a
16 model. In wood smoke studies (lauan or pine wood), the exposure resulted in the following
17 conditions: (1) increased inflammatory cell influx and cytokine production, (2) increased
18 matrix metalloproteinases and tissue inhibitors of metalloproteinases, (3) increased
19 oxidative stress, (4) acute mitochondrial-dependent reductions in respiration, (5) increased
20 airway permeability, and (6) pathological changes including hyperplasia [Granados-Castro
21 et al. 2015; Lin and Kou 2000; Lin et al. 2001; Ramos et al. 2009; Ramos et al. 2013; Ramos
22 et al. 2021]. These studies indicated the guinea pig was a responsive pulmonary model.

23 Airway hyperresponsiveness was seen in several studies as rapid onset and in response to a
24 second exposure following a previous exposure [Hsu et al. 1998a, Hsu et al. 2000]. The
25 mechanisms centered around oxidative stress, cholinergic signaling, and tachykinins [Hsu
26 and Kou 2001; Hsu et al. 1998b; Hsu et al. 2000]. Intervention studies evaluating these
27 specific mechanisms lessen bronchoconstriction [reviewed in Adetona et al. 2016].

28 **Allergic Response**

29 Eucalyptus, northern red oak, and Irish peat were used to expose female BALB/c mice by
30 inhalation to either flaming or smoldering conditions using a house dust mite allergic
31 model. All conditions reduced respiration with some of the smoldering conditions being
32 worse. The allergic groups had similar or weakened responses compared with controls
33 [Hargrove et al. 2019].

34 Following ovalbumin challenge, a method to stimulate an allergic reaction, acute hardwood
35 smoke inhalation caused a mild exacerbation in allergic inflammation. However, this
36 exacerbation did not occur when an 11-day recovery period took place between the
37 ovalbumin and hardwood smoke exposure [Barrett et al. 2006]. In Brown Norway rats
38 treated with ovalbumin and exposed to wood smoke (1,000 µg/m³) for 70 days, pulmonary
39 function was altered, resulting in increased inflammation following an allergen challenge
40 compared with a filtered-air control group [Tesfaigzi et al. 2005].

1 Infection Susceptibility

2 Pulmonary exposure in the occupational setting can affect how the lung responds to a
3 secondary infection. In studies by Zelikoff et al. [2002], Sprague-Dawley rats were exposed
4 to wood smoke generated from red oak for 1 hr/d for 4 days. At various times from 3–120
5 hr following exposure, rats were challenged with *Staphylococcus aureus*. The inhaled wood
6 smoke persistently suppressed bacterial clearance [Naehrer et al. 2007; Zelikoff et al. 2002].

7 Similar adverse findings include wood smoke exposure as a mixture of soft woods (fir, pine,
8 etc.) in BALB/c mice, which decreased the ability to clear *Streptococcus pneumoniae* in a
9 macrophage dependent manner [Migliaccio et al. 2013]. Mice exposed to smoldering
10 plywood smoke three times for 2-minute intervals induced pulmonary inflammation and
11 susceptibility to a CXCL1 dependent *Pseudomonas aeruginosa* [Dunn et al. 2018]. Further,
12 immune suppression effects are not confined to the lung. For example, A/J mice exposed to
13 hardwood smoke for 6 months by inhalation (30–1,000 $\mu\text{g}/\text{m}^3$) resulted in systemic
14 immune suppression, as measured by splenic T-cell responses [Burchiel et al. 2005].

15 However, not all responses reported were adverse. Male C57BL/6 mice exposed for up to 6
16 months at 1,000 $\mu\text{g}/\text{m}^3$ to oak hardwood smoke had no altered clearance of *Pseudomonas*
17 *aeruginosa* [Reed et al. 2006]. Male C57BL/6 mice challenged with single or repeated
18 administrations of wood smoke particles (100 μg or 250 μg) by oropharyngeal aspiration
19 were then challenged with an influenza virus. In this study, the severity of viral infection
20 was reduced with prior wood smoke exposure [Vose et al. 2021]. Samuelson et al. [2009]
21 exposed female BALB/c mice to *Listeria monocytogenes*. This was done simultaneously or at
22 1- or 7- days post-exposure to wood smoke particles (100 μg ; birch wood). These mice had
23 a reduced pulmonary bacteria load 24 hr after exposure.

24 Grouping / Modeling

25 For air pollution/wildland fire exposures, the specific composition or combination of
26 pollutants in the air determines the toxicity. To estimate the risks associated with highly
27 variable types of wildland fire smoke, it is important to understand how different
28 components and combinations of mixtures contribute to adverse health effects. The
29 knowledge can lead to developing generalizable information that can be used to assess the
30 potential harm caused by wildland fire exposure.

31 An initial study used multiple additive regression trees to evaluate different emissions
32 including wood smoke. The combined analysis indicated the gases sulfur dioxide, ammonia,
33 nitrogen oxides, and carbon monoxide were chemical components associated with
34 cardiovascular disease pathways. However, the study was limited by the few experimental
35 endpoints for discrimination to determine causal relationships [Seilkop et al. 2012].

36 Rager et al. [2021] found that coupling mixture computational modeling with chemical and
37 in vivo biological response profiles allowed for better interpretation than treating an
38 aerosol as a single unit or evaluating by a single chemical. The analysis helped identify
39 potential repressors (e.g., methoxyphenols) and inducers (e.g., inorganic elements, ionic
40 constituents) of biological responses [Rager et al. 2021].

1 In another study, five biomass fuels (northern red oak, peat, ponderosa pine needles,
2 lodgepole pine, and eucalyptus) were used to expose female CD-1 mice at 100 µg by
3 oropharyngeal aspiration. Collected smoke condensate for exposures was collected from
4 flaming and smoldering conditions. Pulmonary transcriptomic data were used as a tool for
5 grouping by similarity scoring. The results indicated that different biomass exposure
6 groupings mimicked pulmonary transcriptomics with flaming peat and eucalyptus
7 exposures being the most toxic. Conversely, grouping by chemical composition (86 different
8 chemicals) did not match the grouping by pulmonary transcriptional outcomes, although it
9 was noted that using more sensitive chemical fingerprints in the future may help [Koval et
10 al. 2022]. As computational methodology and experimental data increase, understanding
11 combinations of mixtures related to specific biological outcomes will emerge.

12 **3.3.4.2 Cardiovascular**

13 Particulate matter inhalation exposure is widely established to contribute to cardiovascular
14 morbidity and mortality [Brook et al. 2010]. In vivo studies illustrated the mechanisms,
15 which include autonomic dysfunction, inflammatory mediator spillover into the general
16 circulation, and particle translocation.

17 The research of various engineered nanomaterials, from carbon-based materials to metal
18 oxides, indicated very similar findings. In fact, the cardiovascular system can be more
19 sensitive to a pulmonary exposure than the lung itself [Nurkiewicz et al. 2006; Nurkiewicz
20 et al. 2008]. In terms of wildfire smoke, a review of the epidemiological studies on wildfire
21 or household biomass smoke, controlled human exposure studies, in vivo studies, and in
22 vitro studies generally have a positive association for adverse-cardiovascular-related
23 outcomes. However, significant research gaps remain [Chen et al. 2021].

24 Several studies evaluated cardiac function. As described in the pulmonary section, coarse,
25 fine, and ultrafine fractions of PM were derived from samples collected during the
26 active/smoldering or nearly extinguished portion of the 2008 North Carolina peat fire.
27 Female CD-1 mice were exposed by oropharyngeal aspiration to 100 µg of peat fire PM. At
28 24 hr post-exposure, hearts were removed and challenged using the Langendorff isolated
29 heart perfusion model.

30 Kim et al. [2014] reported that baseline hemodynamics remained unaltered by exposure.
31 However, mice exposed to the ultrafine fraction of smoldering stage, but not glowing stage
32 PM exhibited decreased cardiac function and increased infarct size following ischemia
33 reperfusion. The researchers concluded that both particulate size and source influence
34 cardiovascular responses. Notably, they found that active/smoldering PM contained four
35 times more organic matter than nearly extinguished PM, suggesting a relationship between
36 PM chemical composition and physiological effects.

37 Ultrasound was used to assess the cardiac function of male Sprague-Dawley rats 24 hr after
38 they were exposed to 35 µg or 350 µg of peat smoke PM extracts. Exposure was associated
39 with an altered regulation of left ventricular volumes and pulmonary artery hemodynamics
40 suggesting irritant/autonomic responses [Thompson et al. 2018].

1 In another study, male Wistar-Kyoto rats with implanted telemetry devices were exposed
2 by inhalation to low (0.38 mg/m³) and high (4.04 mg/m³) concentrations of peat smoke for
3 1 hr. Exposure increased systolic and diastolic blood pressure. The low peat exposure
4 elevated baroreflex sensitivity and induced cardiac arrhythmia; however, the high exposure
5 did not have the same effect. The authors considered that exposure may affect homeostatic
6 function for the cardiovascular system. They concluded that combustion pollutants can
7 induce sensitivity of effect in a nonspecific fashion [Martin et al. 2020a].

8 Male BALB/cOlaHsd mice were exposed to spruce pellet derived biomass burning by
9 intratracheal instillation. The mice received one dose of 50 µg or three repeated doses of 50
10 µg every third day. The effects of the collected biomass sample were modest, but some
11 inflammatory measures were increased in the heart. The responses were less and different
12 when compared with diesel exhaust particles. These results suggest that chemical
13 composition influences the outcomes [Farina et al. 2019].

14 Implications for vascular effects and altered circulating factors impacting endothelial cell
15 function have been previously described. In this study, male ApoE^{-/-} mice, a model of
16 atherosclerosis, were exposed to smoldering Douglas fir smoke by whole body inhalation
17 for 2 hours a day, 5 days a week, for a total of 8 weeks. The exposure was entirely volatilized
18 organic material. The deposition aimed to mimic a mid-career wildland firefighter.
19 Exposure induced aortic thickening, stiffening, and reduced augmentation capacity
20 occurred. Using magnetic resonance imaging, larger end-systolic volume with reduced
21 ejection-fraction was measured in the exposed mice. These effects may contribute to
22 microvascular damage while increasing the risk of cardiac failure and ischemia [Eden et al.
23 2023].

24 Male C57BL/6 mice were exposed for 6 hours to various pollutants including hardwood
25 smoke (380 µg/m³) generated from oak using a wood burning stove. Serum was collected
26 18-hr post-exposure and used to expose aortic rings collected from naïve mice and murine
27 cerebrovascular endothelial cells. Serum collected from wood smoke exposed mice was able
28 to induce inflammation in endothelial cells and decrease vasorelaxation in the aortic rings
29 of naïve mice. These results indicate exposure to wood smoke induces circulating factors
30 that may contribute to endothelial and vascular dysfunction [Aragon et al. 2016].

31 Flaming or smoldering peat or red oak was used to expose female CD-1 mice (100 µg) by
32 oropharyngeal aspiration. Transcriptional changes were differentially expressed in the
33 heart and lung following flaming exposures. Altered miRNA expressions in circulating,
34 extracellular vesicles suggested cardiovascular disease and a hypoxia/cell stress mediated
35 response, especially for exposures to the flaming condensates. Integrating the
36 transcriptional changes with the extracellular vesicle altered miRNAs indicated crosstalk
37 between the pulmonary and systemic tissues (meaning the pulmonary response impacted
38 the systemic response) [Carberry et al. 2022].

39 Individuals with underlying cardiovascular risk factors are known to be at higher risk
40 following PM exposure [Brook et al. 2010]. Studies specific to wildland fire exposure are
41 lacking, but evidence suggests that wood smoke derived PM can alter cardiovascular

1 adaptations. Male Wistar Kyoto rats were exposed to peat smoke generated from an Irish
2 bog at low (0.36 mg/m³) and high (3.30 mg/m³) concentrations for 1 hr. The rats were then
3 administered a high-fat substance (high-fat challenge) directly into their stomachs (i.e.,
4 gavage). Cardiac effects were generally unaffected with the exception of an increase in
5 isovolumetric relaxation time following low peat exposure [Martin et al. 2018].

6 A similar concept was employed using male Sprague-Dawley rats exposed by inhalation to
7 eucalyptus smoke for 1 hr at 700 µg/m³ followed by gavage of a high carbohydrate
8 suspension at 24 hr post-exposure. Cardiovascular assessments were made 2 hr following
9 the gavage to assess postprandial responses. The authors noted that eucalyptus exposure
10 modified cardiac function, including cardiac output, stroke volume, and ejection fraction, in
11 response to a high carbohydrate exposure. They concluded that a single exposure could
12 sensitize the cardiovascular response to systemic triggers. If the exposure was prolonged, it
13 may have the potential to contribute to progression of cardiovascular disease and
14 remodeling [Martin et al. 2020b].

15 In another study, Martin et al. [2023] exposed male Sprague-Dawley rats to eucalyptus
16 wood smoke by inhalation in a single (1 hr at 964 µg/m³) or repeated (2 times per week for
17 4 weeks) design. The researchers aimed to assess the cardiovascular impact following an
18 exposure combined with disruption of the sleep cycle. They found that wood smoke
19 exposure exaggerated sleep-disruption-induced-changes in heart rate and blood pressure,
20 as well as altered ventricular gene expression. These results indicate the potential for
21 worsened sleep-disruption-related cardiovascular pathophysiology when in conjunction
22 with a wood smoke exposure [Kyle Martin et al. 2023].

23 **3.3.4.3 Neurological**

24 To date, the most convincing study evaluating neurological effects occurred at a mobile
25 laboratory in New Mexico, more than 300 kilometers away from the naturally occurring
26 wildfires in California, Arizona, and Washington. Male C57BL/6 mice were exposed by
27 inhalation to an average of 104 µg/m³ PM_{2.5} for 4 hr/d for 20 days. They were sacrificed 24
28 hr after the final exposure. The exposure would be equivalent to <20 µg/m³ PM_{2.5} for a 24-
29 hr period, adding to the relevance of the study.

30 The researchers used levoglucosan, a marker of wood smoke, to indicate days when wood-
31 derived PM_{2.5} made the greatest contribution. Although the effects were modest, increased
32 inflammatory markers and macrophage accumulation were found in the lung. The response
33 in the brain included neuroinflammation, decreased neuroprotective metabolites, and
34 altered markers consistent with accelerated aging and Alzheimer's disease and related
35 disorder pathogenesis. While not directly compared, the authors further indicated that
36 based on previous research, the wildfire PM was more potent than other mediators of air
37 pollution (e.g., diesel emissions, ozone) in inducing adverse neurological effects [Scieszka et
38 al. 2022].

39 Few studies using surrogate wood smoke exposures exist. A study of pinon wood chips to
40 generate wood smoke was used to expose female C57BL/6 mice by inhalation to 500 µg/m³
41 for 4 hr per day for 14 days with post-exposure points of 1, 3, 7, 14, and 28 days. The

1 pulmonary exposure caused a change in the phenotype of cerebrovascular endothelial cells
2 in a temporal fashion, supporting a proinflammatory state. An increased expression of
3 inflammation markers in microglia began at day 7 post-exposure with an overall increase in
4 the percentage of activated microglia at day 28. These results suggest that following a
5 pulmonary wood smoke exposure, a dynamic endothelial cell neuroinflammatory response
6 transitions into an immune cell response. Microglial activation was sustained through 28
7 days post-exposure, suggesting the potential for long-term changes [Scieszka et al. 2023b].

8 While not a traditional inhalation design model, smoke particulate generated from different
9 wood sources was applied ex vivo to brain nerve terminals from male Wistar rats. The
10 smoke aerosols were collected during the entire combustion phases from flaming to
11 smoldering. The direct exposure of the nerve terminals to the different smoke aerosols was
12 considered adverse by the alteration in GABA and glutamate uptake. Furthermore, different
13 smoke aerosols conveyed differential responses [Paliienko et al. 2022]. These studies
14 implicate potential toxicity if particles can directly translocate to the brain.

15 **3.3.4.4 Cancer**

16 IARC has designated outdoor air pollution as carcinogenic to humans (Group 1). This
17 designation was made because of the sufficient evidence for increased lung cancer and the
18 positive association for bladder cancer [IARC 2015]. IARC also recently designated
19 firefighting as an occupation as a Group 1 carcinogen for mesothelioma and bladder cancer
20 [IARC 2023]. Lung cancer was not noted with the current available research. Both IARC
21 designations were supported with strong mechanistic evidence that included many key
22 characteristics of a carcinogen (see Section 3.1.1.3).

23 Several studies have indicated positive associations of wood smoke using a mutagenicity
24 assay [Asita et al. 1991; Kim et al. 2018; Mutlu et al. 2016] or through DNA damage
25 [Ihantola et al. 2020]. These came from studies assessed by comet assay in collected lavage
26 cells and lung tissue, with greater effects seen for pine aerosols compared with spruce
27 [Ihantola et al. 2020].

28 In the study by Kim et al. [2018], authors found smoldering biomass samples to be more
29 mutagenic while flaming biomass samples were more toxic to the lung. Another study
30 exposed A/J mice by inhalation to hardwood smoke (60% black and 40% white oak), for up
31 to 1,000 $\mu\text{g}/\text{m}^3$ for 6 months with 6 months recovery. Authors noted no increased tumor
32 incidence or multiplicity compared with air controls [Reed et al. 2006]. In a study of rats
33 and mice exposed to representative indoor wood smoke for 15–19 months, authors found a
34 significant increase of tumorigenesis in mice (17% in control group vs. 46% in exposed) but
35 not rats (1% vs. 0%) [Liang et al. 1988].

36 **3.3.4.5 Other**

37 Few studies have looked at other endpoints known to be affected by particulate exposure
38 for wildland fire exposure. One active area of evaluation is an investigation of gut
39 microbiome effects following a toxicant exposure. Wood smoke inhalation exposure in
40 ApoE^{-/-} mice at 450 $\mu\text{g}/\text{m}^3$ for 50 days altered diversity and profiles of microbiota in a
41 direction consistent with inflammation [Fitch et al. 2020].

1 Pregnancy, including generational studies, is another area of active toxicology research
2 involving inhalation exposures. Authors observed adverse reproductive effects in rhesus
3 macaque monkeys exposed to the 2018 California Camp Fire wildfire. These effects included
4 loss of pregnancy with altered behavioral responses including impaired memory. Sensitivity
5 early in pregnancy was also indicated [Capitanio et al. 2022; Lasley 2023; Willson et al.
6 2021].

7 PM exposure is suggestive of but not sufficient to infer a causal relationship for metabolic
8 syndrome (Section 3.1.1.1). Wildland fire smoke showed modest but positive associations in
9 exposed humans (Section 3.2.2.5). However, no in vivo studies were identified exploring
10 metabolic syndrome with wildland fire exposure. While other routes of exposure were not
11 considered for this section, oral and dermal exposures cannot be dismissed. For example,
12 one study found that male F344 rats orally exposed to wood smoke PM induced oxidative
13 and mutagenic damage to DNA in the liver [Danielsen et al. 2010]. These studies reflect the
14 sparsity of data in certain toxicological models, especially with pregnancy, and they
15 illustrate the concentrated need for additional research.

16 **3.3.4.6 Conclusions from In Vivo Studies**

17 Overall, these highlighted studies on PM-related exposure from wildland fires indicate
18 toxicity with similar mechanisms of pulmonary and systemic effects as seen in the broader
19 PM literature. A few studies with direct comparisons show a greater level of toxicity for
20 wildfire PM than representative PM without added combustion components. Systemic
21 responses, including neurological and cardiovascular, are sensitive endpoints. Surrogate
22 wood smoke/biomass studies indicate that toxicity depends on phase (flaming vs.
23 smoldering) and composition. Adding man-made materials, such as plastics or engineered
24 composites, have the potential to further exacerbate toxicity.

25 Fewer studies of wildland fire smoke or its surrogate have been done compared with
26 research on air pollution/PM or engineered nanomaterial. Moving forward, more
27 information is needed. Studies looking at exposures involving PM during a wildland fire
28 event are extremely informative. However, few of these studies exist compared with
29 surrogate-designed research. There is an opportunity to expand these studies when
30 possible.

31 Well-defined mixture studies would be beneficial to evaluate the primary components
32 and/or mixtures contributing to the pulmonary and systemic effects seen following
33 inhalation exposure. Expanding data in this area will also aid computational models for risk
34 prediction. For the outdoor worker, understanding the impact of the aging of wildland fire
35 particles in the air, and how the particles can affect health, is critical, especially when
36 exposure can occur even at a considerable distance from the source. Further, when wildland
37 fire affects air quality, outdoor workers (e.g., agriculture, construction) may experience co-
38 exposures with risks through both occupational hazards and air quality affected by fire.

39 Opportunities exist to address susceptible populations (e.g., people with diabetes, older
40 workers) exposed to wildland fire. Initial neurological studies suggest sensitivity to
41 exposure without overt pulmonary effects, so additional studies would help improve

1 understanding. Very little gestational research and subsequent generational studies
2 following wildfire or surrogate wood smoke/biomass exposure are available. While some
3 limited research for the pulmonary and cardiovascular toxicity of wildfire smoke can be
4 found, significant knowledge gaps exist. These gaps include (1) information on how
5 different mixtures and occupational co-exposures affect pulmonary injury and function, (2)
6 pulmonary cancer risk, which may also be affected by co-exposure, (3) the effect of
7 inhalation on microvasculature function, and (4) the identity of altered circulating factors
8 causing systemic effects.

9 Progress has been made in understanding how wildland fire smoke exposures can cause
10 health effects using in vivo models. Additional research is warranted to fully appreciate the
11 potential acute and chronic human health impacts.

12 **3.4 Conclusions on Health Effects of Concern**

13 This section presents information on the health effects associated with exposure to
14 wildland fire smoke from three evidence streams. The first stream includes information
15 available in authoritative weight-of-evidence reviews from global sources. This material
16 serves as the foundation for identifying health effects of concern (Section 3.1). A second
17 evidence stream builds upon this foundation. This stream presents a scoping review from
18 studies on human health effects from wildland fire emissions published since the
19 authoritative reviews (Section 3.2). The third evidence stream includes studies that were
20 reviewed and analyzed to understand the mechanistic nature of how wildland fire
21 emissions might cause health concerns. This third stream examines the biological reasons
22 there may be causal associations between wildland fire emissions and specific health
23 conditions (Section 3.3). Salient findings from each evidence stream are described in
24 Sections 3.4.1, 3.4.2, and 3.4.3. Section 3.4.4 offers a summary and recommendations for
25 future research.

26 **3.4.1 Authoritative Reviews**

27 The first evidence stream comprised evidence from authoritative reviews, including the
28 EPA review of PM in its integrated risk assessment [EPA 2019, 2022] and a comparative
29 study of wildfire and prescribed fire [EPA 2021]; reviews supporting the global air quality
30 guidelines set by the World Health Organization [WHO 2021]; and information on the
31 carcinogenicity of outdoor air pollution [IARC 2015] and firefighting [IARC 2023] found in
32 IARC monographs.

33 Collectively, findings from the group of independent assessments were remarkably
34 consistent despite differences in data and the approach used for making causal
35 determinations. Taken as a whole, there is sufficient evidence of a causal relationship
36 between exposure to PM_{2.5} and cardiovascular effects and nonaccidental mortality. There is
37 also strong evidence of PM carcinogenicity, primarily from the WHO and IARC assessments,
38 although the 2019 PM ISA also found that the relationship between long-term PM_{2.5}
39 exposure and cancer was likely to be causal. The PM ISA also found a likely causal
40 relationship between long-term PM_{2.5} exposure and nervous system effects, as well as short-

1 and long-term PM_{2.5} exposure and respiratory effects, which aligned well with the WHO
2 assessment for its air quality guidelines. There was lesser evidence of other exposure-
3 related effects, such as reproductive, developmental, and metabolic effects.

4 An important limitation to these data is a lack of directly examining health effects among
5 working populations exposed to wildland fire emissions. Most studies reviewed were
6 population-based studies of health effects associated with ambient air pollution from all
7 sources, which might have included contributions from wildland fires. An exception was the
8 IARC monograph on cancer in firefighters. Although IARC examined cancers from
9 occupational exposures in firefighting, the available information was inadequate to examine
10 wildland firefighters separately. Therefore, the degree to which exposure hazards from
11 wildland fires are represented by the IARC monograph is uncertain. Another limitation
12 common among these reviews is the lack of data on protracted exposures and late onset or
13 persistent nonmalignant health effects (e.g., COPD, neurologic disorders).

14 Finally, it is noted that the authoritative reviews have focused mostly on health effects
15 associated with PM, primarily PM_{2.5}, as the largest threat to public health. This hazard
16 review also considers PM_{2.5} to be the primary agent of concern for wildland fire smoke. This
17 is based largely on the authoritative reviews and complementary evidence from toxicology,
18 epidemiology, and exposure science studies presented in the scoping review and elsewhere,
19 as discussed below. Nonetheless, outdoor workers may also be exposed to other agents and
20 size fractions of PM from wildland fire smoke, either as primary emissions or the formation
21 of secondary particles in the atmosphere. Furthermore, interactions with other hazards
22 unrelated to wildland fires (e.g., strenuous activity, work stress, lifestyle factors) that the
23 affected workforce also experiences are not clear. The roles played by these other agents
24 and experiences in occupational health are largely uncertain relative to our knowledge on
25 PM_{2.5} and should be further investigated.

26 **3.4.2 Additional Epidemiological Evidence** 27 **from the Scoping Review**

28 The scoping review searched the relevant English-language literature for studies published
29 from January 2017 through February 2024 that (1) became available after recent
30 authoritative reviews [EPA 2019, 2021, 2022; IARC 2015, 2023; WHO 2021], and (2)
31 specifically assessed physical health effects associated with wildland fire exposures.

32 The review assessed the potential to extend the evidence base set by authoritative reviews.
33 The review did not include a formal weight-of-evidence assessment or quality evaluation;
34 however, it identified studies that overall were relevant and aligned with the conclusions in
35 the authoritative reviews, which serve as an appropriate basis for hazard identification per
36 NIOSH guidance [NIOSH 2020].

37 The scoping review found 138 observational studies that tested for an association between
38 exposure and physical health outcomes, as well as 37 health impact assessments that
39 estimated the health burden associated with exposure. Health effects analyzed included
40 cardiorespiratory, reproductive and developmental, cancer, neurological, metabolic,

1 infectious diseases, sub-clinical changes, nonaccidental mortality, and other outcomes. Most
2 studies evaluated health effects in the general population. Among studies of occupational
3 populations, nearly all investigated firefighters. Most studies assessed PM exposures,
4 primarily PM_{2.5}.

5 The studied health effects largely mirrored those in authoritative reviews; thereby
6 augmenting existing information and providing opportunity for contrasting effects from
7 ambient air pollution with those from wildland or prescribed fires. Overall, findings on the
8 association between wildland fire PM and physical health effects appeared reasonably
9 consistent with those found in the literature on ambient air pollution. For example, the
10 scoping review revealed consistent evidence supporting associations between short-term
11 exposure to PM_{2.5}, defined as durations of hours up to a month, from wildland fire smoke
12 and acute adverse cardiorespiratory effects.

13 In contrast, limited research existed on the effects of long-term exposure (durations >1
14 month to years), late onset (accounting for disease latency) or persistent health effects, and
15 some chronic health conditions (e.g., cerebrovascular disease, metabolic syndrome,
16 neurological outcomes, and cancer) that were assessed in the EPA PM ISA. For example,
17 exposure-related malignant solid tumors are often associated with long latency periods
18 lasting years to decades in studies involving other occupational settings [NIOSH 2020].

19 Several analyses focused on a broad range of reproductive and developmental outcomes;
20 however, consistent with the PM ISA, evidence was somewhat limited or inconsistent. The
21 scoping review provided new information on an association between exposure and
22 infectious disease, COVID-19 in particular, suggesting an emerging field of research not
23 evaluated in the PM ISA.

24 Although emerging evidence of occupational health risks among wildland firefighters has
25 been published, the scoping review revealed little information on other working
26 populations, such as workers in agriculture, forestry, and construction. However, wildland
27 fire smoke exposure is a known hazard encountered by these workers, in addition to
28 multiple other hazards faced on the job [NIOSH 2015, 2023].

29 The lack of occupational studies in the available literature is a strong limitation. Other
30 limitations common to observational studies include the following: (1) the use of
31 aggregated exposure and health outcome measures (e.g., ecologic designs), (2) a potential
32 for residual confounding from unmeasured risk factors, (3) effect sizes that are relatively
33 small or imprecise and prone to bias, and (4) a lack of information on temporal factors (e.g.,
34 disease latency or persistence) and effect modification by age, sex, race, or other factors.

35 **3.4.3 In Vitro and In Vivo Evidence**

36 Studies have shown that several factors can affect the chemical composition of smoke,
37 including the material that is burning and the combustion phase in which the smoke was
38 generated [Cascio 2018; Kim et al. 2018]. Differences in chemical composition of smoke
39 have been shown to cause different biological responses. In vitro toxicity studies have
40 revealed several possible mechanisms by which smoke exposure can affect the

1 cardiovascular and other biological systems. These mechanisms fall into three general
2 categories: oxidative stress, autonomic system dysfunction, and translocation of toxic
3 particles through the circulatory system [Newby et al. 2015; Stone et al. 2017].

4 While in vitro exposures are only models to represent real-world smoke exposure, evidence
5 from these studies appears coherent with the health effects observed in epidemiological
6 studies of persons exposed to wildland fire emissions, including increased mutagenicity,
7 inflammation, and oxidative stress after smoke exposure. Furthermore, several in vitro
8 toxicity studies have shown evidence of cellular injury or alterations to pathways that can
9 lead to the development or aggravation of various respiratory diseases (see Sections 3.3.2
10 and 3.3.3 for further detail on in vitro studies).

11 Results from in vivo toxicity studies further confirmed that overall toxicity and mechanisms
12 of toxicity are determined by smoke composition, PM source, and combustion phase. Most
13 of the literature on in vivo effects from exposure showed evidence consistent with that of
14 general PM studies on pulmonary effects. These included increased inflammation and
15 oxidative stress and cardiovascular effects such as acute autonomic dysfunction and
16 systemic inflammation-mediated cardiovascular dysfunction.

17 Several studies have also shown evidence of smoke exposure as a sensitizer for
18 cardiopulmonary and circulatory conditions in which prolonged exposure could lead to
19 progression of diseased states. Initial in vivo studies on the neurological effects of smoke
20 exposure have shown increases in neuroinflammation, altered phenotype of
21 cerebrovascular cells, and changes in markers consistent with disease related pathologies.
22 These effects were persistent at post-exposure time points, indicating the potential for long-
23 term neurological effects (see Section 3.3.4 for further detail on in vivo studies). In vivo
24 studies with very limited designs related specifically to wildland fire exposure include
25 pregnancy, metabolic disease, and cancer. While much of the toxicological evidence focused
26 on acute effects, there were a few studies that investigated chronic effects.

27 Overall, the in vivo and in vitro data for toxicity resulting from smoke exposure focused
28 primarily on acute effects of the pulmonary and cardiovascular systems. Further research is
29 needed to understand the mechanistic effects in other systems and the differential toxicity
30 of smoke variants. The development of a “smoke library” would be beneficial in determining
31 the toxicological effects that may take place after a certain type of smoke exposure.

32 **3.4.4 Summary**

33 The body of evidence from recent studies on health effects of exposure to wildland fire
34 smoke was consistent with that on ambient air pollution and particulate matter previously
35 reviewed by WHO, IARC, and EPA [EPA 2019, 2022; IARC 2015; WHO 2021],
36 notwithstanding notable size differences between evidence bases.

37 The evidence in authoritative reviews was strongest for cardiovascular effects, cancer, and
38 nonaccidental mortality, followed by respiratory and neurological effects, then other
39 outcomes. Likewise, the scoping review identified evidence linking PM exposures to various
40 health outcomes: respiratory, cardiovascular, reproductive and developmental, metabolic,

1 and neurological effects, as well as cancer and mortality. These findings were generally
2 supported by the toxicological studies reviewed in Section 3.3. For example, evidence for
3 acute cardiorespiratory outcomes associated with short-term inhalation exposure was well-
4 supported by the conclusions in the authoritative reviews, the scoping review on recent
5 epidemiological studies, and the toxicological evidence base.

6 While there were fewer studies on cancer and other chronic diseases associated with long-
7 term exposure included in the scoping review, mechanistic evidence (e.g., oxidative stress,
8 inflammation, DNA changes) from limited epidemiological studies in the scoping review
9 and, to a larger extent, in vitro and in vivo toxicological research, generally supported the
10 biological plausibility of causal relationships identified in authoritative reviews. Similarly,
11 the evidence for adverse reproductive outcomes from in vivo toxicological studies augments
12 the limited evidence contributed by epidemiological studies in the scoping review.
13 Moreover, the scoping review identified studies presenting emerging evidence of
14 associations between wildland fire smoke and infectious disease, which was not discussed
15 in the authoritative reviews.

16 Exposures in recent epidemiological and toxicological studies were often based on
17 measured or estimated levels of PM, primarily PM_{2.5}. Some epidemiological studies
18 attempted to restrict the source of PM_{2.5} to a fire source by eliminating background sources
19 of PM_{2.5} while others used all-source PM_{2.5} (i.e., not specific to a fire source) measured at the
20 same time as a fire event. Few examined other agents.

21 Comparisons between exposure sources were scarce; however, a few toxicological studies
22 contrasted other sources of air pollution PM with wildland fire PM and found toxicity
23 generated by wildland fire PM to be potentially greater on an equal mass basis. Therefore,
24 while the health impacts of PM generally overlap with wildland fire-specific PM, these
25 studies imply a possible increased severity of health impacts. Additional evidence is needed
26 to confirm such conclusions, as outdoor workers might be exposed to higher levels of
27 toxicants from wildland fire sources (during a wildland fire event), and their per-unit
28 inhalation exposure might be more toxic, when compared with ambient air pollution
29 sources.

30 In summary, the authoritative reviews in Section 3.1 provided sufficient evidence of causal,
31 or likely to be causal, relationships between PM exposure and select cardiorespiratory
32 effects, neurological effects, cancer, and nonaccidental mortality [EPA 2019, 2021, 2022;
33 IARC 2015, 2023; WHO 2021]. The exposure hazards assessed, and study conclusions
34 described in Sections 3.2 and 3.3 were reasonably analogous to those assessed in the
35 authoritative reviews. Thus, coherence exists between the hazards described in the
36 authoritative reviews and the hazards anticipated from occupational exposure to wildland
37 fire smoke.

38 However, uncertainty remains in whether the risk among populations exposed to ambient
39 air pollution (or as firefighters) mirrors the risk experienced by working populations
40 exposed to wildland fire smoke. Given few occupational studies and varied exposure
41 potentials, multiple areas remain for continued epidemiological and toxicological research,

1 which are discussed in Sections 3.2 and 3.3, respectively, as well as briefly described in
2 Chapter 6.

3

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1

Chapter 4: Exposure Assessment

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Key Chapter Takeaways

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- Wildland fire smoke contains a wide variety of airborne contaminants; PM_{2.5} is the primary hazard of concern and is used as an indicator for wildland fire smoke exposure and risk management.
- Classifying workers into similar exposure groups (SEGs) enhances the efficiency of exposure assessments by accounting for the various factors affecting exposures. This includes the smoke source, weather conditions, geographical location, duration of exposure, job tasks and work environment.
- NIOSH created exposure control categories (ECCs) that align with U.S. Environmental Protection Agency (EPA) Air Quality Index (AQI) classifications to provide a framework for specific recommended actions, decisions, communications, and mitigation strategies (discussed in Chapter 5).
- A tiered exposure assessment approach is used to evaluate and manage risk, incorporating tools such as monitors, models, sensors, and sampling methods.
- Essential elements needed for an effective sampling strategy are discussed.

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A traditional approach to exposure assessment has been defined as “the process of estimating or measuring the magnitude, frequency, and duration of exposure to an agent, along with the number and characteristics of the population exposed” [NIOSH 2020]. However, outdoor workers exposed to wildland fire smoke face a unique scenario where the exposure frequency is uncertain, the composition of the contaminants is variable, and the duration and magnitude of exposure continue to change. These factors make exposure assessment extremely challenging.

1 This chapter provides an approach for estimating wildland fire smoke exposures to outdoor
2 workers. The approach begins with defining the purpose and considerations of the
3 exposure assessment. Then, the approach covers the tools and their associated tradeoffs
4 available to measure exposures and concludes with a discussion of the treatment of data for
5 decision making. This approach is based on knowledge and experience from conducting
6 routine monitoring of worker exposures to hazards in the workplace. It also provides the
7 basis for further study on how best to perform exposure assessments through the
8 optimization of available information and resources.

9 **4.1 Purpose and Objective of an Exposure Assessment**

10 A critical step in conducting an exposure assessment is to define the purpose and objective
11 of the assessment. For wildland fire smoke, an exposure assessment may be necessary to:

- 12 • Identify which hazards exist.
- 13 • Communicate hazards.
- 14 • Identify the type of control needed to mitigate exposure (e.g., engineering controls
15 vs. administrative measures vs. the use of personal protective equipment).
- 16 • Identify worker groups to prioritize for exposure monitoring.
- 17 • Identify worker groups to place under medical surveillance.
- 18 • Perform emergency response planning.

19
20 Identifying the purpose of the assessment is important to determine the type of exposure
21 information needed and the confidence associated with those measures.

22 When estimates of exposure are obtained during routine monitoring of worker exposures to
23 hazards in the workplace, they are compared against an occupational exposure level of
24 interest (often occupational exposure limits). By comparing these values, the assessment
25 can determine whether workers are exposed to an elevated exposure level of concern. This
26 type of assessment is used to characterize worker safety and exposure in order to
27 understand key risk mitigation strategies and efforts. An acceptable exposure group is one
28 where the 95th percentile of the exposure distribution, with an appropriate level of
29 confidence (e.g., 70% confidence), for a reasonably homogeneous group of workers is less
30 than the single shift exposure level of interest. More precisely, if the 70% upper confidence
31 level of the 95th percentile is less than the exposure level of interest, then the exposure is
32 deemed acceptable. An unacceptable exposure group is one where this is not true [Jahn et
33 al. 2015].

34 This type of approach should be considered when developing a wildland fire smoke
35 exposure assessment; however, the complexity of the problem makes this challenging.

36 **4.2 Exposure Assessment Assumptions**

37 The following assumptions should be considered in the development of an assessment
38 strategy for wildland fire smoke exposure:

1 **Inhalation as the Primary Exposure Route:** Workers could be exposed to substances
2 produced during wildland fires via inhalation and dermal routes (and sometimes ingestion).
3 However, since inhalation is the primary exposure route, this chapter focuses on assessing
4 this route of exposure.

5 **PM_{2.5} as the Primary Indicator of Exposure:** As described in Chapter 2, wildland fire
6 smoke contains a wide variety of airborne contaminants. It contains ultrafine particles
7 (particles with a diameter less than 100 nm or 0.1 μm), fine particulate matter (PM_{2.5},
8 particles smaller than 2.5 μm diameter), coarse particle matter (PM₁₀, particles smaller than
9 10 μm diameter) and its constituents such as elemental and organic carbon. As stated in
10 Chapter 3, in the United States, fires make up about one-third of ambient particulate matter
11 (PM) contributions [EPA 2019] and PM_{2.5} is the primary component of concern regarding
12 adverse health outcomes. PM_{2.5} has been shown to be a reliable marker of smoke exposure
13 from wildland fires. PM_{2.5} is a criteria air pollutant according to National Ambient Air
14 Quality Standards (NAAQS) set by the EPA. PM_{2.5} is routinely monitored by air quality
15 networks and can also be estimated using satellite data, and chemical transport models,
16 providing spatial and temporal exposure estimates. The widely available PM_{2.5}
17 concentration estimates make it a reliable surrogate for estimating exposures and serving
18 as an indicator for exposure to wildland fire smoke to outdoor workers. PM_{2.5} is not
19 generally considered an occupational exposure metric [Vincent 2012]. Instead, respirable,
20 thoracic, and inhalable particles are commonly used occupational exposure metrics [ACGIH
21 1999]. See Chapter 2 (Section 2.1.2.1, Particle Size-Selective Criteria and Standards) and
22 Figure 2–5 for discussion of the applicable particle penetration curves.

23 **Presence of Other Sources of Particulate Matter Exposure:** Additional sources of PM_{2.5}
24 exposure include a variety of natural and anthropogenic influences and behaviors such as
25 (combustion based) road and industrial emissions, energy transformation and extraction,
26 surface transportation mechanisms, other agricultural activities, and residential and
27 commercial-level waste disposal and handling. These sources persist throughout the
28 calendar year, regardless of proximity to a wildfire. Work-related processes in various
29 industries and sectors can also generate PM exposure.

30 **4.3 Considerations for an Exposure Assessment**

31 Although various exposure assessment approaches and tools are available to estimate
32 population exposures, a tiered approach is an effective decision-making scheme commonly
33 used in exposure assessment to evaluate complex exposure scenarios. It allows for easy
34 decision-making that builds on previous decisions [Brouwer et al. 2011]. The initial tier
35 uses simple, easily available tools or data on how to assess exposures and make decisions
36 on whether to stop the exposure assessment or decide on exposure mitigation and further
37 exposure assessment. Each successive tier uses more complex exposure assessment tools or
38 data to obtain more valid, precise, and detailed exposure estimates to make risk
39 management decisions. This assessment strategy for outdoor worker exposure to wildland
40 fire smoke uses a three-tiered approach. This approach involves assessing exposures and

1 making decisions about continued exposure assessment, determining acceptability of
2 exposures, and implementing measures to mitigate exposure risks.

3 **Tier 1 assessments** use readily available data from models, satellite sensors, community
4 monitors, and consumer (low-cost) sensors to quickly identify the potential for wildland
5 fire smoke and any exposure mitigation actions needed, or whether to progress to the next
6 tier of workplace exposure assessment (see Section 4.4.1). Although Tier 1 data may be less
7 accurate or specific, the decision criterion to progress to a higher tier is not stringent (e.g.,
8 measurements exceeding Exposure Category 1, described in the next section) for additional
9 confidence in exposure assignment. Alternatively, reliance on Tier 1 levels may result in
10 more conservative risk management but can be used for initial decision-making.

11 **Tier 2 assessments** use direct-reading instruments (DRIs) to assess workplace exposures
12 to wildland fire smoke and other workplace-generated exposures. These tools can quickly
13 identify exposures to workers so that risk can be managed. Although DRIs cannot
14 selectively identify wildland fire smoke constituents, they can identify the amount of
15 particulate exposure to workers, regardless of source. DRIs enable quick action to protect
16 workers from particulate exposure, which constitutes the biggest exposure risk to wildland
17 fire smoke. Tier 2 assessments improve confidence in the amount and variability of
18 exposure and include all sources of PM. Finally, Tier 2 assessments provide valuable
19 information for Tier 3 assessments.

20 **Tier 3 assessments** use specific sampling and analytical methods to characterize distinct
21 wildland fire smoke constituents. These types of methods can provide additional detail
22 regarding particulate matter exposure and can identify additional risks to workers that may
23 result from gases, volatile organic compounds (VOCs), and non-volatile/semi-volatile
24 chemical exposures. This is useful because Tier 2-based mitigation action may not be
25 adequate in these cases and refinement may be needed. Thus, a more complete mitigation
26 strategy may be developed in Tier 3.

27 Such a tiered approach used within the framework of an exposure assessment strategy
28 (described in Section 4.4) can be a pragmatic, effective, and efficient approach to protect
29 workers by assessing their exposure risk to wildland fire smoke. Note that the tools and
30 data from Tier 1 and Tier 2 assessments have limitations in terms of accuracy and
31 specificity of exposure estimates as described in Section 4.4. However, these tools are fit for
32 purpose and quickly provide data to enable rapid action to protect workers and to inform
33 the following tier. At the same time, efforts can continue towards higher tier assessments to
34 obtain more accurate and specific exposure estimates to refine the initial estimates and
35 validate the control actions taken. The decision on whether to implement exposure
36 mitigation actions based on a particular tier of assessment or proceed to the next tier
37 depends on multiple factors. These factors include the results of the exposure assessment,
38 confidence in the results, accuracy and variability of the results, whether the results are
39 representative of the population (e.g., proximity and plume direction relative to sensors),
40 available resources for mitigation and further assessment, urgency of the mitigation actions
41 required, and other similar factors. Additionally, conservative actions may be taken

1 immediately based on a particular tier of assessment while higher tier assessments are
2 ongoing to confirm the control actions taken or to modify the actions.

3 This exposure assessment strategy includes the collection, statistical analysis, and
4 interpretation of exposure data relative to an exposure level of interest. This concept is
5 defined for wildland fire smoke exposure in Section 4.5.

6 Several factors should be considered in designing exposure assessment strategies. One of
7 the most important is exposure variability. Because exposures vary between workers, over
8 time, shift, and location, the sampling strategy should be effective in capturing these
9 variabilities. At the same time, the strategy must be feasible and efficient in that it should
10 not require an inordinately large number of samples. Because occupational hygienists
11 usually operate with limited resources that preclude large sample sizes, the dual
12 requirements of effectiveness (i.e., the ability to provide correct exposure decisions) and
13 efficiency (i.e., the need to minimize the number of measurements) need to be optimized.







14 Therefore, the exposure assessment strategy in the context of a wildland fire should fulfill
15 the following criteria: (1) effectively determine the workers who are at different levels of
16 risk, (2) identify the appropriate actions needed to mitigate their risks at each level, and (3)
17 achieve these goals as efficiently and promptly as possible.

18 **4.3.1 Exposure Control Categories**

19 For wildland fire smoke exposure, NIOSH created ECCs that align with the EPA AQI
20 classification scheme and are consistent with the anticipated health effects from exposure.
21 For each ECC, specific recommended actions, decisions, communications, and mitigation
22 strategies are employed (see Chapter 5). These actions should be decided by an employer
23 with recommendations from an occupational hygienist in advance of wildland fire smoke
24 events.

25 The air quality PM concentration data (PM_{2.5}) can be categorized into air quality indices
26 based on likely health effects, populations, and sub-populations. EPA created a color-coded
27 tool with AQI groupings associated with exposure levels. These can be applied as exposure
28 control category ratings as outlined in Table 4–1 below.

Table 4–1. Exposure control categories by Air Quality Index groupings

Air Quality Index (AQI)*	PM _{2.5} (µg/m ³)*	Exposure control category (ECC)†
 Good (0–50)	0.0 to 9.0	1
 Moderate (51–100)	9.1 to 35.4	2
 Unhealthy for sensitive groups (101–150)	35.5 to 55.4	3
 Unhealthy (151–200)	55.5 to 125.4	4
 Very unhealthy (201–300)	125.5 to 225.4	5
 Hazardous (301+)	225.5+	6

* Established by the EPA (discussed further in Section 5.2.1).

† Recommended by NIOSH (discussed further in Section 5.2.1).

Workers at a worksite may not perform the same tasks or experience the same exposure levels. They may be at different distances from the wildfire source, upwind or downwind of a fire, or on different sides of a hill with a wildfire. Each situation requires different levels or types of exposure management and mitigation. Therefore, it is important to classify the workers into groups based on their similarity of exposures. Section 4.3.2 describes the process of assigning workers to similar exposed groups (SEGs).

4.3.2 Creation of Similar Exposure Groups

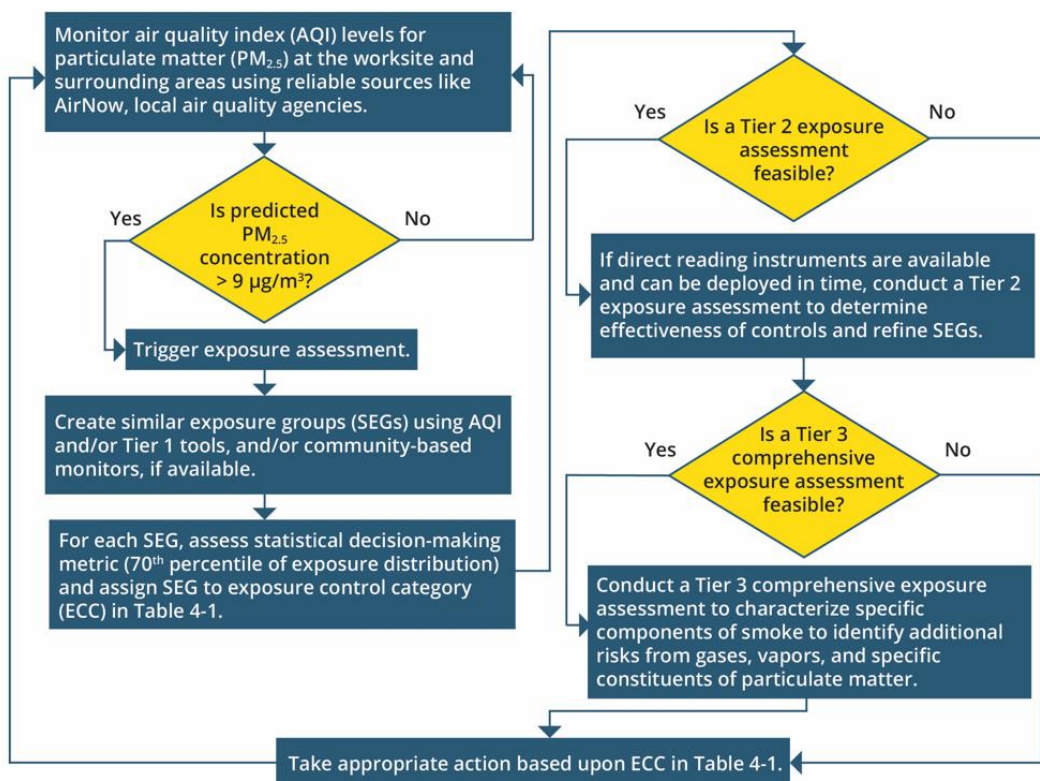
Since assessing the exposure of every worker in the workplace can require significant time and resources, common practice is to classify workers into SEGs and then randomly sample a subset of workers within each SEG. This facilitates the efficient exposure assessment of a large workforce. To classify workers into SEGs, it is assumed that workers have similar exposure distributions. This means that each worker’s exposure profile is very similar to every other worker in the SEG. Workers are grouped or assigned to “exposure zones” based on their work similarities. These include job task profiles, the presence of chemicals and other hazardous agents, and environmental similarity (e.g., ventilation characteristics and processes). In the context of wildland fire smoke exposures, SEGs could be created based on whether groups of workers are upwind or downwind of the source, geographical location (e.g., on different sides of a hill or distance from the source), and the degree to which the immediate terrain is forested. Outside workers might also work mostly in enclosed environments, such as cabs of vehicles, or they may work completely outside.

Physical and chemical exposure modeling or air pollution dispersion modeling techniques can also be used to develop exposure predictions (see Section 4.4.1.1). Physical and chemical exposure models are commonly used in occupational exposure assessment to estimate exposures based on the underlying processes that generate exposures [Keil et al. 2009]. Thus, SEGs are established by observing proximity to the source, task, and expected

1 exposure to PM_{2.5} using mathematical models and monitoring of airborne concentrations of
2 PM_{2.5}. Given the dynamic nature of a wildland fire, it is important to acknowledge that these
3 SEGs could also rapidly change over time. SEGs used for making decisions to mitigate risks
4 might differ from a post-facto analysis of the risks faced by different types of workers. This
5 might drive other decisions such as the need for medical surveillance.

6 Figure 4–1 (below) shows an approach to developing an exposure assessment strategy to
7 assess and manage outdoor workers’ exposures to PM_{2.5} from wildland fire smoke during
8 wildland fires.

9 4.3.3 Exposure Assessment Guidance



10
11 **Figure 4–1. An example strategy for exposure assessment supporting risk**
12 **management decisions**

13 The AQI for PM_{2.5} concentrations at or near worksites should be continually monitored in
14 geographical regions prone to wildland fires, and during wildfire season.

15 Action levels should be established based on AQI or PM_{2.5} for implementing protective
16 measures. Although PM comes from a variety of sources and should be regularly monitored
17 for elevated concentrations, Figure 4–1 indicates that if the predicted PM_{2.5} levels are in
18 ECC 2 or above, then an exposure assessment should be initiated. Predicted levels are either

1 current information from AQI values or community monitors, or they could be forecasted
2 levels from various models in Tier 1, if available. The trigger is used as an example, and
3 different worksites may have different levels decided by the employer in consultation with
4 a health and safety expert.

5 Once an exposure assessment is initiated, SEGs will be created based on a combination of
6 AQI predictions, Tier 1 tools, and community-based monitors (see Section 4.4.1.3). Using
7 the available data, estimate the exposure distribution of PM_{2.5} for workers in each SEG (see
8 Section 4.5). If, for instance, only an AQI value is available for the geographical region of the
9 wildfire, then the predicted PM_{2.5} level should be used to estimate the 95th percentile (X95).
10 If a more local community monitor or Tier 1 model prediction is available, then it will be
11 used to arrive at an estimate of X95. This X95 estimate is compared with the exposure level
12 of interest and assigned using the appropriate ECC in Table 4–1. For improved and more
13 spatially granular confidence, a Tier 2 assessment measuring PM_{2.5} or respirable dust using
14 field-deployable DRIs (see Section 4.4.2) should be used to further refine the exposure
15 estimates and ECC assignments. If this is not feasible, then the ECC assignments using a
16 combination of AQI value, Tier 1 tools, and community-based measurements (or just one of
17 these) become the final assignments. The ECC assignments correspond to appropriate
18 recommended mitigation actions to be taken (see Chapter 5).

19 More detailed Tier 2 and Tier 3 measurements can be obtained to improve confidence and
20 granularity in exposure estimates, as well as to assess the effectiveness of various control
21 measures, further refining SEGs. They can also occur concurrently depending upon the type
22 of information desired and the availability and familiarity with sampling equipment. Tier 3
23 measurements use comprehensive and well-established indirect air sampling practices and
24 produce contaminant specific, but not immediate, results. Additional information regarding
25 Tier 2 and Tier 3 measurements, tools, and limitations can be found in Section 4.4.

26 Table 4–2 below outlines how the strategy may be implemented at different Tiers based on
27 the source or type of data available, the relevant exposure metrics to calculate, the test to be
28 conducted to determine if action is needed, and finally the actions to be taken. It also
29 provides information on how to decide to proceed to subsequent Tiers.

1 **Table 4–2. Implementation strategies at different tiers based on assessment tools**

Assessment tool	Test	Action
AQI value	Is AQI (X95 score) > 50 (i.e., in ECC 2 or greater)? Do AQI hourly averages on a day indicate peak exposures that are different from other days' patterns?	If SEG = ECC 1, no action needed. Continue to check local area monitors. If low confidence in ECC 1 determination, proceed to Tier 2 assessment.
PM _{2.5} concentration*	Is PM _{2.5} (X95 estimate) > 9 µg/m ³ (i.e., in ECC 2 or greater)? Do PM _{2.5} hourly averages on a day indicate peak exposures that are different from other days' patterns?	If SEG = ECC 2 or higher, take control action (see Chapter 5) to immediately protect workers. If low confidence in ECC level determination, also proceed to Tier 2 assessment if feasible.
GM, GSD, full-shift or short-duration concentration from respirable dust or fine particle monitors	Is PM _{2.5} (X95 estimate) > 9 µg/m ³ (i.e., in ECC 2 or greater)? Do short-duration (15-min moving averages) of PM _{2.5} concentrations on a day indicate peak exposures that are different from other days' patterns?	Refine PM _{2.5} X95 of SEGs. Determine patterns for short duration exposures. Confirm or modify ECC and related control action. Decide whether to proceed to Tier 3.
Exposure sampling and analytical methods	Identify the exposure risk category (e.g., AIHA) of the X95 for specific gases, particles, VOCs, and semi-volatiles.	Match exposure risk categories (e.g., AIHA) to the ECC or OEL to determine action.

2 Abbreviations: AIHA, American Industrial Hygiene Association; AQI, air quality index; AQS, Air Quality
3 System; ECC, exposure control category; GM, geometric mean; GSD, geometric standard deviation; OEL,
4 occupational exposure limit; PM_{2.5}, particulate matter with a nominal mean aerodynamic diameter less
5 than or equal to 2.5 µm; SEG, similar exposure group; VOC, volatile organic compound; X95, 95th
6 percentile of exposure distribution.

7 * Refer to Table 4-3 for Data Sources.

8

4.4 Tools for Defining Exposure

A long-standing NIOSH assumption remains that personal samples obtained from a worker's breathing zone provide the most accurate representation of the worker's inhalation exposure [Vincent 2007]. This is because the concentration measured by the personal sampler is close to the concentration of the pollutant being inhaled by the worker. However, in exposure scenarios where timely action is important, the deployment of personal samplers may not be feasible. When personal measurements are unavailable, estimates of AQI and PM_{2.5} obtained from federal monitoring networks, area samplers placed near wildfire locations, as well as predictions by models can be used. The following sections explain the various approaches to consider when developing an exposure assessment strategy. They also describe the modeling and measurement tools used to execute the strategy.

4.4.1 Tier 1

4.4.1.1 Models

Several databases provide the necessary information to compute population-level exposure to smoke from wildland fires. They can also assess fire impacts and develop fire mitigation plans (see Table 4-3). These tools consider and evaluate a complex range of variables including fuel type, fuel load, fire behavior and smoke dispersion mechanisms, plume size, and the fire source. Wildland fire modeling tools also assess geographical terrain and surrounding weather variables. Thus, wildland fire and smoke models are often defined as numerical tools that provide current or projected information on intensity, pollutant type or concentrations, spatial information, and temporal details [Liu et al. 2019].

Table 4–3. Data sources available for assessing smoke exposure in the United States

Regulatory monitors and low-cost sensors				
Data source	Time period	Spatial resolution	Temporal resolution	Parameters
AirNow.gov National Maps	1980–present	Monitors	Hourly basis	NAAQS* framework Air Quality Index Fire and smoke plume details
EPA Air Quality System	1980–present	Monitors	Hourly basis	NAAQS framework
Purple Air	2016–present	Monitors	Hourly basis	PM _{2.5} PM ₁₀ O ₃
Fire and smoke information from satellites				
NOAA Hazard Mapping System—Fire and Smoke Products[†]	2008–present	Plume density polygons	N/A	Fire and smoke PM _{2.5} [‡]
NOAA Satellite and Smoke Text Product[†]	2008–present	Text Product	2x/day	Fire and smoke description
Smoke and air quality information from models				
Weather.gov Air Quality Forecast Guidance[§]	2016–present	12×12 KM, gridded	Hourly basis	PM _{2.5} Smoke and dust O ₃
Public health modeling systems				
CDC National Environmental Public Health Tracking Network	2009–present	Monitors	Every 4 hours	Public health Frameworks [#]

* All criteria pollutants regulated under the National Ambient Air Quality Standards including PM_{2.5}, PM₁₀, O₃, NO₂, SO₂, and Pb.

[†] Product designed and regulated by the National Oceanic and Atmospheric Administration (NOAA).

[‡] Algorithms are used to approximate PM_{2.5} concentration values and plume density information.

[§] Product designed and regulated by the National Weather Service and NOAA.

^{||} Network applies mapping tools, data visualization, environmental conditions, community health profiles, and citizen science applications. This tool was designed and is regulated by the CDC [Vaidyanathan et al. 2018].

[#] This network works to track and connect 24 public health frameworks from four overarching subject areas including environment, exposures, health effects, and population characteristics (see Appendix B).

Modeling approaches are commonly used to understand the complex and interconnected relationships among the driving factors of wildland fires [Oliveira et al. 2021]. These approaches are also used to perform risk assessments [Zou et al. 2023]. Due to various

1 complex factors [Sayad et al. 2019], modeling systems must also anticipate uncertain
2 wildfire behaviors (e.g., temperature, humidity, wind direction, atmospheric instability).

3 However, these modeling systems only provide limited information on potential outdoor
4 worker exposures or risk in the nearby or distant working environments. When using
5 modeling tools, note that current modeling and forecasting tools may only provide a crude
6 estimate of occupational safety and health (OSH) risks when studying exposure to PM_{2.5} or
7 other wildland fire-related pollutants. Consequently, combining environmental, community-
8 based, and OSH factors into future wildland fire smoke predictive modeling could be a
9 powerful tool to help protect agricultural and other outdoor workers near or distant to
10 these working environments.

11 Another limitation of these tools is the challenge of providing timely and accurate wildland
12 fire smoke and pollutant forecasts. These forecasts are becoming increasingly necessary as
13 the wildland fire season lengthens and the severity of fires grows. Some modeling tools that
14 characterize smoke emissions or plumes use trend assumptions that may be inaccurate for
15 specific situations. For example, these models may assume that as temperatures rise
16 throughout the day, more intense burning and wildfire movement occurs in the afternoon,
17 with much less during the morning or overnight hours [O'Neill and Raffuse 2021]. However,
18 this assumption does not always accurately forecast emissions. Not every wildland fire
19 follows the same burn or smoke dispersion profile. This is especially critical as wildland fire
20 fuel loads and vegetation landscapes are constantly changing and developing. In addition,
21 the type of terrain, climate, fuel load, as well as any preventive measures in place, all
22 interact with the smoke intensity and makeup in the air. These characteristics all play a role
23 in determining the smoke pollution and health-related size fraction particulates in the
24 nearby and distant working environments. Thus, accurate and timely modeling systems that
25 track the current state of a wildfire, its fuel sources and combustion, and the associated
26 smoke dispersion, are crucial for obtaining relevant information to evaluate OSH risks and
27 impacts to outdoor workers.

28 **4.4.1.2 Satellite Remote Sensing Systems**

29 Remote satellite sensing systems are another way to predict or model wildland fire and
30 smoke. These systems are commonly equipped with thermal and optical sensors to identify
31 the many physical characteristics of a wildland fire, including the associated smoke [Ghali
32 and Akhloufi 2023]. Further, satellite-based modeling systems can provide smoke plume
33 development and fire progression details. Given the known health impacts of wildland fire
34 smoke, satellite sensing systems can provide critical information on the movement and
35 physical characteristics of the smoke. Geographically, satellite sensing systems are useful
36 for detecting and monitoring wildland fires, smoke plumes, and behaviors in remote or
37 rural areas, or vastly forested territories [Shadrin et al. 2024]. Satellite sensing systems and
38 modeling could also be critical in these area types when DRIs, personal sampling, or
39 environmental- and community-based efforts aren't available. For example, public health
40 specialists can use satellite tools and agents to evaluate ignition probabilities and fire smoke
41 propagation patterns to anticipate and potentially prevent exposures to wildland fire smoke
42 and its agents. Furthermore, satellite-based modeling allows for observational review of

1 geographical and territorial characteristic changes over time and physical space, which can
2 be helpful when deciphering public health mitigation strategies [Oliveira et al. 2021].

3 In contrast, satellite sensing tools may provide challenges such as underestimation of fire
4 activity and spread, as well as spatial and temporal resolution issues. For example, it is
5 difficult to maintain a high-level spatial and temporal resolution system, because those with
6 a high spatial resolution typically monitor smaller areas. As a result, it requires more time
7 for satellite sensing and modeling tools with an overall smaller field of vision (or resolution)
8 to cover a larger spatial area. Satellite sensing and modeling systems also require technical
9 expertise and are often computationally expensive. Therefore, using these systems may
10 provide obstacles to rapid response or operationally-robust environments. In particular,
11 obstacles may occur when expeditious data analysis or emergency response and decision-
12 making is required.

13 **4.4.1.3 Community-Based Monitors**

14 Community-based monitors are sited outdoors as area samplers. They provide air quality
15 assessments of the air surrounding the monitor and exposures to community-level
16 populations. The monitors require minimal investment (e.g., initial cost, maintenance, or
17 training) for outdoor workers or their advocates. However, a method for accessing the
18 publicly available data is required.

19 **Particulate Matter Monitors Meeting Federal Standards**

20 The first category of community-based monitors meets stringent federal reference methods
21 (FRMs) or federal equivalent methods (FEMs) criteria to assess compliance with NAAQS. An
22 air sampling inlet for a stationary environmental air sampler is symmetrical around a
23 vertical axis and provides relatively unbiased air sampling from any direction
24 (omnidirectional) and varying wind speeds (up to 10 meters per second). An upper cowl
25 prevents precipitation from entering the sampling train. Internally, a particle size-selective
26 device that uses the inertial properties of the particles in air (aerodynamic diameter)
27 removes particles larger than the size of interest. Two devices are typically employed: an
28 impactor or cyclone, and either may be used in FRMs or FEMs [EPA 2024a]. These devices
29 are carefully calibrated at specified air flow rates. The particle fractions of interest, PM₁₀, or
30 PM_{2.5} (see Figure 2–5) are capable of further penetration into the sampler and the PM mass
31 is either monitored directly for continuous monitors or collected for further analyses.

32 FRMs for PM consist of high-volume air passing particle size-selective devices, and the
33 particulate phase is collected onto clean filter media over a 24-hour period. FRMs require
34 daily maintenance by a skilled technician. Conditioned filter media are pre- and post-
35 weighed under controlled conditions with a gravimetric balance following air sampling. The
36 mean 24-hour PM concentration is determined by the increase in filter mass (μg) divided by
37 the sampled volume (m^3). Following gravimetric weighing, which is non-destructive, filter
38 media may be further subjected to compositional analyses. Federal reference data are not
39 available in real time. Data are available following the completion of sampling and analyses
40 and are used as a quality control for FEMs. Federal reference monitors are not available at
41 every monitoring location, and the number of available monitors is decreasing [EPA 2019].

1 FEMs, or automated equivalent methods for PM, are the continuous monitors sited outdoors
2 at air monitoring networks nationwide. They provide community-based PM_{2.5} and PM₁₀
3 concentrations on much shorter timescales (e.g., every hour or less) and as rolling 24-hour
4 averages. Similar inlets, protection, and size-selective devices are used in the reference
5 methods above, but the mass determination is conducted differently. Equivalent methods
6 use a property closely related to particle mass, but one that may be measured regularly and
7 continuously with periodic maintenance.

8 The first automated approach involves mechanical oscillation. The oscillation frequency of a
9 filter affixed to a tapered tube is directly proportional to its mass. The change in the
10 oscillating frequency can be detected and related to the deposited PM mass. Wearable dust
11 monitors using miniaturized versions of this technology have been developed to monitor
12 exposure to harmful respirable coal dust [MSHA 2014].

13 A second automated approach involves beta particle attenuation. Beta particles (high-speed
14 electrons) may be emitted from a radioactive source (carbon-14 is typical). They may pass
15 through a filter, but as the filter becomes loaded with PM, penetration through the filter is
16 reduced (attenuated). The change in attenuation is detected and related to the deposited
17 PM mass.

18 A third automated approach uses the optical properties of suspended particles. The
19 interaction of particles with light may be influenced by the particle size distribution, particle
20 shape, refractive index, and particle concentration. Incident light may be scattered in the
21 presence of particles. Depending on the device's mode of operation, the amount of light
22 scattered from the source is either measured (photometric mode or scattering volume) or
23 the scattered light is detected (optical particle counting). The angle at which the light is
24 scattered can inform an optical particle size distribution. The particle mass concentration
25 may be inferred from either the scattering volume in photometric mode or the optical
26 particle size distribution. Optical techniques are routinely used in occupational exposure
27 assessment as described in the next assessment tier.

28 The three automated PM measurement approaches (FEMs) above are routinely used across
29 the United States for NAAQS compliance. State-level air quality monitoring systems
30 (meeting federal standards) are also used to detect, report, and summarize levels of PM in
31 the ambient environment. The stringent requirements for attaining PM equivalent method
32 designation result in excellent data quality. These are the most robust and reliable of the
33 community-based measurements. Note that the contribution to PM from wildland fire
34 smoke may not be entirely captured by the PM_{2.5} fraction alone, but the PM_{2.5} fraction is a
35 reliable surrogate for wildfire smoke exposure.

1 **Consumer (Low-Cost) Networked Particulate Matter Monitors**

2 A second category of community-based PM monitors are consumer-based monitors that
3 may be networked together. One popular example is the PurpleAir network, which is free
4 and publicly available on the internet. If an outdoor device is nearby, it can provide useful
5 data. For less than \$300, a monitor can be purchased and installed in a working area as an
6 area sampler that requires power and internet access. Alternatively, an internal clock and
7 micro-SD memory card can allow the device to record and store data for subsequent upload
8 when connectivity is unavailable. However, this may be a significant limitation if air quality
9 data are needed in near real-time for timely decisions regarding workers or their working
10 environment.

11 The devices use an optical measurement based on light or laser scattering and counting as
12 the underlying principles of operation. No accuracy or precision criteria or requirements
13 exist for consumer sensors. At best, data provided are estimated concentrations in that
14 location. In carefully controlled laboratory tests, consumer sensors underestimated PM by
15 up to a factor of ~4 when compared with reference PM masses [Tryner et al. 2020]. The raw
16 collected data need to be processed and adjustments made through algorithms for the
17 consumer monitoring data to be useful [Jaffe et al. 2023]. This is usually accomplished
18 through comparison with a collocated FEM noted above. Once again, connectivity to the
19 internet is required.

20 **Strengths and Limitations for Community-Based Monitors**

21 If available, near real-time data for PM_{2.5} or PM₁₀ from FEMs or consumer (low-cost)
22 monitors is highly advantageous. The monitors require minimal investment (e.g., initial
23 cost, maintenance, or training) for outdoor workers or their advocates. A method for
24 accessing the publicly available data is required. Changes in PM concentration can provide
25 adequate warning of wildland smoke plume proximity and estimated exposure level,
26 informing risk mitigation decisions for outdoor workers. This information can be used to
27 determine whether further exposure assessment is required in Tier 2 or Tier 3 and inform,
28 if feasible, the next tiers. Continuous community-level PM measurements can also provide
29 an estimate of cumulative PM exposure, including work and nonwork-related sources. This
30 could be important if a worker remains in a smoke-impacted location during nonwork
31 hours. A major disadvantage is proximity of the worker to the monitor, as greater distances
32 reduce exposure estimate accuracy. The FEMs located for compliance with NAAQS tend to
33 be clustered in and around urban areas. Coverage in rural locations is sparse, yet these are
34 the areas where both wildland fires and outdoor workers may be present. Wind speed and
35 direction relative to the wildland fire source, the monitoring location, and the worker are
36 also important considerations. Furthermore, community-based monitors may not account
37 for localized or point particle source contributions, such as particulate exposures generated
38 from work duties. Tier 2 (direct reading) or Tier 3 (comprehensive) approaches are better
39 for assessing overall worker exposure and accounting for additional localized particle
40 sources as a potentially mixed exposure.

4.4.2 Tier 2

4.4.2.1 Direct-Reading Instruments

Once the potential exposure of outside workers to particles in wildland fire smoke has been identified and anticipated with the support of Tier 1 monitoring tools, DRIs or sensors can be used for improved exposure estimates. Tier 2 monitoring efforts confirm and build upon the idea that wildland fire smoke particles have entered a specific outdoor workplace environment and environment-specific monitoring is needed [AIHA 2022]. Although DRIs generally do not provide compositional information of the particles monitored, they can provide an excellent estimate of the concentration in the vicinity of workers. Aethalometer instruments might be an exception since they monitor black carbon, which is a small component of wildland fire smoke (see Chapter 2) but not being portable they are rarely used by safety and health professionals in the field. In general, if DRIs are properly positioned and directed, and the data properly interpreted, they can also inform if the particles measured originated onsite or if they traveled from outside the specific workplace, such as the case of wildland fire smoke particles considered for this review.

The primary benefit of using DRIs is their ability to quickly detect and estimate the concentration of particles, such as those from wildland fire smoke, in occupational environments with a granularity that is higher than the Tier 1 tools. This is unlike laboratory sample analysis collected for a period of time, which can be delayed. Although DRIs can generate time series data, this strength should be considered secondary to rapidly assessing the presence and concentration of particles from wildland fire smoke. This capability allows safety and health professionals to receive immediate feedback and make timely risk management decisions. This aspect has been recognized for other hazards, such as gas and vapors for first responders [NIOSH 2012].

Tier 2 assessments begin before getting on a worksite. Professionals involved at this level need to consider the type of wildland fire smoke particles potentially present onsite, the type of workplace and its intrinsic hazards in terms of particles, the selection and preparation of DRIs, and the level of training and preparation needed for using them. These aspects are discussed further in NIOSH's Center for Direct Reading and Sensor Technologies' "Right Sensors Used Right" approach [Cauda and Hoover 2019].

Selection of the proper DRI requires a preliminary understanding of the particle size of interest. As mentioned in Chapter 2, wildland fire smoke particles can evolve in time and based on distance from the fire. For example, the particles can be fresh and ultrafine or evolve into aged particles with a larger size. Based on the preliminary estimation, if wildland fire smoke is primarily composed of nanoparticles and ultrafine particles, condensation particle counters are the appropriate type of DRI to be selected. Conversely, if aged particles are more likely to be present with a super-micron size range, photometers and optical particle counters are preferable.

Condensation particle or condensation nucleus counters detect particles from a few nanometers up to ~1 μm . Various working fluids are employed in practice, consisting of

1 water or alcohols. A saturated vapor from the working fluid condenses onto preexisting
2 particles present in the sampled air. These devices grow particles into liquid droplets of a
3 sufficiently large size to be counted optically. The devices are normally used as area
4 monitors in occupational exposure sampling but have been routinely used in assessing
5 indoor and outdoor worker exposures, including smoke [Evans and Fent 2015; Fent et al.
6 2018]. The measurement provides a particle count rather than a particle mass. A sub-1 μm
7 primary mode was observed in the (volume equivalent) particle size distribution on smoke-
8 impacted days, at the regional scale (see Figure 2-1). It suggests that the particle size range
9 of operation for condensation particle or condensation nucleus counters could be useful in
10 monitoring the presence and level of wildland fire smoke. However, a major limitation is
11 that no equivalent AQIs are available to compare particle number concentrations or counts.
12 A brief overview of the strengths and limitations of the possible DRI tools is provided in
13 Table 4-4.

1 **Table 4–4. Strengths and limitations of direct-reading instrument tools**
 2 **for Tier 2 assessment**

Technology	Strengths	Limitations
Condensation particle counters	<ul style="list-style-type: none"> • Sensitive to the presence of particles smaller than 1 µm, such as fresh wildland fire smoke. • Insensitive to particles larger than 1 µm generated locally in the worksite. • Accuracy of the measurement minimally affected by the chemical and physical properties of the particles. 	<ul style="list-style-type: none"> • Measurements misaligned with the AQI values. • Insensitive to particles larger than 1 µm, such as aged wildland fire smoke particles.
Optical particle counters	<ul style="list-style-type: none"> • Measurements aligned with AQI values. • Sensitive to the presence of particles larger than 0.5 µm, such as aged wildland fire smoke particles. • Marginally sensitive to particles smaller than 0.5 µm. 	<ul style="list-style-type: none"> • Accuracy of the measurement affected by the chemical and physical properties of the particles. • Marginally sensitive to fresh wildland fire smoke particles. • Cross-sensitivity with particles larger than 0.5 µm generated locally in the worksite.
Photometers	<ul style="list-style-type: none"> • Measurements aligned with the AQI values. • Sensitive to the presence of particles larger than 0.5 µm, such as aged wildland fire smoke particles. • Insensitive to particles smaller than 0.5 µm. 	<ul style="list-style-type: none"> • Accuracy of the measurement affected by the chemical and physical properties of the particles. • Insensitive to fresh wildland fire smoke particles. • Cross-sensitivity with particles larger than 0.5 µm generated locally in the worksite.

3 The second aspect to consider are particles generated from other sources, such as vehicular
 4 traffic, combustion products from engines, or typical worker activities. Agriculture sites can
 5 have inhalable and respirable particles [Lee et al. 2006; Rumchev et al. 2019], and
 6 construction sites are dusty environments where respirable dust from several activities is
 7 known and assessed [Flanagan et al. 2003; Linch 2002; NIOSH 2014; Thompson and Qi
 8 2023]. Considering the focus on wildland fire smoke particles and the abovementioned
 9 options of DRIs, additional particles might be considered confounders, and the selection of
 10 DRI should consider their potential presence and incidence. For example, if wildland fire
 11 smoke particles are considered ultrafine particles, the presence of micron-size particles
 12 generated in construction sites should not be an issue when using condensation particle
 13 counters. On the other hand, the presence of aged wildland fire smoke particles and
 14 respirable dust in agriculture sites might constitute a problem when using photometers.

1 The idea is not to avoid using proper DRIs for the particles of interest, but to be aware of the
2 environment.

3 Various limitations need to be considered when using DRIs. They cannot provide
4 information on the chemical composition of wildland fire smoke particles, nor can they
5 selectively monitor particles of a certain chemical composition [Vosburgh et al. 2022].
6 Another limitation is the variable accuracy of DRI tools in monitoring the mass
7 concentration of particles because reporting is based upon the optical and physical
8 characteristics of the particles. This means that, particularly for wildland fire smoke, the
9 absolute value reported in terms of mass concentration can have different degrees of bias
10 [Delp and Singer 2020; NIOSH 2021]. Some DRIs can report mass concentration levels in
11 terms of respirable particles. This feature is important for an occupational exposure
12 assessment perspective [NIOSH 2021], especially when performed with Tier 3 evaluation
13 techniques. Other DRIs report values in terms of PM_{2.5} or PM₁₀, which are environmental
14 measures that align with Tier 1 applications and the overall focus of this exposure
15 assessment strategy. Most importantly, either of these size fractions allow decisions to be
16 made on the spot.

17 Finally, appropriate skills and knowledge are needed to select and operate the DRI tools.
18 The American Industrial Hygiene Association committee on real-time detection systems
19 published a framework on the use of DRIs, describing the responsibilities and expectations
20 at different levels [AIHA 2020]. It is critical to recognize that the selection and use of DRIs is
21 more demanding compared with selection and use of consumer monitors in Tier 1
22 applications.

23 Sufficient time should be allotted to set up the DRIs and perform a minimal quality
24 assessment. This includes conducting a zero calibration and assessing the instruments,
25 generally carried out with small high-efficiency particulate air (HEPA) filters [NIOSH 2021].
26 If only one DRI unit will be used, the initial assessment of the device's performance is
27 limited to a zero calibration and a bump test. A bump test exposes the device to particles in
28 the environment and verifies its ability to monitor the presence of an exposure [Jankovic et
29 al. 2015]. If multiple units of the same DRI will be used, be sure to assess the interunit
30 variability of the units onsite. This can be done by co-locating, even for a short time, the DRI
31 units in an environment with a minimal particle concentration.

32 The next step should be to detect the presence and roughly estimate the mass concentration
33 of wildland fire smoke particles in absence of work activities. This can be done before or
34 after the work starts or upwind of the workplace operations. Although workplace
35 monitoring is preferable, offsite monitoring with a good understanding of wind direction
36 can be accepted. At this point, only Tier 1 tools have been used to detect wildland fire
37 smoke particles onsite, confirming the need for localized workplace exposure assessment.
38 However, DRIs struggle to accurately measure particle mass concentrations unless they are
39 specifically calibrated involving collection of samples on filter media and laboratory
40 analysis such as gravimetric analysis. Without calibration, DRI data should be considered an
41 estimation rather than an accurate measurement [NIOSH 2021; Vosburgh et al. 2022].

1 At this point, worksite surveys or personal exposure monitoring can be conducted,
2 considering SEGs, while collecting contextual information like worksite activities, tools
3 used, and engineering controls. Video-assisted monitoring can record this context, but
4 worker interaction is crucial [Patts et al. 2020]. The goal is to provide additional
5 information to the DRI data stream in case of personal monitoring; the additional
6 information can inform in which location of the workplace the worker was exposed to
7 particles, and if the particles were generated onsite. The key is the presence and relative
8 intensity of wildland fire smoke particle exposure, instead of accurate quantification. This is
9 described in Tier 3 evaluation below.

10 Another activity is monitoring particle concentrations inside and outside enclosed
11 environments (e.g., cabs) with engineering controls (e.g., pressurization and filtration).
12 When done properly away from onsite particle sources, this indicates if the enclosure
13 reduces worker exposure to wildland fire smoke particles. Two simultaneous DRI units
14 (inside/outside) or one alternating unit can assess the relative concentration reduction,
15 which is more important than the actual DRI values if intra-unit variability is low enough.
16 Although cabs may minimize onsite particle exposure, their effectiveness in the presence of
17 wildland fire smoke particles must be evaluated to inform the final assessment tier and
18 provide immediate guidance on using these controls.

19 **4.4.3 Tier 3**

20 **4.4.3.1 Sampling and Analytical Methods**

21 Wildland fire smoke is a complex mixture of particulate (solid and liquid) and gas phase
22 components. The constituents that make up the wildland fire smoke depend on the nature
23 of the wildland fire and the surrounding environment (see Section 2.1.). Table 4–5 shows
24 air sampling and analytical methods (listed as reference methods) for components that are
25 present at elevated levels during wildland and wildland-urban interface fire events. The
26 validated methods focus on those developed and published by federal agencies such as the
27 Occupational Safety and Health Administration (OSHA), NIOSH, and EPA. The methods have
28 been tested and developed to measure established occupational exposure limits or limits
29 within environmental regulations. Methods that have been developed to measure
30 occupational exposures at levels near a recommended exposure limit or permissible
31 exposure limit often lack adequate sensitivity to measure individual components at the
32 levels found in wildland fire smoke. Methods that clearly have insufficient sensitivity for the
33 measurement of components in wildland fire smoke will not be addressed. Air sampling and
34 monitoring methods for outdoor air quality assessment provide estimates of community-
35 level population exposures. Methods developed for occupational exposure assessment
36 typically rely on an assessment of personal exposure to contaminants in a worker's
37 breathing zone. Some methods allow area sampling of indoor contaminants and provide an
38 assessment of air quality within a limited indoor or outdoor environment. Personal
39 sampling methods often provide the best estimates of personal inhalation exposure to
40 workers [Vincent 2007]. However, the benefit of a personal sample is greatly diminished in
41 outdoor areas with a low spatial gradient of air contaminants. At large distances, away from

1 the fires (at the regional spatial scale in Figure 2-1), exposure levels and spatial variability
2 of gas and particulate contaminants are typically low. Therefore, the choice of measurement
3 method should rely more on the method's adequate sensitivity and selectivity than its
4 ability to collect a personal sample. The different types of methods and their relevance to
5 outdoor workers exposed to wildland fire smoke are discussed in greater detail in
6 Appendix C.

Table 4–5. Air sampling and analytical methods for smoke components that may be present at elevated levels during wildland and wildland-urban interface fire events and relevant considerations for choosing methods

Methods for gas phase pollutants						
Pollutant	Analysis technique	Reference method	Method sensitivity	Reported concentrations in smoke*	NIOSH recommended exposure limit [†]	Other exposure limits
Nitric oxide/nitrogen dioxide	UV/Vis spectrophotometer	NIOSH 6014	1 ppm (NO, 1.5-L); 0.5 ppm (NO ₂ , 3-L)	—	—	—
Nitrogen dioxide	Ion chromatography	OSHA ID-182	≤0.051 ppm (when modified for passive samplers) [SKC 2018]	Local ~20–90 ppb [‡]	1 ppm (STEL)	100 ppb (1-hr) [§]
Nitric oxide	Ion chromatography	OSHA ID-190	0.11–0.32 ppm (6-L)	—	—	—
Sulfur dioxide	Ion chromatography	NIOSH 6004	0.2 ppm (100–L)	—	2 ppm (TWA); 5 ppm (STEL)	75 ppb (1-hr) [§]
Sulfur dioxide	Ion chromatography	OSHA 1011	RQL 0.118 mg/m ³ (TWA) or 0.152 mg/m ³ (15 min sample) = 45 ppb (TWA) or 58 ppb (15 min)	—	2 ppm (TWA); 5 ppm (STEL)	75 ppb (1-hr) [§]
Carbon monoxide	Direct-reading	NIOSH 6604	LOD 1 ppm	≤ 18 ppm	35 ppm (TWA); 200 ppm (Ceiling)	9 ppm (8-hr) [§] ; 35 ppm (1-hr) [§]
Hydrogen cyanide	UV/Vis spectrophotometer	NIOSH 6010	< 3 mg/m ³ (3-L)	0.1–100 µg/m ³ [#]	5 mg/m ³ (STEL)	0.8 µg/m ³ ^{**}
Hydrogen cyanide	Ion chromatography	OSHA 1015	RQL 0.44 ppm (0.48 mg/m ³)	0.1–100 µg/m ³ [#]	5 mg/m ³ (STEL)	0.8 µg/m ³ ^{**}

1

Aldehydes

Pollutant	Analysis technique	Reference method	Method sensitivity	Reported concentrations in smoke*	NIOSH recommended exposure limit†	Other exposure limits
Formaldehyde	DNPH deriv, LC-UV	NIOSH 2016	0.015 mg/m ³ (15-L)	Smoke day max 56 µg/m ^{3††} ; Smoke day mean 4.9 µg/m ^{3††}	Ca ^{‡‡}	55 µg/m ^{3§§} ; 9.8 µg/m ^{3**}
Formaldehyde	DNPH deriv, LC-UV	EPA TO-11A	low ppb (1–24 h), ppm (5–60 min)	Smoke day max 56 µg/m ^{3††} ; Smoke day mean 4.9 µg/m ^{3††}	Ca ^{‡‡}	55 µg/m ^{3§§} ; 9.8 µg/m ^{3**}
Acetaldehyde	DNPH deriv, LC-UV	NIOSH 2018	MDL 13 µg/m ³	Smoke day max 32 µg/m ^{3††} ; Smoke day mean 3.3 µg/m ^{3††}	Ca ^{‡‡}	470 µg/m ^{3§§} ; 9 µg/m ^{3**}
Acetaldehyde	DNPH deriv, LC-UV	EPA TO-11A	low ppb (1–24 h), ppm (5–60 min)	Smoke day max 32 µg/m ^{3††} ; Smoke day mean 3.3 µg/m ^{3††}	Ca ^{‡‡}	470 µg/m ^{3§§} ; 9 µg/m ^{3**}
Acrolein	GC-MS	EPA TO-15A	MDL 7 pptv (0.016 µg/m ³)	Smoke day max 5.1 µg/m ^{3††} ; Smoke day mean 0.65 µg/m ^{3††}	Ca ^{‡‡}	2.5 µg/m ^{3§§} ; 0.02 µg/m ^{3**}
Aliphatic aldehydes	DNPH deriv, LC-UV	NIOSH 2018	0.05 mg/m ³ (15-L)	—	Ca ^{‡‡}	Various

3

Volatile Organic Compounds (VOC)

Volatile organic compounds	GC-MS	NIOSH 2549	not determined	≤ 100 µg/m ³ per VOC [#]	Various; benzene: Ca ^{‡‡}	Various; benzene: 27 µg/m ^{3§§} ; 30 µg/m ^{3**}
Volatile organic compounds	GC-MS	EPA TO-17	≤ 0.5 ppb	≤ 100 µg/m ³ per VOC [#]	Various; benzene: Ca ^{‡‡}	Various; benzene: 27 µg/m ^{3§§} ; 30 µg/m ^{3**}
Volatile organic compounds	GC-MS	NIOSH 3900	≥ 0.24 ppb	≤ 100 µg/m ³ per VOC [#]	Various; benzene: Ca ^{‡‡}	Various; benzene: 27 µg/m ^{3§§} ; 30 µg/m ^{3**}
Volatile organic compounds	GC-MS	EPA TO-15A	MDL 1–7 pptv	≤ 100 µg/m ³ per VOC [#]	Various; benzene: Ca ^{‡‡}	Various; benzene: 27 µg/m ^{3§§} ; 30 µg/m ^{3**}

1

Methods for Other Exposure Metrics

2

Pollutant	Analysis technique	Reference method	Method sensitivity	Reported concentrations in smoke*	NIOSH recommended exposure limit†	Other exposure limits
PM mass	Gravimetry	NIOSH 0600	LOD <30-75 µg/sample	5-550 µg/m ³ (as PM _{2.5})	—	—
PM mass	Gravimetry	NIOSH 0501	LOD <30-75 µg/sample	5-550 µg/m ³ (as PM _{2.5})	—	—
Particulate metals	ICP-OES	NIOSH 7300 series (7300 , 7301 , 7302 , 7303 , 7304 , 7306)	LOD ≥0.005 µg/sample	Heavy metals ≤ 0.3 µg/m ³ ††	Various	Various
Particulate metals	ICP-MS	OSHA 5003	RQL ≥0.0365 µg/m ³	Heavy metals ≤ 0.3 µg/m ³ ††	Various	Various
Particulate metals and metalloids	ICP-AES	OSHA ID-125G	Quantitative detection limits ≥0.20 µg	Heavy metals ≤ 0.3 µg/m ³ ††	Various	Various
Particulate metals	Atomic absorption spectroscopy	EPA Compendium IO-3.2	MDL ≥0.0001 ng/m ³	Heavy metals ≤ 0.3 µg/m ³ ††	Various	Various
Polynuclear aromatic hydrocarbons	GC-MS SIM	NIOSH 5528 ‡‡‡	LOD 0.08-0.2 µg/sample	Near field ΣPAH ~100-3000 ng/m ³ [Wentworth 2018; Navarro 2019]	Various; naphthalene: 50 mg/m ³ (TWA); 75 mg/m ³ (STEL)	Various; naphthalene: 3 µg/m ³ **
Polycyclic aromatic hydrocarbons	GC-MS	EPA TO-13A ‡‡‡	10 pg-1 ng	Near field ΣPAH ~100-3000 ng/m ³ [Wentworth 2018; Navarro 2019]	Various	Various
Particulate elemental/organic carbon	Thermal-optical analysis	NIOSH 5040	LOD ~2 µg/m ³ (960-L)	Near field PM _{2.5} ≤ 350 µg/m ³ (NAS 2022 Chapter 6); Regional PM _{2.5} ≤ 150 µg/m ³ (NAS 2022 Chapter 6)	—	PM _{2.5} : 35 µg/m ³ (24-hr) [§] ; PM ₁₀ : 150 µg/m ³ (24-hr) [§]

3

Other Hazardous Pollutants

Pollutant	Analysis technique	Reference method	Method sensitivity	Reported concentrations in smoke*	NIOSH recommended exposure limit†	Other exposure limits
Brominated and organo-phosphate flame retardants	UPLC/APPI/MS/MS	La Guardia and Hale [2015]	Detection limit 0.1 ng/m ³	—	—	—
Chlorinated furans and dioxins	HRGC/HRMS	EPA Method 23	≤ 0.18 ng/m ³ [Fent et al. 2020]	—	—	—

- 2 * See Section 2.1 for expanded information on potential chemicals found in wildfire smoke.
- 3 † NIOSH Pocket Guide to Chemical Hazards.
- 4 ‡ [Na and Cocker 2008].
- 5 § EPA National Ambient Air Quality Standard.
- 6 || [Adetona 2016].
- 7 # [O'Dell 2020].
- 8 ** EPA Reference Concentration for Inhalation Exposure.
- 9 †† [Rice 2023].
- 10 ‡‡ Potential carcinogens, see NIOSH Pocket Guide to Chemical Hazards Appendix A and/or Appendix C.
- 11 §§ California EPA Acute Reference Exposure Level.
- 12 |||| Although not a PAH specifically included in this method, retene could be measured as it is a PAH
13 associated with wildfire smoke [Navarro 2019].
- 14 Source: [EPA 1999a,b,c,d,e, 2023b; La Guardia and Hale 2015; NIOSH 2016, 2018; OSHA 1989, 1991a,b,
15 2002, 2007, 2010, 2019; SKC 2018].

4.5 Elements of an Effective Sampling Strategy

4.5.1 Exposure Metrics

18 The choice of exposure metric, a summary statistic, depends on the sampling objectives and
19 the type of exposure measurements collected. Exposures are not constant, but rather highly
20 variable in time and in space. Consequently, during sampling campaigns, multiple
21 measurements are collected that make using statistical tools necessary to summarize and
22 analyze data, as well as to run models. Common methods for summarizing data include
23 calculating summary metrics such as 24-hours, full-shift, or short-duration geometric mean
24 (GM), geometric standard deviation (GSD), arithmetic mean, and standard deviation.
25 Different quantiles, such as X95, as well as the highest exposure and particle size
26 distribution, are also used [Jahn et al. 2015].

27 Additional metrics used to summarize exposure include frequency, duration, and various
28 measures of intensity of exposure such as cumulative and peak exposures [Checkoway and

1 Rice 1992]. The following section highlights the relevant exposure metrics based on the
2 objectives and data types.

3 **4.5.1.1 Metrics for Tier 1 Data**

4 A variety of exposure assessment tools are available for Tier 1 assessment, including
5 models and real-time or near-real-time monitors. The wildland fire smoke models provide
6 crude information on the potential exposures to outdoor workers near or distant to the fire
7 event. The final decision from these models is whether a potential for exposure to wildland
8 fire smoke exists in a geographic area, without specifying any particular workplace.

9 Community, consumer, and low-cost based-monitors are often DRIs that provide real or
10 near real-time exposure data. DRIs can calculate running averages over selected durations
11 (e.g., 1, 5, 15, 60 minutes, or 8, 12, 24 hours). Filter-based sampling offers 24-hour averages
12 as exposure metrics. Additional exposure metrics from Tier 1 tools can include the
13 frequency with which exposures are exceeding certain exposure levels of interest, such as
14 the number of days above an AQI value. To detect deviations, daily averages can be
15 compared with prior levels or to exposure levels of interest.

16 Because wildland fire smoke is unpredictable, fleeting exposure episodes may not be
17 reflected in the longer period averages (e.g., 8- or 24-hour averages). However, they are
18 important for acute respiratory effects. Short-term DRI averages (e.g., 1, 5, 15, 60 minutes)
19 compared with prior patterns, after accounting for influencing factors like traffic, weather,
20 fire distance, and wind, can identify changes over shorter periods. Such changes can
21 indicate the potential for exposure from unusual sources like wildland fire smoke. With all
22 these variables to consider, it is important to note that the objective of Tier 1 analysis is to
23 determine if there is an increased risk of wildland fire smoke exposure. This information
24 can then be used to decide on the exposure mitigation action, and whether to progress to a
25 Tier 2 workplace assessment. Also, when a Tier 2 assessment is not feasible, or data are
26 unavailable, Tier 1 data such as AQI and PM_{2.5} may be used to make decisions on exposure
27 mitigation actions using the decision metric X95. As noted in Section 4.3, the decision to
28 stop the assessment, implement controls, proceed to the next tier assessment or some
29 combination of the above depends on numerous factors. These factors include the results of
30 the exposure assessment, confidence in the results, accuracy and variability of the results,
31 and other similar factors.

32 **4.5.1.2 Metrics for Tier 2 Data**

33 Tier 2 assessment focuses on using DRIs to assess particulate exposures of workers in
34 specific outdoor workplace SEGs, with an aim of refining the exposure estimate and the SEG,
35 mitigating exposure, determining the effectiveness of controls, and documenting exposure
36 risk. With strategic sampler placement, it may be possible to separate work-related
37 exposure sources from other sources, such as wildland fire smoke, for each SEG. Data for 8-
38 hour time-weighted average (TWA) exposures can be extracted from DRI data. Data
39 analysis can then be conducted for each SEG and exposure source (e.g., work-related vs.
40 wildland fire smoke source).

1 Because the objectives in this tier assessment are to mitigate exposure and document the
2 risk, several summary exposure metrics can be calculated for an SEG. These include GM,
3 GSD, arithmetic mean, standard deviation, and X95, to characterize the exposure. The X95 of
4 the full-shift concentrations is particularly relevant to make decisions on the controls
5 needed (see Chapter 5). It is also useful for informal comparison with relevant exposure
6 levels of interest. Formal comparison would require using approved sampling instruments.
7 The process of calculating X95 of the full-shift concentrations from DRI measurements and
8 comparison to exposure levels of interest is described in detail by Jahn et al. [2015].

9 However, decisions on the required control measures are often based on shorter duration
10 tasks or processes that generate exposures. In this case, moving averages of selected
11 durations (e.g., 1, 5, 15, 60 minutes) can provide important information on the short-term
12 variability of exposure within a work-shift. This information may be important for
13 identifying exposure excursions, developing control strategies, and calculating metrics of
14 peak exposures for epidemiologic studies.

15 The DRI data can be plotted to show the time of day when exposure excursions (peaks)
16 occurred, and then be visually or quantitatively assessed as exposures above a certain
17 concentration. Fifteen-minute moving averages are particularly useful for making decisions
18 on the need for controls because each point on the plot represents a 15-minute average.
19 These points can be directly used to make a decision for an individual worker. A similar
20 approach can be used when placing samplers to assess wildland fire smoke exposure to
21 understand the patterns of such exposures. Because the DRIs produce time series exposure
22 data, the computational complexity of analyzing or modeling such time series is significant
23 and requires assistance from a statistician or individuals familiar with working with these
24 types of data sets. The process is briefly described in Section 4.5.2.3.

25 **4.5.1.3 Metrics for Tier 3 Data**

26 The focus of the Tier 3 assessment is to evaluate the composition of wildland fire smoke for
27 workers in specific outdoor workplaces using time-integrated methods. Similar to the Tier 2
28 strategy, carefully placing samplers and selecting specific analytes can make it possible to
29 separate work-related exposure sources from other sources such as wildland fire smoke for
30 each SEG. The exposure metrics are the same as the 8-hour TWA particulate exposures
31 noted in Tier 2, with the choice of the X95 as a decision metric. Tier 3 assessment may
32 identify additional worker protection needed for specific chemicals such as gases and
33 various VOCs not offered by Tier 2 mitigation steps.

34 **4.5.2 Data Analysis**

35 **4.5.2.1 Data Processing**

36 Once sampling is completed, data from all tiers need to be stored in a spreadsheet or similar
37 tool and prepared for data analysis. DRI data require several steps including downloading
38 data from air monitor websites (Tier 1) or from the instrument (Tier 2) to a designated
39 software for further analysis and evaluation of the working environment. Also, as explained
40 in the DRI section (Section 4.4.2.1), a simple correction factor can be calculated as a ratio of

1 the full-shift average concentration from a reference sample collected on filter to the full-
2 shift average particle concentration from the DRI. This correction factor can be applied to
3 the real-time particle concentrations to adjust the readings to get the filter-equivalent-
4 corrected particle concentrations.

5 In some instances, particle concentrations may be below the limit of detection (LOD) of the
6 instrument. Further, DRIs (from Tier 1 or Tier 2) may treat such measurements differently,
7 such as leaving the concentration as blank or zero for those times. Recognizing the LOD of
8 the DRI, as well as other chemical analysis methods (Tier 3), is important to correctly
9 address any measurements that were at or below the LOD. Flawed assumptions about such
10 measurements can lead to incorrect inferences. Different methods can be used to calculate
11 summary statistics (e.g., GM or X95), accounting for LOD values such as those described by
12 Chen et al. [2021], Hewett and Ganser [2007], Houseman and Virji [2017], Huynh et al.
13 [2016], and Tian et al. [2024].

14 **4.5.2.2 Data Quality Control Measures**

15 Exposure measurements are affected by uncertainties that arise from multiple sources.
16 Examples are environmental variability or the bias and variability in different measurement
17 systems. Previous sections describing Tier 1–3 assessments address the data quality issue
18 from each measurement system type and the inferences that can be made using data from
19 these sources. More details on measurement uncertainty can be found in sampling and
20 analytical methods documentation and in DRI operating manuals and guidance documents
21 [NIOSH 2021].

22 Guidance is available on how to statistically analyze and assess the quality of environmental
23 datasets [EPA 2023a]. Tielemans et al. [2002] also propose guidelines for evaluating the
24 quality of exposure data to assess the relative value of data for risk assessment. Such a
25 procedure ensures the systematic evaluation of exposure datasets and identifies biases or
26 limitations. Furthermore, it helps create consistency among assessors. Such a procedure
27 also allows for pooling or aggregating exposure datasets from different sources. Several
28 researchers have provided details of the data quality evaluations and dataset developments
29 used in their studies [De Vocht et al. 2005; Vincent and Werner 2003]. Before performing
30 exposure data analysis, understanding the quality of the data and its representativeness is
31 critical to enhancing the interpretation of analytical results.

32 **4.5.2.3 Quantitative Analysis**

33 Exposure measurements are highly variable in time and space. Characterizing this
34 variability is important to make accurate inferences. Exposure variability arises from many
35 sources such as location, tasks, processes, activities, weather conditions, topography, and
36 engineering controls. The joint effects of all these factors can generate a wide range of
37 exposures with measurements that are orders of magnitude apart [Rappaport and Kupper
38 2008].

39 The distribution of many occupational and environmental measurements shows a right-
40 skewed shape when visualized, which is a characteristic of the lognormal distribution. Many
41 occupational and environmental measurements are commonly assumed to follow a

1 lognormal distribution, (i.e., the natural logarithm of the exposure data has a normal
2 distribution). This assumption enables the use of a large array of statistical data analysis
3 and modeling tools. These tools can be used to make inferences about various worker
4 populations when combined with the concept of SEGs.

5 As previously noted, the GM, GSD, and X95 are the typical exposure metrics calculated from
6 lognormally-distributed exposure data [Jahn et al. 2015; Rappaport and Kupper 2008]. The
7 AM is also the relevant metric for calculating cumulative exposure [Smith 1992].

8 Along with extracting TWA data from the exposure time series, the time series exposure
9 data can be analyzed using statistical models. This is especially true when observations or
10 self-reported activity diaries are collected during sampling. However, several issues in
11 addition to the performance of DRI can make the statistical analysis of real-time data
12 difficult. This includes the likelihood for nonstationary autocorrelation among successive
13 measurements, and the presence of left-censoring caused by LOD. A statistical approach has
14 been developed to summarize and model real-time exposure data that address these issues
15 [Houseman and Virji 2017].

16 **4.6 Summary**

17 Wildland fire smoke is a complex mixture of particulates and gases, and exposures to it can
18 vary in space and in time. These factors make exposure assessment challenging, requiring a
19 rational approach to best understand worker exposure. Fortunately, $PM_{2.5}$ is an excellent
20 indicator of a wildland fire smoke mixture; therefore, $PM_{2.5}$ measurements can be useful for
21 risk management. A tiered approach to measuring $PM_{2.5}$ can assist with decision-making by
22 improving confidence in exposure assignment. Tier 1 methods are readily available and can
23 be useful but may lack timeliness and confidence. Tier 2 methods improve confidence and
24 include all sources of exposure but require equipment and trained personnel to operate.
25 Tier 3 methods are established methods for assessing health risk in the workplace. They
26 offer the most confidence for exposure assessment, but they are not as timely for decision-
27 making purposes.

28 Understanding the effectiveness of available exposure assessment tools is critical to address
29 assessment objectives and determine how best to protect outdoor workers from wildland
30 fire smoke exposure.

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3 Chapter 5: 4 Controlling Workplace Exposures

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Key Chapter Takeaways

- The U.S. Environmental Protection Agency (EPA) Air Quality Guide for Particle Pollution recommendations for Air Quality Index (AQI) and the hierarchy of controls form the basis of NIOSH worker protection recommendations to reduce wildland fire smoke exposure to outdoor workers. These recommendations should be incorporated into an overall workplace safety and health program.
- Elimination should be practiced using wildland management to reduce the likelihood of wildland fires.
- Substitution is not applicable for wildland fire events.
- Engineering controls can be applied using enclosed buildings, temporary or permanent structures, and vehicles, where filtered air is provided to reduce workers' exposure.
- Administrative controls begin with preparation for wildland fire smoke events, along with worker training and education. Various administrative control approaches are discussed to include relocation, reduction of shift length, rotation of workers, work-rest cycles, and a reduction of work intensity.
- Respirators, when selected and used properly as part of a respiratory protection program (RPP), can protect against inhalation hazards from wildland fire smoke.
- Workers should be allowed to seek medical care if they experience signs or symptoms of injury or illness due to wildland fire smoke exposure.

25 A crucial aspect of ensuring the health and safety of all workers is to control workplace
26 exposures, and in the case of outdoor workers, their potential for exposure to wildland fire

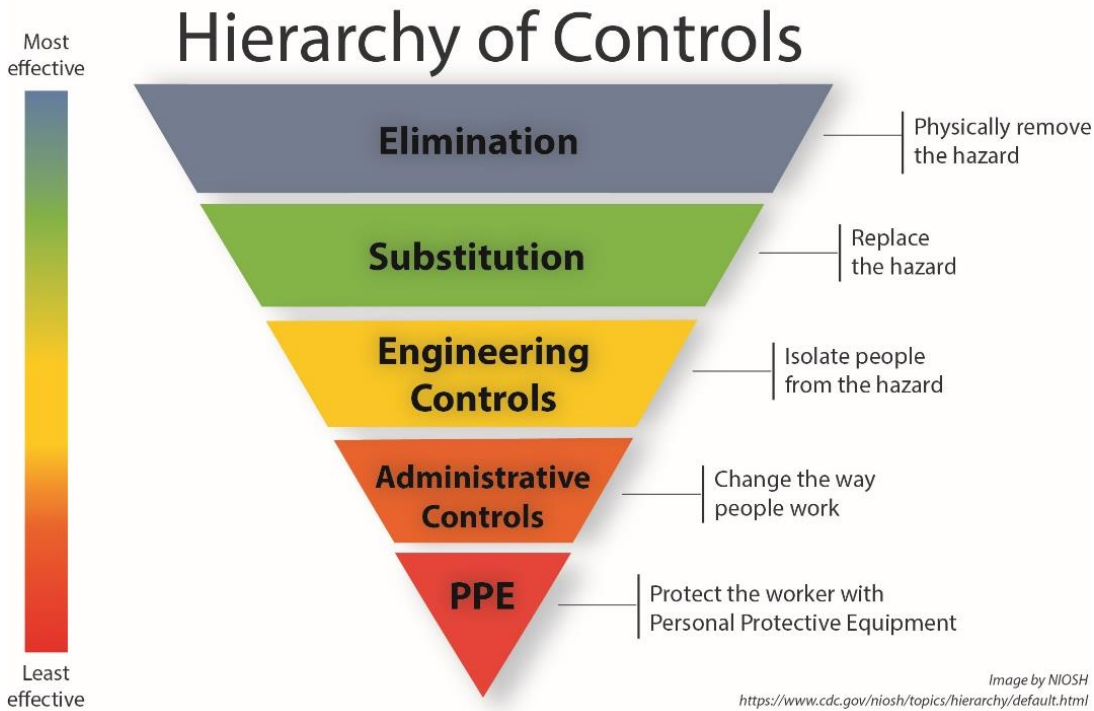
1 smoke. This should be part of an overall workplace safety and health program, a proactive
2 approach to achieve higher levels of safety and health along with increasing productivity
3 and business operations. See the Occupational Safety and Health Administration (OSHA)
4 [Recommended Practices for Safety and Health Programs](#) and [Safety Management](#) as well as
5 a topic page on [Wildfires](#), which provides information for workers and employers on
6 preparing for a wildfire and protecting themselves in the wildfire’s aftermath.

7 **5.1 Control Options Using the Hierarchy of Controls**

8 The hierarchy of controls forms the basis of NIOSH worker protection recommendations
9 (see Figure 5–1). The hierarchy of controls strategy outlines, in descending order of priority
10 and effectiveness, using elimination, substitution, engineering controls, administrative
11 controls, and lastly personal protective equipment (PPE). See NIOSH’s [About Hierarchy of](#)
12 [Controls](#) for more information.

13 In the hierarchy of controls strategy, hazard elimination and substitution are the most
14 effective controls. While in some situations wildland management may reduce the
15 likelihood of wildland fires, or the timing and extent of prescribed burns may limit the
16 hazard of wildland fire smoke, these options may not be viable control options to reduce
17 exposures to wildland fire smoke for outdoor workers. Substitution is not applicable for
18 wildland fire events.

19 Instead, the primary categories for managing wildland fire smoke exposures include
20 engineering controls, administrative controls, and PPE, which should be considered in that
21 order. This chapter outlines control options for each of these categories, recognizing that
22 there may be practical considerations in both space and time, followed by a recommended
23 approach to controlling wildland fire smoke exposure to outdoor workers.



1
2 **Figure 5–1. NIOSH hierarchy of controls**

3 **5.1.1 Engineering Controls**

4 Engineering controls reduce hazards or
5 prevent hazards from coming into contact
6 with workers. Engineering controls can
7 include using protective barriers or installing
8 ventilation or filtration, and more.

9 For wildland fire smoke exposures, one
10 possible engineering control is using enclosed
11 buildings, structures, or vehicles where the
12 air is filtered to reduce workers' exposure
13 [California Code of Regulations 2019; Oregon
14 Administrative Rules 2022a,b; Washington
15 Administrative Code 2024]. The use of
16 enclosures, including structures or vehicles,

17 to provide a respite area with cleaner air (especially during breaks) for workers may be
18 helpful during times of poor air quality due to wildland fire smoke. In addition, EPA has
19 recommended reducing infiltration of smoke into the indoor environment by closing doors
20 and windows, using higher efficiency mechanical filters in heating, ventilation, and air
21 conditioning (HVAC) systems, and using portable air cleaners (see EPA's [Wildland Fires and
22 Smoke](#)). These efforts are estimated to provide an exposure reduction to particulate matter
23 in the range of 20%–80% [Laumbach 2019].

Engineering controls to protect workers exposed to wildland fire smoke can be applied by moving workers into enclosed buildings, temporary or permanent structures, or vehicles, where:

- Outside air infiltration is minimized,
- Smoke particles are effectively filtered, and
- Enclosure air PM_{2.5} concentrations are monitored to assess control effectiveness.

1 While the American Society of Heating, Refrigerating and Air-Conditioning Engineers
2 (ASHRAE) is developing guidelines to protect occupants from wildland fire smoke in indoor
3 spaces such as commercial buildings, institutions, and healthcare facilities [Javins et al.
4 2021], very little has been published regarding outdoor workers exposed to wildland fire
5 contaminants. PM_{2.5} is commonly used as a surrogate for the components of outdoor smoke
6 given its ability to penetrate deep into the lungs, causing short- and long-term health effects
7 (see Chapter 3). Several considerations apply to properly developing and maintaining an
8 enclosure with controlled particulate levels, including: (1) how to minimize outdoor air
9 infiltration; (2) how to effectively filter air to reduce airborne particles, including PM_{2.5}; and
10 (3) how to monitor the airborne PM_{2.5} concentration within the enclosure [Javins et al.
11 2021].

12 The development of temporary cleaner air enclosures, specifically for outdoor worker use,
13 has not been studied extensively. If a temporary or permanent enclosure will be used as a
14 method to reduce outdoor employee exposure to wildland fire smoke, the following issues
15 should be considered. Infiltration is the primary path for exposures of building occupants to
16 outdoor particulate matter. Diapouli et al. [2013] reviewed several published studies that
17 looked at rates of infiltration of PM_{2.5} into buildings from outdoor sources and showed mean
18 infiltration values ranging from 0.4 to 0.8 (representing the ratio of inside to outside PM_{2.5}
19 concentrations) [Diapouli et al. 2013]. Guidance on mitigating exposure to wildland fire
20 smoke from the EPA typically centers around keeping doors and windows closed, sealing
21 cracks and openings, and positively pressuring occupied spaces to reduce infiltration of
22 pollutants from outdoor sources [EPA 2023a]. Minimizing air infiltration requires the
23 sealing of pathways for contaminants to enter the enclosure, including through doors,
24 windows, or any other penetrations, as well as leakage through the enclosure envelope. This
25 may be especially challenging in a temporary enclosure without hard walls or connections
26 to the ground. The amount of infiltration may also be susceptible to environmental
27 conditions such as high winds, which can increase enclosure infiltration.

28 Effectively removing particulate matter requires a filter with capabilities for the particle
29 size range of interest. A mechanical filter with particle removal effectiveness in the range of
30 PM_{2.5} would be necessary as a minimum requirement to remove particulates generated by
31 wildland fire smoke.

32 Azimi et al. [2014] modeled the filtration effectiveness of air filters across a range of
33 minimum efficiency reporting values (MERV) and outdoor particle size distributions. They
34 showed that the PM_{2.5} removal efficiency ranged from 1% to 8% for filters of MERV 7 or
35 lower to over 99% for high efficiency particulate air (HEPA) filters. Although these
36 simulations did not evaluate the MERV 13 filter, it is the filter most often recommended for
37 building filtration for smoke particles, where the primary exposure of concern is the PM_{2.5}.
38 This is due to its lower airflow resistance compared with HEPA and reasonable filtration
39 effectiveness against smaller particle sizes [Javins et al. 2021].

40 Joseph et al. [2020] reviewed and summarized the evidence from peer-reviewed literature
41 about the effectiveness of air filtration as an intervention to decrease exposure to wildland
42 fire smoke and protect health when sheltering indoors. Overall, they found that using HEPA

1 filters and electrostatic precipitators seemed to be effective in reducing exposure to air
2 pollutants produced by wildland fires and may potentially limit adverse health impacts
3 from exposure as well. While a HEPA filter would provide more effective particle removal
4 than lower MERV-rated filters, it does so at a cost. HEPA filters require a larger fan and
5 more power and have higher filter replacement costs compared with other filters [Stephens
6 et al. 2022].

7 Using portable air filtration systems, also referred to as portable air cleaners, can augment
8 other approaches when creating cleaner air spaces. These systems can provide
9 supplemental filtration to assist in areas where there are higher occupant densities or
10 where the general ventilation system is not meeting indoor particle concentration goals.
11 The EPA provides guidance on how to select portable air cleaners for enclosed spaces based
12 on the Clean Air Delivery Rate (CADR), a metric developed to help consumers select the
13 right air cleaner based on room size (see EPA's [Guide to Air Cleaners in the Home](#)). These
14 units can be used to remove small, medium, and large particles, as represented by tobacco
15 smoke, dust, and pollen. To address wildland fire smoke, a portable air cleaner that has a
16 high CADR for tobacco smoke should be selected.

17 The ability to monitor PM_{2.5} or other air quality metrics inside cleaner air spaces is
18 important to ensure that controls are working effectively. The authors of the draft ASHRAE
19 "Guideline 44P, Protecting Building Occupants from Smoke During Wildfire and Prescribed
20 Burn Events," have released a planning framework that suggests using one or more low-cost
21 air monitors equipped with a PM_{2.5} sensor to assess trends in air quality in indoor spaces
22 [Javins et al. 2021]. These monitors could be used to measure changes in both the outdoor
23 and indoor air quality and provide key information such as whether there are sources of
24 infiltration due to space envelope leaks or degraded filter performance [Javins et al. 2021].
25 Chapter 4 provides more information on real-time particulate monitors.

26 Another potential location for a cleaner air space is inside a vehicle [California Code of
27 Regulations 2019; Oregon Administrative Rules 2022a,b; Washington Administrative Code
28 2024]. Some data have been collected on the ability of vehicle filtration to remove particles
29 from outdoor sources. A test conducted on vehicles in Sweden and China showed that inside
30 PM_{2.5} concentrations were significantly reduced using a new cabin air filter, but the
31 reduction effects were highly degraded with an aged filter. The ratio of inside to outside
32 PM_{2.5} concentrations was 0.2 for new filters versus 0.6 for the aged filter [Wei et al. 2020].

33 Another study has shown that exposure reduction from automotive cabin filtration systems
34 differed depending on particle sizes with higher efficiencies for smaller particles—44% for
35 100 nm particles and 72% for 20 nm particles [Qi et al. 2008]. Further, this study showed
36 that at higher ventilation fan rates, the filtration efficiency was reduced (from 40% to 23%
37 for particles less than 1 µm in size). Using the recirculation setting in a vehicle (versus the
38 fresh air setting) was also shown to result in enhanced particle reduction, taking only 3
39 minutes at this setting to substantially lower cabin particle concentrations [Qi et al. 2008].
40 The potential for hazards (carbon monoxide [CO] and PM) arising to vehicle occupants or
41 bystanders from the vehicle exhaust during idle should be taken into account when
42 considering this approach to protect workers.

1 NIOSH has conducted research on the fundamental performance of enclosed cab and booth
2 filtration effectiveness for the mining industry. While most of this work is focused on the
3 effective mitigation of respirable dusts, and not specifically for wildland fire smoke, many of
4 the basic principles would apply to removing smoke particles as well if the appropriate
5 filter media was employed. NIOSH found that intake filter efficiency and using a
6 recirculation filter were the most influential factors on cab penetration performance
7 [NIOSH 2008]. It is important to consider that an effective filtration system and a cab with
8 solid integrity (ability to achieve positive pressurization with minimal leakage) are needed
9 to ensure operator safety [NIOSH 2018]. While using higher filtration efficiency media
10 removes a greater number of smaller particles, there is a trade-off between real-world
11 filtration and fixed-fan power [Patts et al. 2018]. NIOSH has published research on using
12 these same concepts (high-efficiency filters, tight seals on doors and windows, and a
13 pressurized cabin) to protect agricultural workers in tractor cabins during pesticide
14 applications [Hall et al. 2002, Heitbrink et al. 2003, Moyer et al. 2005].

15 The potential for using engineering controls to reduce worker exposure during wildland
16 fires depends on many logistical factors and requires preplanning. Implementing a
17 temporary enclosure in the field for outdoor workers would require access to power and
18 the ability to construct a facility that meets the key minimum requirements discussed
19 above. Cooling would also likely be necessary depending on the outdoor temperature and
20 number of workers to be protected by the enclosure. A study looking at the co-burden of
21 heat and PM_{2.5} exposures for agricultural workers from 2010 to 2018 in Washington
22 showed that peak PM_{2.5} exposures occurred when the heat index was around 85°F and
23 during the summer when wildland fires are most prevalent [Austin et al. 2021]. Using new
24 approaches, as well as using existing buildings or vehicles as a cleaner air refuge, needs to
25 be fully assessed before implementation.

26 **5.1.2 Administrative Controls**

27 Administrative controls refer to measures taken to manage and control workplace hazards
28 through policies, procedures, and practices. While administrative controls should be
29 considered as a tool to reduce exposure, they are considered less effective than engineering
30 controls due to their reliance on effectively communicating these controls to workers, as
31 well as worker understanding and action. The administrative controls discussed in this
32 section should be considered as part of the hierarchy of controls approach to manage
33 wildland fire smoke exposure to outdoor workers.

34 **5.1.2.1 Preparedness**

35 Most outdoor workers will have some level of wildland fire smoke exposure during their
36 career, and therefore it is important for employers to prepare workers for these events.
37 Preparation should be included as part of a comprehensive workplace health and safety
38 program. While holistic preparation guidance should be developed (see [OSHA's Wildfire
39 Preparedness](#) website), preparation for limiting exposure, based upon good industrial
40 hygiene practice, should include the following activities:

- 1 • **Identify sensitive populations:** Prior to wildland fire events, employers should
2 identify workers who are considered susceptible to smoke exposure, based on a
3 physiological condition or potentially increased exposure. These workers may be at
4 greater risk for developing adverse health effects. The AQI uses the term “sensitive
5 groups.” This includes people with asthma or other respiratory diseases, people
6 with cardiovascular disease, children under 18 years old, pregnant people, older
7 adults, people of low socio-economic status and outdoor workers, [EPA 2024c].
8 Employers should advise workers who are in sensitive groups in advance of wildfire
9 smoke events on methods to protect themselves.
- 10 • **Provide communication methods:** Plan ways to communicate with personnel on
11 methods to anticipate, recognize, and control wildland fire smoke exposure. This
12 should include communication methods in a language and format which are
13 understood by all workers, including those who may have low literacy or are unable
14 to read.
- 15 • **Establish control measures:** Anticipate the needs of the workforce by purchasing
16 appropriate engineering controls and PPE. Ensure the PPE is adequately stored and
17 maintained.

18 5.1.2.2 Worker Education and Training

19 Educating and training workers on the potential hazards of wildland fire smoke are critical
20 to preventing adverse health effects from exposure. Training has been shown to be effective
21 in protecting workers when the following conditions are met:

- 22 • Workers are educated about the potential hazards of their job.
- 23 • Knowledge and work practices become improved.
- 24 • Workers are provided the necessary skills to perform their jobs safely.
- 25 • Management shows commitment and support for workplace safety [NIOSH 2010].

26 Requirements for worker education and training are specified in the OSHA Hazard
27 Communication standard, 29 CFR 1910.1200 [OSHA 2024b]. Employers should provide
28 health and safety training to workers and their supervisors before they begin working in
29 wildland fire smoke. Supervisors also should be trained on how to monitor air quality and
30 weather advisories.

31 Heat is another potentially hazardous condition that may accompany wildland fire smoke
32 environments. NIOSH [2016b] provides training recommendations for workers and their
33 supervisors where there is a reasonable likelihood of heat injury or illness. Training topics
34 include the health hazards of heat stress, signs and symptoms of heat stress, and first aid
35 procedures [NIOSH 2016b]. This guidance may also apply in wildland fire smoke
36 environments.

37 Workers who should receive training on the potential hazards of wildland fire smoke
38 include those whose work is essential during a wildland fire smoke event, those who are
39 likely to be exposed when a wildland fire smoke event occurs, and managers or supervisors
40 who are responsible for assignment and oversight of those workers’ tasks. A program for

1 educating workers should also include both instruction and “hands on” training that
2 addresses the following:

- 3 • The potential health risks associated with exposure wildland to fire smoke.
- 4 • Procedures and tools for avoiding or minimizing exposure to wildland fire smoke.
5 These include properly using engineering controls (such as cleaner work
6 environments with filtered air), work practices (such as reducing physical tasks and
7 taking breaks), and PPE (such as respirators).
- 8 • Recognizing and preventing signs of heat stress, particularly when PPE is used.
- 9 • Instructions for reporting health symptoms.

10 According to OSHA’s Respiratory Protection standard (29 CFR 1910.134), which applies to
11 General Industry (part 1910), Shipyards (part 1915), Marine Terminals (part 1917),
12 Longshoring (part 1918), and Construction (part 1926) employers must provide a full
13 respiratory protection program, including worker education and training, for their
14 employees when workers are required to wear respiratory protection by the employer or
15 according to an applicable OSHA standard [OSHA 1998]. Employers with workers engaged
16 in agricultural operations (e.g., farmworkers) are covered by OSHA’s safety and health
17 standards for agriculture in 29 CFR Part 1928 and must also comply with the General Duty
18 Clause of the Occupational Safety and Health Act of 1970 (OSH Act). This requires
19 employers to provide their employees “employment and a place of employment which are
20 free from recognized hazards that are causing or are likely to cause death or serious
21 physical harm” 29 USC 654(a)(1). OSHA State plan states may have other training
22 requirements for respiratory protection in addition to those required by OSHA.

23 All training and associated materials should be in a language and format that is understood
24 by all workers, including those who have low literacy levels or are unable to read. Training
25 should be conducted using materials and in a manner that ensures health equity for all
26 workers (see Section 2.4).

27 **5.1.2.3 Hygienic Practices**

28 Although the amount and extent of dermal and oral exposure to outdoor workers during
29 wildland fire events is uncertain, prudent public health practice would be for workers to
30 wash their hands before eating, drinking, or smoking. This will reduce the potential for oral
31 exposures from wildland fire smoke. Additionally, after each shift, workers should:

- 32 • change clothes or wash all contaminated clothing before or immediately after
33 returning home, and
- 34 • take a shower including thoroughly washing their hair as soon as possible following
35 outdoor work. This will likely reduce dermal exposures to wildland fire smoke
36 components by removing contaminants from the skin.

37 **5.1.2.4 Relocation**

38 To reduce or eliminate exposure to wildland fire smoke, outdoor workers may be relocated
39 from a place or job site with potential exposure to an area with limited or no exposure.

1 However, relocation may not be possible for certain jobs or due to the widespread
2 dispersion of wildland fire smoke. When relocating workers, it is crucial to monitor and
3 control wildland fire smoke exposures as much as possible. This includes taking exposure
4 measurements, as outlined in Chapter 4, to ensure the workers have been moved to an
5 environment with minimal or no smoke. The primary advantage of relocation is limiting
6 exposure to lower or no levels of wildland fire smoke, making it an effective control
7 mechanism when feasible.

8 **5.1.2.5 Reduction of Shift Length**

9 During periods of wildland fire smoke exposure, reducing shift length for work in affected
10 areas can help lower the overall exposure burden on workers. However, this approach may
11 not be feasible due to time-sensitive work requirements or limited workforce availability.
12 When implemented, a shorter shift duration should result in reduced wildland fire smoke
13 exposure for workers. Workers should be advised to use their off-shift time to relocate to
14 areas with minimal or no smoke exposure as well as on methods to reduce exposure at
15 home. Effective communication with workers is necessary to coordinate such measures.

16 **5.1.2.6 Rotation of Workers**

17 Rotating workers or moving them from one job location to another can be an option to
18 consider for limiting outdoor workers' exposure to wildland fire smoke. Rotating workers
19 limits their exposure to the time spent working in the affected area. This approach assumes
20 that the remaining work shift is spent in an area with minimal or no smoke exposure.
21 Confirming exposure levels during rotation is crucial. However, this method has a few
22 disadvantages. First, it requires oversight and comprehensive monitoring of workers'
23 exposure times, which can be resource intensive. Additionally, this approach results in more
24 workers being exposed to some level of wildfire smoke. Furthermore, worker rotation may
25 not be feasible if there is a limited pool of available workers for the job.

26 **5.1.2.7 Work-Rest Cycles**

27 Work-rest cycles, involving alternating periods of work in an exposed environment
28 followed by periods of no or limited exposure, can be used to reduce the overall exposure
29 burden on outdoor workers. This should be applied as part of scheduled breaks during a
30 workshift. Similar to worker rotation, this approach requires oversight and monitoring of
31 workers' exposure duration, as well as an understanding of exposure levels. This option
32 assumes that during the rest periods, workers are in areas with minimal or no smoke
33 exposure, which should be confirmed through air monitoring.

34 **5.1.2.8 Work Intensity**

35 Work intensity can influence the burden of exposure, and reducing the type and amount of
36 activity should be considered as a potential administrative control. As work intensity
37 increases, individuals breathe more deeply and rapidly, which will result in a higher intake
38 of wildland fire smoke. It is unclear how changes in work intensity quantitatively impacts
39 exposure to wildland fire smoke and resulting health impacts. Furthermore, while

1 performing less intense activities on days where exposure levels are higher is an option, the
2 reduction of exposure is difficult to estimate.

3 **5.1.3 Personal Protective Equipment**

4 PPE is the last line of defense and should not take the place of engineering and
5 administrative controls [NIOSH 2022c]. However, PPE may be needed while other control
6 measures are being implemented or if a combination of controls is necessary. This section
7 will discuss the methods and challenges associated with using PPE to protect outdoor
8 workers from wildland fire smoke. To select PPE, the employer must first perform a hazard
9 assessment of the workplace, in accordance with OSHA's Personal Protective Equipment
10 standard, 29 CFR 1910.132 [OSHA 2016]. This assessment identifies potential or existing
11 hazards, which determines what controls, including PPE, are necessary to protect the
12 worker. In addition to selecting PPE to protect against wildland fire smoke, all other
13 associated workplace hazards (e.g., heat) should also be considered before a final PPE
14 strategy is adopted. As mentioned in previous chapters, NIOSH recognizes PM_{2.5} as the
15 primary hazard of concern in wildland fire smoke to outdoor workers due to its ability to
16 cause direct health effects and capacity to migrate long distances with the smoke plume.
17 Consequently, the following sections focus on PPE strategies that mitigate PM_{2.5} exposures.

18 **5.1.3.1 Respiratory Protection**

19 NIOSH Approved® respirators (known as filtering facepiece respirators [FFRs]) filter
20 particulates from ambient air. A respirator class known as N95® is capable of filtering
21 atmospheric particulates (free of oil aerosols) with up to 95% efficiency [Air-purifying
22 particulate respirators; description, 2023]. A respirator class known as P100® is capable of
23 filtering atmospheric particulates with a filter efficiency of 99.97% [Air-purifying
24 particulate respirators; description, 2023]. With their ability to filter particles of all sizes,
25 including PM_{2.5} (and up to ~8-25 times larger than 2.5 µm), FFRs provide effective
26 respiratory protection against particulate inhalation hazards from wildland fire smoke. It is
27 important to understand that FFRs do not protect against gases, such as carbon monoxide.
28 These classes of respirators also do not supply air to the wearer and will not protect against
29 a low oxygen (hypoxic) atmosphere [Williams 2010]. It is important to be aware of the
30 potential health risks associated with increased heat stress when using respirators [NIOSH
31 2016b; Williams and Cichowicz 2020].

32 Reusable elastomeric respirators with interchangeable filters are another option and
33 typically are in the respirator class of P100®. Since these respirators are reusable and the
34 filters are replaceable, they require a prescribed cleaning and maintenance process
35 between each use.

36 **5.1.3.2 Respiratory Protection Program**

37 When respirator use is required, employers covered by OSHA's Respiratory Protection
38 standard, 29 CFR 1910.134, must comply with the standard, including implementing a full
39 respiratory protection program (RPP) [OSHA 1998, 2000], when applicable. 29 CFR
40 1910.134 applies to General Industry (part 1910), Shipyards (part 1915), Marine Terminals

1 (part 1917), Longshoring (part 1918), and Construction (part 1926). Employers with
2 workers engaged in agricultural operations (e.g., farmworkers) are covered by OSHA’s
3 safety and health standards for agriculture in 29 CFR Part 1928 and must also comply with
4 the General Duty Clause of the Occupational Safety and Health Act of 1970 (OSH Act). This
5 requires employers to provide their employees “employment and a place of employment
6 which are free from recognized hazards that are causing or are likely to cause death or
7 serious physical harm” 29 USC 654(a)(1). OSHA State Plan states with existing
8 requirements to protect workers from wildland fire smoke may have specific requirements
9 for respiratory protection. A respiratory protection program must include the following key
10 requirements.

- 11 • Written program with policies and procedures for the specific workplace.
- 12 • Hazard evaluation and respirator selection.
- 13 • A designated and qualified RPP administrator.
- 14 • Initial and refresher training in respiratory hazards and proper use and
15 maintenance of respirators.
- 16 • Respirator medical evaluation (see OSHA’s [Respirator Medical Evaluation](#)
17 [Questionnaire](#)).
- 18 • Fit testing procedures.
- 19 • Positive (exhaling gently) or negative pressure (inhaling sharply) seal checks should
20 be performed each time the respirator is put on to assure an effective seal.
- 21 • Schedules for proper respirator maintenance and care (e.g., cleaning, storage,
22 inspection, repair).
- 23 • Routine and emergency respirator use.
- 24 • Ensure requirements are followed for voluntary use of respirators, when applicable.
- 25 • RPP evaluation.
- 26 • Record keeping.

27 For more on these requirements, refer to the “OSHA Small Entity Compliance Guide for the
28 Respiratory Protection Standard” [OSHA 2011], which contains checklists for establishing
29 an RPP, respirator selection, medical evaluations, training, fit testing, and proper use,
30 maintenance, and care of respirators.

31 A comprehensive RPP is essential to help ensure the protection (i.e., the assigned protection
32 factor in Table 1 of OSHA’s 29 CFR 1910.134 [OSHA 1998]) that a NIOSH Approved®
33 respirator claims can be achieved. Therefore, it is important that those using respiratory
34 protection in the workplace follow all components of the program and carefully read and
35 follow the respirator manufacturer instructions. NIOSH, OSHA, and many respirator
36 manufacturers also provide information, including donning posters and videos, that can
37 help novice users become familiar with the requirements and best practices for respirator
38 use. [NIOSH 2022a,b; 2023a; OSHA 2016].

39 Proper planning should be considered an essential ingredient for respiratory protection.
40 This includes maintaining an adequate supply of FFRs and/or reusable elastomeric
41 respirators as well as conducting training, fit testing, and appropriate

1 cleaning/decontaminating procedures (in the case of reusable elastomeric respirators) in
2 advance of need, and all other requirements of the RPP.

3 **5.1.3.3 Voluntary Use of Respirators**

4 If respiratory protection is not required by an applicable OSHA standard or by the
5 employer, workers can choose to voluntarily wear respirators. The employer will determine
6 that wearing respirators voluntarily will not in itself create a hazard, and follow the
7 requirements in OSHA's Respiratory Protection Standard, 29 CFR 1910.134, for voluntary
8 use of respirators by employees. Respirators worn voluntarily can be acquired by the
9 worker or provided by the employer and are strongly recommended to be NIOSH
10 Approved®. If the employer determines that voluntary respirator use is permissible, the
11 employer must provide respirator users with the information contained in Appendix D of
12 the standard, [Information for Employees Using Respirators When Not Required Under the](#)
13 [Standard \[OSHA 1998\]](#).

14 **5.1.3.4 Dermal Personal Protective Equipment**

15 It is important to acknowledge that dermal exposures will occur with workers in the path of
16 a plume of wildland fire smoke. Most of the information on dermal exposures to wildland
17 fire smoke comes from research on the fire service [Fent et al. 2017; Kesler et al. 2021;
18 Mayer et al. 2019; Mayer et al. 2020; Mayer et al. 2023], and little is known about these
19 effects on outdoor workers. In the absence of detailed information on the dermal
20 contribution of exposure to wildland fire smoke, outdoor workers should consider changing
21 clothes after each shift, showering as soon as possible following outdoor work, and washing
22 all contaminated clothing.

23 **5.1.3.5 Challenges to Wearing Respiratory Protection**

24 Factors, both physiological and psychological, may determine whether a person is willing or
25 able to wear an FFR to protect against environmental PM_{2.5}. Understanding the
26 psychophysiological responses to wearing an FFR must be central to developing a
27 respiratory hazard plan that mitigates threats to health [ISO 2013].

28 Users have reported the following negative issues associated with wearing a respirator: (1)
29 air hunger, (2) headache, (3) thermal discomfort, (4) dry mouth, (5) claustrophobia, and (6)
30 anxiety. Wearing certain types of respirators can also reduce the field of vision due to the
31 physical structure of the respirator obstructing parts of view [ISO 2013; Williams 2010;
32 Williams and Cichowicz 2020]. Certain types of respirators may also increase the
33 perception of heat stress by stimulating facial thermal receptors that provoke the sense of
34 heat. These responses may be aggravated by a preexisting physiological state (e.g.,
35 increased heart rate). Some of the psychological tolerability of wearing FFRs may depend on
36 the perceived external threat. For example, the wearer of an FFR may be more motivated to
37 wear and tolerate the discomfort of the FFR in a setting where the external risk is exposure
38 to a hazardous environment such as wildland fire smoke [ISO 2013]. In addition,
39 perceptions of comfort and tolerability may improve over time given proper fit, training,
40 and wearer experience with the FFR (at least in hospital settings) [Pompeii et al. 2020;
41 Pompeii et al. 2024]. In any event, thermal stress (either perceived or that which changes

body core temperature) is an important factor when considering the use of PPE protection in a hot outdoor environment. The possible risks of wearing a respirator during wildland fire smoke exposures are summarized by EPA and partners [EPA 2021]. The health risk that may be imposed on the wearer of the FFR should not surpass the risk of exposure to the environmental hazard.

Many studies conducted in occupational settings suggest that respirator tolerance and effective use rely on effective RPPs, including fit testing and seal checks prior to use as well as respirator training [Goko et al. 2023; Hannum et al. 1996; Jones et al. 2013]. Training can use different methods to achieve proper respirator use and effective fit [Jones et al. 2013].

Thus, the decision to use FFRs or elastomeric respirators should consider the cost versus the benefit, physiological burden (to include heat stress), availability, specific training requirements, a documented RPP, and user acceptance [Andrews et al. 2021]. Dust masks, surgical masks, bandanas, and clothes (wet or dry) do not protect the wearer from these particulate inhalation hazards [Fisher et al. 2020; Lindsley et al. 2021].

5.2 Control Recommendations Based Upon Exposure

5.2.1 Basis for the NIOSH Recommendations

NIOSH recommends using the EPA AQI for PM_{2.5} [EPA 2024a,b] to define exposure control categories (ECCs) to take actions to protect outdoor workers from potentially harmful exposure to wildland fire smoke (Table 5–1). ECCs are developed using an exposure assessment approach as outlined in Chapter 4. As described in Chapters 2 and 3, PM_{2.5} is considered the air pollutant of concern in wildfire smoke, with strong evidence of a causal relationship between both short- and long-term PM_{2.5} exposure and adverse health effects. NIOSH concludes that the scientific evidence cited in the National Ambient Air Quality Standards for Particulate Matter [EPA 2024a] and reviewed in Chapter 3 supports the need for employers to assess the potential exposure to wildland fire smoke to their workers and to be prepared to take actions to control exposures.

The EPA's AQI values, associated health categories, and breakpoints between those categories are based on the available scientific evidence of pollutant-related health effects [EPA 2024a]. See the AQI Category Breakpoints for PM_{2.5} section in EPA [2024a] for more information. Also see Figure 1-8 and Table 5-1, which show the AQI categories and breakpoints. These breakpoints were derived by the EPA using scientific judgement following extensive review and synthesis of the available information on health effects from PM_{2.5} exposures. Briefly, lower breakpoints (AQI values 50, 100 and 150) utilized the EPA's health-based primary PM_{2.5} annual and 24-hour standards. The first breakpoint (AQI value of 50) between the categories of "Good" and "Moderate" is based on the primary annual PM_{2.5} standard, which was revised in 2024 from 12 µg/m³ to 9 µg/m³ [EPA 2024a; EPA 2024b]. The primary annual PM_{2.5} standard is meant to protect the public including at-risk populations from adverse health effects from PM_{2.5} exposures experienced, on average, during the year. The next breakpoint (AQI value of 100) between the categories of "Moderate" and "Unhealthy for Sensitive Groups" is based on the primary 24-hour PM_{2.5}

1 standard (35 $\mu\text{g}/\text{m}^3$), which is unchanged in the EPA [2024a] rule. The 24-hour standard is
2 considered protective of the public including the most sensitive individuals from short-term
3 exposures to $\text{PM}_{2.5}$ concentrations that have the potential to result in adverse health effects.
4 The next breakpoint (AQI value of 150) between the “Unhealthy for Sensitive Groups” and
5 “Unhealthy” categories (55.4 $\mu\text{g}/\text{m}^3$) is also unchanged in the current standard [EPA 2024a].
6 It is based on a proportional increase in the breakpoint concentration at the AQI value of
7 100 (i.e., the 24-hour standard of 35 $\mu\text{g}/\text{m}^3$). The epidemiological studies evaluated by EPA
8 did not provide any new evidence to inform a revision to the breakpoint concentration at
9 the AQI value of 150 [EPA 2024a].

10 The remaining breakpoints are based on new information from the EPA [2024a] evaluation
11 of the scientific evidence on health effects across a range of exposure concentrations. Based
12 on its review of the evidence, the EPA set the breakpoint between the “Unhealthy” and
13 “Very Unhealthy” categories (AQI value of 200) at a daily (i.e., 24-hour average) $\text{PM}_{2.5}$
14 concentration of 125 $\mu\text{g}/\text{m}^3$. This concentration is in the lower range of concentrations that
15 consistently showed an association with increased cardiorespiratory effects in controlled
16 human exposure studies of young, healthy adults following short-term exposures (e.g., 2–3
17 hours). This is also within the range of the 5-day average and the maximum concentrations
18 in a study that reported significant associations between the $\text{PM}_{2.5}$ exposures and
19 respiratory-related healthcare encounters (emergency department visits, hospital
20 admissions, and outpatient visits) during a wildland fire smoke event in San Diego,
21 California in 2007 [EPA 2024a]. The breakpoint between the “Very Unhealthy” and
22 “Hazardous” categories (AQI value of 300) is set at a daily (i.e., 24-hour average) $\text{PM}_{2.5}$
23 concentration of 225 $\mu\text{g}/\text{m}^3$. This concentration falls between two key values: (1) the 2-
24 hour average concentrations associated with impaired vascular function and/or increased
25 blood pressure in controlled studies of healthy adults, and (2) the maximum 24-hour
26 average $\text{PM}_{2.5}$ concentrations on wildfire smoke days linked to increased all-cause
27 respiratory hospitalizations among adults 65 and older from 2008–2010 in 692 U.S.
28 counties [EPA 2024a]. AQI values of 301+ are in the “Hazardous” category and carry a
29 health warning of emergency conditions, where everyone is more likely to be affected at
30 these levels. See Airnow’s [AQI Basics](#) for more information.

31 While studies focusing on outdoor workers are limited (see Chapter 3), NIOSH considers the
32 studies cited by EPA as the basis for the AQI to be relevant since workers are part of the
33 general population including sensitive individuals. NIOSH considers the AQI category of
34 “Unhealthy for Sensitive Groups” (ECC 3) to be relevant to workers who are sensitive to
35 wildland fire smoke, and the AQI category of “Unhealthy” (ECC 4) to be relevant to all
36 workers. During a wildfire smoke event when the AQI for $\text{PM}_{2.5}$ is in these categories (ECC 3,
37 4, or higher), NIOSH recommends employers take actions to reduce exposures to wildland
38 fire smoke to their outdoor workers (Table 5-1). The AQI categories for $\text{PM}_{2.5}$ are based on a
39 24-hour average concentration, but NIOSH determined that 24-hour average concentrations
40 of $\text{PM}_{2.5}$ are relevant to workers since they may also be exposed to the wildland fire smoke
41 when they are not at work.

1 The EPA provides recommended actions for sensitive populations and general populations
2 to reduce their exposures to particulate pollution [EPA 2023b]. These EPA
3 recommendations provide a starting basis for the NIOSH recommendations in Table 5-1.
4 The EPA recommendations include limiting outdoor activities for sensitive populations at
5 AQI values of 101-150 (Unhealthy for Sensitive Populations) [EPA 2023b]. For the general
6 population, EPA recommends reducing outdoor exposures and taking more breaks during
7 outdoor activities at AQI values of 151-200 (“Unhealthy”). More stringent controls are
8 recommended by EPA as the AQI values increase, including avoiding all outdoor activities
9 for sensitive populations at AQI values of 201-300 (“Very Unhealthy”) and at AQI values of
10 301-500 (“Hazardous”) for the general population.

11 In the workplace, NIOSH recognizes that avoiding all outdoor activities during a high AQI
12 event is not always feasible for reasons such as a short agricultural harvesting window, or
13 law enforcement activities. Although removing workers from the hazardous environment is
14 the optimal control approach, other approaches based on engineering, administrative and
15 PPE solutions, using the hierarchy of controls paradigm are recommended as well (Table 5-
16 1). These options include moving activities indoors, rescheduling outdoor activities and
17 other controls. Additional options include preparations and training for wildfire smoke
18 events before they occur, exposure controls for sensitive workers when the AQI for PM_{2.5} is
19 “Unhealthy for Sensitive Groups”, and exposure controls for all outdoor workers when the
20 AQI for PM_{2.5} is “Unhealthy” during a wildland fire event. The hierarchy of controls has long
21 been used to provide multiple options for reducing workplace exposures (see Table 5–1)
22 [NIOSH 2011, 2013b, 2015, 2016a].

23 Some other federal and state agencies also use the AQI for PM_{2.5} to recommend or require
24 actions to reduce worker exposures to wildland fire smoke (see Section 5.2.3). NIOSH
25 reviewed these recommendations and requirements but did not base its recommendations
26 specifically on those of other agencies. All these agencies describe similar types of
27 recommendations, (e.g., preparedness and training, engineering or administrative controls,
28 and respirators). However, the AQIs associated with these recommendations may vary
29 across agencies (see Section 5.2.3).

30 **Preparation** is essential to reducing the health risks to outdoor workers in a wildland fire
31 smoke event [EPA 2021; NIOSH 2023b; OSHA 2024a]. The NIOSH recommended exposure
32 control options listed in Table 5–1 are intended to help employers and occupational safety
33 and health professionals to plan for such events. Employers should have in place a
34 workplace safety and health program that includes measures to protect their workers in the
35 event of unhealthy air quality due to a wildland fire smoke event. This program should
36 consider health equity considerations discussed in Section 2.4.

37 **Training**—Based on prudent occupational practices and similar to other occupational
38 hazards, NIOSH recommends that employers implement workplace hazard training (see
39 Section 5.1.2.2) before the AQI becomes unhealthy for workers. The training should explain
40 the measures for protecting all workers including sensitive groups (Table 5–1). Training
41 should also include information about voluntary or required respirator use, if applicable,

1 according to OSHA's Respiratory Protection standard, 29 CFR 1910.134 (see Section 5.1.3)
2 [OSHA 1998].

3 **Engineering controls**—NIOSH recommends that employers use the hierarchy of controls
4 (see Section 5.1) to mitigate exposures to wildland fire smoke to their outdoor workers
5 (Table 5–1). Employers should assess the use of engineering control options when the AQI
6 is Unhealthy for Sensitive Groups (ECC 3) as well as when the AQI is Unhealthy (ECC 4), for
7 all outdoor workers (see Section 5.1.1).

8 **Administrative controls**—NIOSH recommends that employers assess their administrative
9 control options when engineering controls are not feasible. Administrative controls should
10 be implemented for sensitive workers when the AQI is Unhealthy for Sensitive Groups (ECC
11 3) and for all outdoor workers when the AQI is Unhealthy (ECC 4) (Table 5–1).
12 Administrative control actions may include modifying, postponing, or relocating certain
13 work tasks when the air quality is unhealthy due to a wildland fire smoke event (see Section
14 5.1.2).

15 **Personal protective equipment**—NIOSH recommends that employers assess the needs
16 for PPE after performing a hazard assessment of the workplace [OSHA 2016] for outdoor
17 workers, when engineering or administrative controls are not feasible (Table 5–1). NIOSH
18 notes that OSHA requires employers to provide respiratory protection to their employees if
19 other measures are not sufficient to control a respiratory hazard [OSHA 1998]. Respirators
20 are recommended when engineering and administrative controls do not sufficiently reduce
21 exposures. NIOSH Approved® respirators such as the N95® are recommended for sensitive
22 workers when the AQI is Unhealthy for Sensitive Groups (ECC 3) and for all outdoor
23 workers when the AQI is Unhealthy (ECC 4) (Table 5–1), in accordance with the OSHA
24 requirements [OSHA 2016] (see Section 5.1.3). Consistent with preparation
25 recommendations above, NIOSH recommends that employers assess the respiratory
26 protection needs for their outdoor workers prior to wildland fire smoke events.

27 **Summary of control options**—NIOSH has previously provided practical steps that
28 employers and workers can take to reduce exposure to wildland fire smoke [NIOSH 2023b].
29 NIOSH is expanding this guidance for outdoor workers based on its evaluation of the health
30 effects literature (Chapter 3) and on the available exposure control options for wildland fire
31 smoke within the hierarchy of controls (see Section 5.1). These NIOSH recommendations
32 are intended to help employers protect the health of their outdoor workers during wildfire
33 smoke events.

Table 5–1. Exposure control categories with NIOSH exposure control recommendations for employers to use to protect outdoor workers from wildland fire smoke

Exposure control recommendations	Good	Moderate	Unhealthy for sensitive groups	Unhealthy	Very unhealthy	Hazardous
	1 0.0–9.0 µg/m ³ *	2 9.1–35.4 µg/m ³ *	3 35.5–55.4 µg/m ³ *	4 55.5–125.4 µg/m ³ *	5 125.5–225.4 µg/m ³ *	6 ≥ 225.5 µg/m ³ *
Training and preparation	Standard workplace hazard training, including procedures for wildland fire smoke event†	Standard workplace hazard training, including procedures for wildland fire smoke event†	Standard workplace hazard training, including procedures for wildland fire smoke event† and respirator fit training as needed for sensitive outdoor workers	Standard workplace hazard training, including procedures for wildland fire smoke event† and respirator fit training as needed for all outdoor workers	Standard workplace hazard training, including procedures for wildland fire smoke event† and respirator fit training as needed for all outdoor workers	Standard workplace hazard training, including procedures for wildland fire smoke event† and respirator fit training as needed for all outdoor workers
Assess engineering control options (Section 5.1)	—	—	Engineering controls for sensitive outdoor workers	Engineering controls for all outdoor workers	Engineering controls for all outdoor workers	Engineering controls for all outdoor workers
	<ul style="list-style-type: none"> • Enclosed structures with filtered air • Indoor structures <ul style="list-style-type: none"> ○ Close windows and doors ○ Use air filtration ○ Recirculate air 					

Exposure control recommendations	Good 1 0.0–9.0 µg/m ³ *	Moderate 2 9.1–35.4 µg/m ³ *	Unhealthy for sensitive groups 3 35.5–55.4 µg/m ³ *	Unhealthy 4 55.5–125.4 µg/m ³ *	Very unhealthy 5 125.5–225.4 µg/m ³ *	Hazardous 6 ≥ 225.5 µg/m ³ *
If engineering controls are not feasible, assess administrative control options:	—	—	Administrative controls for sensitive outdoor workers	Administrative controls for all outdoor workers	Administrative controls for all outdoor workers	Administrative controls for all outdoor workers
<ul style="list-style-type: none"> • Relocate or reschedule work • Reduce physical activity • Take more frequent breaks • Move to indoor duties 						
If engineering and administrative controls are not feasible assess personal protective equipment (PPE) options, including use of respirators [†]	—	—	Provide respirators to sensitive outdoor workers in accordance with OSHA requirements (29 CFR 1910.134)	Provide respirators to all outdoor workers in accordance with OSHA requirements (29 CFR 1910.134)	Provide respirators to all outdoor workers in accordance with OSHA requirements (29 CFR 1910.134)	Provide respirators to all outdoor workers in accordance with OSHA requirements (29 CFR 1910.134)

* PM_{2.5} concentration consistent with the AQI and PM_{2.5} values announced by the EPA on March 6, 2024 [EPA 2024a,b].

† Standard workplace hazard training for outdoor workers should include basic information and training on using respirators, as identified in the employer’s workplace health and safety plan. If needed, respirator fit testing should be performed before wildland fire smoke events occur in accordance with applicable OSHA requirements, 29 CFR 1910.134 [OSHA 1998]. Employers with workers engaged in agricultural operations (e.g., farmworkers) are covered by OSHA’s safety and health standards for agriculture in 29 CFR Part 1928 and must also comply with the General Duty Clause of the Occupational Safety and Health Act of 1970 (OSH Act). This requires employers to provide their employees “employment and a place of employment which are free from recognized hazards that are causing or are likely to cause death or serious physical harm” 29 USC 654(a)(1).

‡ When respirator use is required, employers covered by OSHA’s Respiratory Protection standard, 29 CFR 1910.134, must comply with the standard, including implementing, when applicable, a full respiratory protection program. Employers with workers engaged in agricultural operations (e.g., farmworkers) are covered by OSHA’s safety and health standards for agriculture in 29 CFR Part 1928 and must also comply with the General Duty Clause of the Occupational Safety and Health Act of 1970 (OSH Act). This requires employers to provide their employees “employment and a place of employment which are free from recognized hazards that are causing or are likely to cause death or serious physical harm” 29 USC 654(a)(1). When respirators are used on a voluntary basis, employers should follow the requirements for the voluntary use of respirators in the workplace, including providing employees with a copy of Appendix D of 29 CFR 1910.134 “Information for Employees Using Respirators When Not Required Under the Standard,” [OSHA 1998].

5.2.2 NIOSH Recommendations for Employers, Workers, and Healthcare Professionals

Employers and employees should prepare for the potential for wildland fire smoke affecting outdoor work in their area. Recent wildland fires in the United States and Canada have impacted many regions across the United States (see Section 1.2). Wildland fire smoke has also influenced the average annual PM_{2.5} concentrations in most states [Austin et al. 2021]. Given these trends, NIOSH recommends that employers should evaluate how to protect their outdoor workers from wildland fire smoke. Employers should provide the tools necessary for employees to learn how to assess their own health risks and how and when to use protective measures or seek medical care.

5.2.2.1 Employers

- Evaluate the potential for a wildland fire smoke event affecting the areas where your employees are working outdoors. Consider any recent events in your area or similar areas. Consider how your employees would be affected by a wildland fire smoke event and what tools are available to mitigate exposures.
- Develop a general safety and health program for your employees, if not already available, including hazard assessment and plans for potential wildland fire smoke events [NIOSH, OSHA 2022; OSHA, no date].
- Develop an exposure assessment strategy (see Chapter 4) that takes into account weather events and wildland fires that may impact the air quality of your outdoor workers.
- Conduct exposure assessments using the tiered approach outlined in Chapter 4. Exposure assessment may be necessary to communicate hazards, identify exposure controls, and plan for emergency response.
- Communicate to workers the current air quality conditions and the available workplace protective measures as needed.
- Implement the hierarchy of controls options as needed for controlling worker exposures to wildland fire smoke (see Table 5–1) based upon the exposure control categories (see Section 5.1)
 - Preparedness is key to effective exposure control. Preparations include a written Health and Safety plan, training for workers and their supervisors, and the acquisition and maintenance of suitable exposure controls (see Section 5.1).
 - Engineering control options include moving workers into enclosed buildings or structures where the air is filtered, keeping doors and windows closed, and using higher efficiency filters in HVAC systems and portable air filters (see Section 5.1.1).
 - Administrative control options include relocation of workers, reduction of shift length, rotation of workers, work-rest cycles, and reduction of work intensity or rescheduling high-intensity work. Additionally, a means of

1 removing contaminants from the skin during and after the work shift should
2 be considered (see Section 5.1.2).

- 3 ○ Education and training for workers and their supervisors are essential to the
4 effective implementation of worker health and safety practices (see Section
5 5.1.2).
- 6 ○ PPE to protect outdoor workers from wildland fire smoke may include use
7 of NIOSH Approved® respirators. When engineering and administrative
8 control options are not feasible or do not adequately lower exposures, OSHA
9 requires employers to perform a hazard assessment of the workplace [OSHA
10 2016], and when indicated, provide NIOSH Approved® respirators to their
11 employees who must work outdoors (see Section 5.1.3).

- 12 ● Be prepared for and allow employees to seek medical care if they experience signs
13 or symptoms of injury or illness due to wildfire smoke exposure.

14 **5.2.2.2 Workers**

- 15 ● Talk with your employer about their plan for a wildland fire smoke event in your
16 work area. Learn what to do and what tools are available if your work is affected by
17 a wildland fire smoke event.
- 18 ● Understand the workplace exposure assessment resources and various exposure
19 control categories when planning to work outdoors. Follow the health advice for
20 your health risk and the exposure assessment category.
- 21 ● If you have asthma or other lung or heart disease, follow your health care provider's
22 directions for taking your medicines and follow your asthma management plan.
23 Contact your health care provider if symptoms worsen (see Section 5.3.2). Identify
24 cleaner spaces where you could go if exposure levels are hazardous for you.
- 25 ● Take the workplace training to learn about the adverse health effects of exposure to
26 wildland fire smoke and the procedures in place to work safely during a wildland
27 fire smoke event.
 - 28 ○ Your employer may ask you to take certain actions to reduce your exposure
29 to wildland fire smoke. These actions may include moving work tasks
30 indoors, adjusting work schedules or tasks, and using a respirator on a
31 voluntary or required basis with appropriate training (see Section 5.1).
 - 32 ○ Learn how to correctly use a respirator during the workplace training. Your
33 employer should provide you with the information, training, and resources
34 you need. More information is available in NIOSH's [Respirator Fact Sheet](#).
35 Learn how to care for and maintain the respirator.
 - 36 ○ Do not use bandanas, clothes (wet or dry), or surgical or dust masks that are
37 not approved by NIOSH. These face coverings will not protect you from
38 inhaling the particulates in wildland fire smoke.
- 39 ● Know the rules and regulations to safely perform your job and who to contact with
40 any concerns (see OSHA's [Worker Rights and Protections](#)).
- 41 ● Wash contaminated skin before eating or drinking and at the end of the work shift.
42 Also change clothing or wash all contaminated clothing before returning home, or as

1 soon as possible. When possible, take a shower, including thoroughly washing hair
2 to remove contaminants, as soon as possible following outdoor work.

3 **5.2.2.3 Healthcare Professionals**

4 NIOSH recommends that qualified healthcare professionals should be trained in how to
5 respond to workers affected by wildland fire smoke events. Healthcare professionals need
6 to understand the chronic conditions that may be exacerbated by exposure to wildland fire
7 smoke, the acute health risks of short- and long-term exposure to wildland fire smoke (see
8 Chapter 3), and any appropriate medical treatments. They should also be ready to respond
9 to workers who may ask them for recommendations on how to reduce health effects from
10 exposure. The Centers for Disease Control and Prevention (CDC) provides education
11 resources for healthcare professionals (see CDC's [Resources for Professionals](#)).

12 **5.2.3 Other Federal or State Recommendations** 13 **or Requirements**

14 The EPA has evaluated the scientific evidence of particulate air pollution including wildland
15 fire smoke [EPA 2019]. The EPA [2021; 2024c] states that outdoor workers may be at
16 greater risk for adverse health impacts of wildland fire smoke due to their “extended
17 periods of time exposed to high concentrations of wildfire smoke.” Outdoor workers may
18 also include those in other sensitive groups such as people with asthma or other respiratory
19 diseases, people with cardiovascular disease, children under 18 years old, pregnant people,
20 older adults, and people of low socio-economic status [EPA 2024c]. Thus, many of the EPA
21 recommendations regarding PM_{2.5} for the public can apply to outdoor workers [EPA 2021].
22 Other recommendations to the general public on reducing exposures to wildland fire smoke
23 may not be relevant to outdoor workers who must continue to work [EPA 2021].

24 The EPA provides a general list of health effects and advisories based on the AQI [Table 4 in
25 EPA 2021]. They also provide recommended actions for public health officials to consider at
26 the different AQI categories [Table 5 in EPA 2021]. Those actions include considering
27 curtailing outdoor work activities “unless the workers have a fully implemented respirator
28 plan in place and clean air respite breaks” when the AQI is Hazardous (AQI>300) [Table 5 in
29 EPA 2021]. The EPA also provides general guidance for outdoor workers that is not specific
30 to the AQI but can be used to reduce exposure to wildland fire smoke, including engineering
31 and administrative controls [EPA 2021].

32 The [OSH Act of 1970](#), which created OSHA, requires employers to provide their employees
33 with working conditions that are free of recognized hazards. OSHA also provides
34 information, training, and assistance to employers and workers. OSHA does not have a
35 specific standard for wildland fire smoke, but other OSHA standards and requirements may
36 apply for protecting workers from wildland fire smoke hazards. In the absence of an
37 applicable OSHA standard, Section 5(a)(1) of the OSH Act requires that employers protect
38 all workers against recognized hazards. In addition, some OSHA State Plan states have
39 specific requirements to protect workers from wildland fire smoke. Apart from hazard-
40 specific requirements, OSHA’s requirements for Hazard Communication [OSHA 2024b] and

1 Respiratory Protection [OSHA 1998] should be part of an overall workplace safety and
2 health plan.

3 The U.S. Geological Survey provides guidance to its field employees that includes
4 consideration of the AQI [USGS 2023]. Centers of the USGS can employ administrative
5 controls when the AQI is Unhealthy. When the AQI is Hazardous, administrative controls are
6 strongly recommended, and outdoor work should be cancelled with exception for
7 emergencies [USGS 2023].

8 Employers and workers should be aware of the requirements in their individual states. For
9 example, as of the publication of this document, three OSHA State Plan states in the West—
10 California, Oregon, and Washington—have specific requirements to protect workers from
11 wildland fire smoke. Certain requirements are triggered by the AQI and PM_{2.5} concentration.
12 The required actions for employers include providing information and training to workers
13 and supervisors, providing workers with cleaner indoor work environments, reducing the
14 amount of time workers are working outdoors during poor air quality days, providing
15 NIOSH Approved® respirators, and having provisions for emergency medical services
16 [California Code of Regulations 2019; Oregon Administrative Rules 2022a,b; Washington
17 Administrative Code 2024]. See California OSHA's [Worker Protection from Wildfire Smoke](#),
18 Oregon OSHA's [Wildfires](#), and Washington state's [Wildfire Smoke](#) for more information on
19 the individual state requirements. The lowest AQI threshold that requires employers to take
20 actions varies by state: Washington is 69, Oregon is 101, and California is 151 (for further
21 information, see Section 1.4.3.3).

22 The National Academy of Sciences (NAS) provided extensive guidance on measuring and
23 controlling exposures to PM_{2.5}, including wildland fire smoke, in indoor environments [NAS
24 2024]. NAS identified several factors associated with health inequity in the availability and
25 feasibility of utilizing exposure control measures, especially in marginalized populations.
26 Many of these disparities may impact outdoor worker populations. Opportunities to
27 address these disparities and increase access to measures to protect outdoor workers from
28 exposure to wildland fire smoke will reduce the health burden among those workers. See
29 Section 1.4 for more information on the activities of other agencies within and outside the
30 United States related to protecting workers from adverse health effects of exposure to
31 wildland fire smoke.

32 **5.3 Medical Surveillance and Medical Monitoring**

33 Medical monitoring programs and medical surveillance programs are related concepts that
34 require definition, as they are often used interchangeably. Medical monitoring, sometimes
35 referred to as medical screening, involves cross-sectional medical examinations with the
36 primary goals of early detection of work exposures, early identification of work-related
37 disease, and/or identification of high-risk worker populations. Medical surveillance, on the
38 other hand, is the systemic analysis of aggregate longitudinal data from potentially exposed
39 workers, often with repeated, scheduled medical examinations, to identify relationships
40 between job tasks, exposures, and health effects in a worker population over time. Both
41 medical monitoring and medical surveillance programs can involve medical questionnaires,

1 medical examinations, and medical testing, and both can be administered by qualified
2 health professionals. The programs serve as risk mitigation tools for primary and secondary
3 prevention of worker health effects [Gochfeld 1992; Trout 2011; Weissman 2014]. At this
4 time, insufficient evidence is available to recommend a full-scale wildland fire medical
5 monitoring or surveillance program with the goal of identifying occupational disease caused
6 by wildland fire smoke for all outdoor workers. However, one or both may serve as
7 elements of a comprehensive risk management plan when hazardous occupational
8 exposures cannot be eliminated by other occupational controls.

9 The decision for if or when to implement a medical surveillance or medical monitoring
10 program is discussed in detail in Weissman [2014] and includes a variety of factors such as
11 a scientific understanding of the risk of the exposure's health effects, anticipated dose to the
12 worker population, and cost-benefit analyses. Medical surveillance and treatment programs
13 have been established in response to a single disaster or after workers developed
14 symptoms of negative health effects [Calvert et al. 2023; Kreiss et al. 2002; NIOSH 2016a].
15 These programs were implemented as tools for secondary prevention years after worker
16 exposures. Few medical surveillance programs have been implemented prior to significant
17 health hazards to the worker population. An exception is the call for medical surveillance of
18 workers exposed to carbon nanotubules and nanofibers due to respiratory risks identified
19 in animal toxicologic studies [Guseva Canu et al. 2020; NIOSH 2013a]. The goal of a medical
20 surveillance program started prior to significant negative occupational health effects is to
21 serve as a sentinel warning system, offering the opportunity to implement occupational
22 controls for primary prevention and eliminating future negative health effects [Weissman
23 2014]. Literature has shown that medical surveillance programs for other health hazards
24 (e.g., work related asthma) can lead to improved worker medical outcomes as well as
25 socioeconomic benefits for the employer [Wilken et al. 2012].

26 For wildland fire smoke, medical monitoring programs are in use for wildland firefighters
27 as part of the Department of Interior medical qualification standards [Medical qualification
28 determinations, 2024], but examples of medical surveillance or medical monitoring
29 programs specific to wildland fire smoke in other outdoor worker populations have not
30 been found.

31 The health effects review outlined in Chapter 3 identified a causal relationship between
32 short-term exposure to PM_{2.5}, non-accidental mortality, and cardiovascular health effects
33 (e.g., ischemic heart disease, arrhythmias, heart failure). Similarly, the review identified a
34 likely causal relationship between short term exposure to PM_{2.5} and respiratory health
35 effects, especially among those with asthma and chronic obstructive pulmonary disease
36 [Rice et al. 2021]. Other health effects (e.g., reproductive and developmental effects,
37 neurologic effects, metabolic effects, as well as cancer and infectious disease) were
38 associated with wildland fire smoke exposure but lacked sufficient evidence to be called
39 causal (see Section 3.4).

40 These conclusions include limitations that are described extensively in Chapter 3 but
41 summarized here. The data for the review mostly came from studies in the general
42 population; some of the PM_{2.5} exposure source data was from general air pollution instead

1 of wildland fire-specific sources; and very few studies included the outdoor worker
2 population defined for this hazard review. Additionally, there is not a current occupational
3 exposure limit for wildland fire smoke exposure, and many of the adverse health outcomes
4 (e.g., respiratory and cardiovascular disease) are common outside of the workplace. Despite
5 these limitations, Chapter 3 stated that outdoor workers occupationally exposed to
6 wildland fire smoke are likely at risk for multiple adverse health effects.

7 As research into hazards of wildland fire smoke in outdoor workers continues,
8 reassessment of available evidence may offer more specific recommendations in the future.

9 While not broadly recommended, medical monitoring or surveillance could currently serve
10 as a tool in a comprehensive risk management plan for specific indications. For example, a
11 medical monitoring program could identify at-risk populations (e.g., workers with existing
12 cardiovascular or pulmonary conditions) that could benefit from occupational controls;
13 medical monitoring programs could assess the effectiveness of previously implemented
14 occupational controls; or a medical monitoring program could serve as part of an outdoor
15 worker wildland fire smoke emergency response plan.

16 Appendix D offers key considerations for working partners (e.g., medical professionals,
17 occupational safety specialists, employers, unions) who wish to explore development of a
18 wildland fire smoke medical surveillance or monitoring program as part of a
19 comprehensive health and safety plan.

20 **5.3.1 Biomonitoring**

21 Biomonitoring is the standardized measurement of environmental contaminants or their
22 breakdown products (metabolites) to assess human exposure. Biomarkers can be used as a
23 tool to determine occupational exposures and are usually tested in urine, saliva, or blood
24 samples.

25 Recent studies in firefighters have investigated biomarker surrogates for smoke exposure
26 including urinary levoglucosan [Naehler et al. 2013; Navarro et al. 2023] and urinary
27 metabolites of polycyclic aromatic hydrocarbons (PAHs) [Gill et al. 2019]. Currently there
28 are no universally accepted biomarkers for wildland fire smoke. The American Conference
29 of Governmental Industrial Hygienists publishes biological exposure indices (BEIs), which
30 provide a concentration below which nearly all workers should not experience health
31 effects. There is a BEI for PAH 1-Hydroxypyrene in urine (2.5 µg/L). However, a limitation
32 exists: PAHs can originate from various exposure sources, and background levels may exist
33 in the population [ACGIH 2023]. This limits the ability to use this BEI as an exclusive marker
34 of wildland fire smoke.

35 At this time, the data is lacking to recommend biomarkers as a specific part of a risk
36 management plan, although further research, especially in high-risk occupations, on the use
37 and effectiveness of biomonitoring for wildland fire smoke, would be beneficial.

5.3.2 When to Seek Medical Attention

Having an emergency medical plan is important for the safety of all workers and is the same for any health event, regardless of whether it is precipitated by wildland fire smoke. Any worker experiencing symptoms should be permitted to be evaluated by a qualified healthcare professional.

Respiratory symptoms that may require evaluation include trouble breathing, shortness of breath, cough that will not stop, or severe wheezing. Be aware that some respiratory effects due to wildland fire smoke may be delayed, and symptoms could begin hours to days after the exposure [EPA 2019]. Cardiovascular symptoms may include chest pain, difficulty breathing, syncope (passing out), and signs or symptoms of a stroke or heart attack. Severe cardiovascular or respiratory symptoms may necessitate calling 911 or transport to an emergency department. After evaluation and treatment by a trained medical professional, a return-to-work timeline should be coordinated with employers and include evaluation of occupational controls to prevent future health exposures.

5.4 Summary

NIOSH recommends using the EPA AQI for PM_{2.5} [EPA 2024a,b] to define exposure control categories to take actions to protect outdoor workers from potentially harmful exposure to wildland fire smoke. Outdoor workers are an at-risk group due to their potential exposure to wildland fire smoke at high concentrations and durations, including from sources many miles away. The EPA Air Quality Guide for Particle Pollution recommendations for AQI and the hierarchy of controls forms the basis of NIOSH worker protection recommendations to reduce wildland fire smoke exposure to outdoor workers [EPA 2023b]. These recommendations should be incorporated as part of an overall occupational safety and health program. This includes assessing hazards at individual worksites to protect workers and taking steps to protect workers from these hazards. For wildland fire smoke events, employers should adequately prepare for and train their workforce for these events. When events occur, estimates of exposure should be made as described in Chapter 4, and then, as appropriate, the hierarchy of controls should be applied in order to protect the workforce. This includes the following options, applied in Table 5-1:

- Engineering controls can be applied through the use of enclosed buildings, temporary or permanent structures, and vehicles, where filtered air is provided to reduce workers' exposure.
- Administrative controls begin with preparation for wildland fire smoke events along with worker training and education. Exposures can be managed with a variety of administrative control approaches, including moving work indoors, rescheduling outdoor work, relocation, reduction of shift length, rotation of workers, work-rest cycles, and a reduction of work intensity.
- Personal protective equipment, such as NIOSH Approved N95[®] respirators (when selected and used properly as part of a respiratory protection program) can protect against inhalation hazards from wildland fire smoke.

1 Workers should be allowed to seek medical care if they experience signs or symptoms of
2 injury or illness due to wildland fire smoke exposure. Healthcare professionals should
3 understand the chronic conditions that may be exacerbated by wildland fire smoke
4 exposure. While not broadly recommended, medical monitoring or surveillance in some
5 situations may best serve as a tool in a comprehensive risk management plan for specific
6 indications, such as identifying at-risk populations that may benefit from layered
7 occupational controls or as part of an outdoor worker wildland fire smoke emergency
8 response plan. Elements of a medical surveillance or monitoring program for wildland fire
9 smoke exposure are explored in Appendix D.

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Chapter 6: Research Needs

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Research needs to address health hazards from wildland fire smoke were previously described in several sections of this hazard review. This chapter provides an overview of previously identified gaps in knowledge on: (1) the characterization of wildland fire smoke, (2) field studies and exposure assessment, (3) health effects from occupational exposure to wildland fire smoke exposure, (4) hazard control and prevention measures needed for worker protection, and (5) health equity research needs.

9

This hazard review has relied on extending relationships between ambient air pollution, primarily measured as PM_{2.5} exposure, and adverse human health conditions to risks expected among outdoor workers exposed to wildland fire smoke. This approach is complemented by a rapidly growing literature describing health burdens in multiple populations affected by major wildfires, as evidenced by the scoping review in Chapter 3. Still, there is a stark absence of direct information from studies of outdoor working populations, such as workers in farming, forestry, oil and gas, landscaping, transportation, maintenance, and construction. Inherent differences in the exposed populations and exposure characteristics (e.g., toxicity, intensity, duration) have not been thoroughly investigated; therefore, questions on external validity remain. Future interdisciplinary research that directly investigates occupational health risks from exposures to wildfire emissions among all affected workers would be beneficial.

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This hazard review responds to a growing awareness of the potential health risks among outdoor workers exposed to wildland fire smoke. As more is learned about the risks, the need for effective interventions becomes apparent. The National Institute for Occupational Safety and Health (NIOSH) recommends using the hierarchy of controls to reduce worker exposures; however, it also recognizes the challenges imposed by outdoor work environments that may limit control options. Limited development has occurred in control technologies and risk mitigation strategies that target the protection of outdoor workers

1 exposed to wildland fire smoke in varied work environments. Research supporting the
2 innovative development and application of effective interventions would be beneficial.

3 Although strong evidence of health risks from exposure to wildland fire smoke exists, there
4 is little information on factors that may modify causal relationships. For example, outdoor
5 workers as a group experience other occupational hazards (e.g., heat, pesticide exposure)
6 that may work synergistically with inhalation of wildland fire smoke; therefore, research
7 would help to understand joint effects. Moreover, risk differences between and within
8 affected occupational groups may need to be investigated. For example, among outdoor
9 workers, migrant farmworkers may experience language barriers and other cultural or
10 societal differences that increase their risk of serious health effects. These are a few
11 examples where characteristics of the workers and their exposures may have large impacts
12 on the adverse effects they experience.

13 To further illustrate potential research needs, some prominent research questions are
14 presented below. This information should not be considered comprehensive, nor is it
15 presented in order of priority. NIOSH recognizes that research needs may include
16 investigating occupational health hazards from wildfire stressors other than wildland fire
17 smoke. Although not the focus of this hazard review, some research gaps regarding these
18 health hazards and stressors have been included in brief descriptions below.

19 **6.1 Research Needs Related to the Characterization** 20 **of Wildland Fire Smoke and Related Exposures**

21 This hazard review uses PM_{2.5} as a surrogate for wildland fire smoke exposure. Strong
22 evidence suggests this is a reasonable assumption, but questions remain regarding the
23 toxicity of key components of wildland fire smoke, including chemical toxicants and the
24 effects of other particle sizes. Research questions related to this include the following:

- 25 • How can smoke mixtures be better defined to evaluate the primary components or
26 mixtures contributing to the various pulmonary and systemic effects following
27 inhalation exposure?
- 28 • How can wildland fire smoke exposures to outdoor workers be better characterized
29 and predicted with consideration of weather, distance, source, and type of wildland
30 fire and other variations of the outdoor work environment? This includes improving
31 modeling and forecasting of smoke constituents, such as PM_{2.5} concentrations.
- 32 • What improvements can be made to sampling and analytical methods used to assess
33 wildland fire smoke exposures, especially in rural areas where farmworkers and
34 other outdoor workers are employed? Considerations should include enhancing the
35 availability and field use of direct reading instruments, and improving sampling
36 techniques for better cost-effectiveness, timeliness and reliability of results, and
37 lower detection limits.
- 38 • What methods or tools can be developed to differentiate exposures from wildland
39 fires from other occupational and non-occupational sources?

- How can integration of data systems, modeling, and sampling methods be improved? This integration can improve the accuracy and confidence in exposure estimates.

6.2 Field Studies and Exposure Assessment Research Needs

Understanding the exposures and health effects of farmworkers and other outdoor workers exposed to wildland fire smoke is essential to determine what types of interventions would be most effective to protect workers. Research questions that address these topics include the following:

- How do we differentiate variations in exposure levels or types of exposures among individuals or groups that are part of a similar exposure group, and account for frequency and duration of exposures, and varying work characteristics (e.g., distance from fire, weather variations, control measures, personal protective equipment [PPE] use)?
- What health- or technology-based refinements to occupational exposure limits for wildland fire smoke constituents are needed?
- Among hazardous agents typically comprising wildland fire smoke, is $PM_{2.5}$ the best indicator of potential health risks from exposure? What other metrics of exposure to wildland fire smoke can be used and compared to $PM_{2.5}$ as a predictor of adverse health effects and to inform exposure control decision-making?
- What factors impact the level of smoke contamination on clothing and PPE for farmworkers and other outdoor workers, and how does that affect health risks from take-home exposure?

6.3 Health Effects Research Needs

The processes underlying wildland fire smoke-related short- and long-term adverse health effects need additional study. More information is needed on the differences in health effects by particle size and smoke components. Along with physical health effects, interest is growing in other occupational hazards from wildfires, such as infectious disease and adverse mental health effects. Research investigating the natural history of these health effects can also inform prevention measures. Understanding the distribution of adverse health effects across populations with different characteristics can help tailor intervention and risk communication strategies. Research questions related to health effects include:

- How do attributable acute and chronic physical and mental health effects differ among various affected occupational groups, such as farmworkers, forestry, and construction workers?
- Along with exposures attributable to wildland fires, outdoor workers are exposed to numerous other physical, chemical, biological, and psychosocial stressors in the workplace. What are the health impacts of these exposures separately and jointly

1 with wildland fire smoke? How do these different exposures influence risk
2 mitigation strategies?

- 3 • What conditions modify the exposure-response association between occupational
4 exposures to wildland fire smoke and acute, late, and chronic health effects? This
5 includes considering any modifying effects of sex, age, race, genetics, comorbidities,
6 co-exposures (occupational, environmental, and behavioral), health inequities, and
7 social determinants of health among exposed workers. Other etiologic factors
8 lacking information include:
 - 9 ○ Timing of onset (latency, acute vs. late-onset diseases)
 - 10 ○ Risk persistence (time since exposure)
 - 11 ○ Effects of exposure duration (acute, fractionated, or protracted), intensity (high
12 vs. low concentration) and route (e.g., inhalation vs. dermal)
 - 13 ○ Effects of smoke-related exposure hazards other than particulate matter
- 14 • What are the links between components in wildland fire smoke mixtures and
15 adverse health outcomes, and how does this information influence exposure
16 controls?
- 17 • How can acute and chronic characteristics of toxicities and their resulting
18 downstream activation be determined after smoke exposure from different sources
19 (e.g., wildland urban interface [WUI] fires, prescribed fires)? What toxicity testing
20 methods can be developed to accurately assess the potential risks posed by various
21 environmental pollutants [Black et al. 2017]?
- 22 • Are there adverse health effects associated with subsequent exposure to fire-altered
23 materials in soils and plants, or the deposition of ash and chemicals from smoke in
24 affected areas?
- 25 • In addition to physical health hazards among outdoor workers that may be
26 associated with wildland fire smoke, other research questions on potential
27 occupational health hazards should be investigated. For example:
 - 28 ○ What is the risk of adverse health effects among outdoor workers resulting from
29 exposures to contamination from fire retardants used in wildland firefighting?
 - 30 ○ What is the risk of adverse mental health or well-being among outdoor workers
31 resulting from exposures to wildland fire smoke and other wildland fire
32 stressors?

33 **6.4 Hazard Control and Prevention Research Needs**

34 Mitigating the hazards of wildland fire smoke exposure poses unique challenges. Using the
35 hierarchy of controls would suggest controlling exposures at the source, but because of the
36 setting and circumstances of exposure, the engineering control strategies are limited.
37 Because of the rural nature of many of the work settings, additional barriers may exist to
38 effectively implementing control strategies. Research questions around hazard control and
39 prevention include the following:

- 40 • What are the barriers and opportunities to implementing health and safety
41 programs that protect farmworkers and other outdoor workers from adverse health

1 effects of exposure to wildland fire smoke? This includes identifying effective
2 controls and incentives that motivate employees and supervisors when it comes to
3 monitoring, education or training, and communication.

- 4 • How effective are existing engineering control and administrative options that
5 reduce exposure to wildland fire smoke in farmworkers and other outdoor
6 workers? For example:
 - 7 ○ Research into the development of cost-effective portable and scalable methods
8 of control for wildland fire smoke events.
 - 9 ○ Research targeting the development and feasibility of temporary enclosures
10 (with air cleaning and temperature control) as an option for providing field-
11 portable clean air locations.
 - 12 ○ Research on the effectiveness of economically feasible (or optimized) options
13 for reducing building indoor PM_{2.5} concentrations (e.g., upgraded building HVAC
14 filtration, low-cost DIY filtration, portable commercial air cleaners).
 - 15 ○ Research on the effectiveness of industrial vehicle cab filters against wildland
16 fire smoke.
- 17 • What are the most effective guidance and training programs for protecting against
18 adverse health effects from wildland fire smoke exposure?
- 19 • What are the effects of medical monitoring and surveillance programs on wildland
20 fire smoke health outcomes?
- 21 • What is the ideal timeline for implementing medical monitoring or surveillance,
22 taking into consideration health outcomes and cost-benefit analyses?
- 23 • What biomarker exposure limits have been established for smoke that correspond
24 to health outcomes (e.g., biological exposure indices)?
- 25 • How can the development and effective use of respiratory protection be advanced
26 for farmworkers and other outdoor workers? This includes consideration for
27 effective and affordable PPE for use under sustained difficult environmental
28 conditions (e.g., heat, increased physical activity levels).

29 **6.5 Health Equity Research Needs**

30 Social determinants and structural disadvantages of health may place some outdoor
31 workers at increased risk of wildland fire smoke exposures. They also can limit outdoor
32 workers' access to worker protections and hinder their ability to cope with adverse
33 consequences of an injury or illness. Further research related to health inequities would
34 increase our understanding of effective partnerships, policies, interventions, and
35 communication strategies to better protect these worker populations. Research questions
36 related to health equity include the following:

- 37 • How do we better understand the impact of non-chemical stressors and social
38 determinants/structural disadvantages of health on the relationship between
39 exposure and the health effects of wildland fire smoke [Cascio 2018]?

- 1 • How do we ensure that health data collection tools and methods are inclusive of
2 individuals from groups that are underrepresented in public health research [Flynn
3 et al. 2021; Rodriguez-Lainz et al. 2018]?
- 4 • What are the social determinants and structural disadvantages that contribute to
5 the inequities for workers from groups at disproportionate risk of wildland fire
6 smoke exposure and how should policies, programs, and interventions be designed
7 to mitigate them?
- 8 • What are the health effects of wildland fire smoke on worker populations at higher
9 risk (e.g., people with diabetes, pregnant workers, young workers, older workers)?
- 10 • How can we better identify and understand the overlapping structural
11 disadvantages that place some populations at higher risk (e.g., socioeconomically
12 disadvantaged) and how can public policy, industry practices, job arrangements,
13 and public health practices be revised to reduce these structural disadvantages?

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Appendix A — Health Effects Scoping Review

Appendix A includes the full list of references that were synthesized for the scoping review on epidemiologic studies on the health effects of wildland fire smoke in Chapter 3, Section 3.2 (n=175 studies). While Section 3.2 discusses aggregate findings across the literature, Appendix A lists all individual publications, first grouped by health outcome category with in-text citations (A1–A10), followed by a list of full reference information (A11).

A1 Studies on Respiratory Health

[Aguilera et al. 2021; Aguilera et al. 2020; Augusto et al. 2020; Barbosa et al. 2022; Beyene et al. 2022a; Beyene et al. 2022b; Blando et al. 2022; Casey et al. 2021; Chen et al. 2023a; Chen et al. 2024; Chen et al. 2021; Chen et al. 2023b; Cherry et al. 2021; de Souza Fernandes Duarte et al. 2023; Doubleday et al. 2020; Doubleday et al. 2023; Douglas-Vail et al. 2023; Duncan et al. 2023; Ferguson et al. 2017; Gan et al. 2017; Gan et al. 2020; Gao et al. 2023; Gianniou et al. 2018; Gould et al. 2024; Hahn et al. 2021; Heaney et al. 2022; Heft-Neal et al. 2023; Hong et al. 2017; Howard et al. 2021; Hutchinson et al. 2018; Jiang et al. 2023; Jie 2017; Karanasiou et al. 2021; Kim et al. 2017; Kiser et al. 2020; Kondo et al. 2019; Kondo et al. 2022; Machado-Silva et al. 2020; Machin et al. 2019; Magzamen et al. 2021; Mahsin et al. 2022; Malig et al. 2021; Martenies et al. 2023; McBrien et al. 2023; Moitra et al. 2021; Mowbray et al. 2022; Nelson et al. 2020; Niyatiwatchanchai et al. 2023; Orr et al. 2020; Pennington et al. 2023; Ramos and Minghelli 2022; Ranse et al. 2022; Reid et al. 2019; Requia et al. 2021; Schroeder et al. 2022; Schwarz et al. 2023; Schweizer et al. 2023; Sheldon and Sankaran 2017; Sheridan et al. 2022; Stowell et al. 2019; Syam et al. 2017; Tarín-Carrasco et al. 2021; Thilakaratne et al. 2023; Tornevi et al. 2021; Wen et al. 2022; Wettstein et al. 2018; Yao et al. 2020a; Yao et al. 2020b; Ye et al. 2021; Ye et al. 2022]

A2 Studies on Cardiovascular Health

[Arabadjis et al. 2023; Augusto et al. 2020; Casey et al. 2021; Chen et al. 2023a; Chen et al. 2024; Chen et al. 2021; de Souza Fernandes Duarte et al. 2023; Doubleday et al. 2020; Doubleday et al. 2023; Douglas-Vail et al. 2023; Duncan et al. 2023; Gan et al. 2017; Gao et al. 2023; Glass et al. 2019; Gould et al. 2024; Hahn et al. 2021; Hasan et al. 2024; Hasnain et al. 2024; Heaney et al. 2022; Heft-Neal et al. 2023; Howard et al. 2021; Hutchinson et al. 2018; Jiang et al. 2023; Jie 2017; Jones et al. 2020; Karanasiou et al. 2021; Kondo et al. 2022; Lankaputhra et al. 2023; Magzamen et al. 2021; Mahsin et al. 2022; Malig et al. 2021; Martenies et al. 2023; McBrien et al. 2023; Ong et al. 2023; Pennington et al. 2023; Ranse et al. 2022; Requia et al. 2021; Schwarz et al. 2023; Stowell et al. 2019; Tarín-Carrasco et al. 2021; Thilakaratne et al. 2023; Wen et al. 2022; Wettstein et al. 2018; Wu et al. 2021a; Xi et al. 2020; Yao et al. 2020a; Yao et al. 2020b; Ye et al. 2021; Ye et al. 2022; Zeigler et al. 2022]

A3 Studies on Reproductive and Developmental Health

[Abdo et al. 2019; Brew et al. 2022; Dhingra et al. 2023; Fernández et al. 2023; Foo et al. 2024; Heft-Neal et al. 2022; Jones and Berrens 2021; Jones and McDermott 2022; Jung et al. 2023a; Jung et al. 2021; Jung et al. 2023b; Kornfield et al. 2024; Li et al. 2021; McCoy and Zhao 2021; Park et al. 2022; Requia et al. 2022a; Requia et al. 2022b; Requia et al. 2022c; Rosales-Rueda and Triyana 2019; Singh and Dey 2021; Xue et al. 2023; Zhang et al. 2023c; Zhang et al. 2023d; Zheng 2023]

A4 Studies on Cancer

[Gao et al. 2023; Glass et al. 2019; Korsiak et al. 2022; Yu et al. 2022]

A5 Studies on Neurological Health

[Cleland et al. 2022; Elser et al. 2023; Gao et al. 2023; Lai et al. 2022; Tan et al. 2019; Zhang et al. 2023a]

A6 Studies on Metabolic Health

[Chen et al. 2023a; Kondo et al. 2022; Mahsin et al. 2022; Malig et al. 2021; Rosales et al. 2022; Yao et al. 2020a]

A7 Studies on Infectious Disease

[Ademu et al. 2022; Cortes-Ramirez et al. 2022; Gonçalves et al. 2023; Kiser et al. 2021; Landguth et al. 2020; Linde et al. 2023; Meo et al. 2021; Mulliken et al. 2023; Sannigrahi et al. 2022; Schroeder et al. 2022; Schwarz et al. 2022; Xi et al. 2020; Yu and Hsueh 2023; Zhou et al. 2021]

A8 Studies on Subclinical Changes

[Abreu et al. 2017; Adetona et al. 2017; Adetona et al. 2019; Aguilera et al. 2023; Kim et al. 2017; Main et al. 2020; Niyatiwatchanchai et al. 2023; O'Dwyer et al. 2021; Wu et al. 2020; Wu et al. 2021b; Xu et al. 2023]

A9 Studies on Total Mortality and Other Health Topics

[Beyene et al. 2022b; Fadadu et al. 2022; Fadadu et al. 2021; Jegasothy et al. 2023; Linares et al. 2018; Sheldon and Sankaran 2017; Syam et al. 2017; Zhang et al. 2023b]

A10 Health Impact Assessment Studies

[Afrin and Garcia-Menendez 2021; Barbosa et al. 2024; Bernstein et al. 2021; Bruni Zani et al. 2020; Butt et al. 2020; Carreras-Sospedra et al. 2024; Cleland et al. 2021; Cromar et al. 2024; Darling et al. 2023; Dickinson et al. 2022; Efimova and Rukavishnikov 2021; Fann et al. 2018; Graham et al. 2020; Graham et al. 2021; Huang et al. 2019; Jiang and Enki Yoo 2019; Johnson and Garcia-Menendez 2022; Kollanus et al. 2017; Liu et al. 2021; Matz et al.

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Appendix B — Topic Areas Defined by CDC’s National Public Health Tracking Network

Table B1. Subject areas defined, tracked, and connected using CDC’s National Environmental Public Health Tracking Network*

Environmental	Exposures	Health effects	Population characteristics
Wildfire smoke	Environmental chemicals	Asthma	Socioeconomics
Air quality	Toxic substance releases	Cancer	Demographics
Extreme heat	Pesticides	Heat stress and illness	Lifestyle risk factors
Drinking water		Heart disease and stroke	Vulnerabilities
Flood vulnerability		Childhood lead poisoning	
Drought		Developmental disabilities	
Community design		Carbon monoxide poisoning	
Radon		Reproductive and birth outcomes	
Sunlight + UV			

* This public health system works to track and connect a nationwide network of integrated health outcomes and environmental data with an overarching goal to improve community health in the United States [Vaidyanathan et al. 2018].

Appendix B References

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Appendix C — Sampling and Analytical Methods

C1 Methods for Gas Phase Pollutants

C1.1 Nitrogen Oxides and Sulfur Oxides

NIOSH Method 6014: Nitric Oxide and Nitrogen Dioxide [NIOSH 1994a] is a fully evaluated sampling and analytical method. Air samples are collected on a sampling device using three tubes: one comprised of chromate oxidizer and two of triethanolamine-coated molecular sieve. Samples are extracted in an aqueous-absorbing solution containing triethanolamine and n-butanol and then further processed to complete color development before analysis by an ultraviolet-visible (UV/Vis) spectrophotometer.

OSHA Method ID-182: Nitrogen Dioxide in Workplace Atmospheres [OSHA 1991a] is a validated Occupational Safety and Health Administration (OSHA) method where air samples are collected using a sorbent tube containing triethanolamine-impregnated molecular sieve. The samples are desorbed in a 1.5% triethanolamine aqueous solution and analyzed as nitrite using ion chromatography (IC). This method may be modified to allow for the use of passive samplers, comprised of tape treated with triethanolamine, for the determination of both sulfur dioxide [SKC 2014] and nitrogen dioxide [SKC 2018]. Of the methods listed for nitrogen oxides, only OSHA ID-182 modified for the use of passive samplers has been evaluated at levels approaching what may be found in wildfire smoke—0.051 ppm.

OSHA Method ID-190: Nitric Oxide in Workplace Atmospheres [OSHA 1991b] uses a similar sampling device to NIOSH 6014, but samples are extracted and analyzed as in OSHA ID-182.

NIOSH Method 6004: Sulfur Dioxide [NIOSH 1994b] is a partially validated National Institute for Occupational Safety and Health (NIOSH) Manual of Analytical Methods (NMAM) 4th ed. method. Samples are collected on two cellulose filters, the second of which is treated with sodium carbonate. The front filter collects sulfuric acid and sulfate and sulfite salts that may be quantitated as total particulate sulfate. The back (treated) filter collects sulfur dioxide. Following extraction, samples are analyzed using IC with a conductivity detector. Sulfur dioxide is quantitated by summing (with appropriate stoichiometric factors applied) sulfite and sulfate responses. The estimated instrumental limit of detection (LOD) is 3 µg/sample.

OSHA Method ID-1011: Sulfur Dioxide [OSHA 2007] is an active sampling method where sulfur dioxide is collected on sodium carbonate-coated filters to form sodium sulfite. The filter samples are extracted, sulfite oxidized to sulfate, and the resulting sample is analyzed using IC with a conductivity detector. The reliable quantitation limit is 0.118 mg/m³ time-weighted average (TWA) or 0.152 mg/m³ (15-min sample).

1 C1.2 Carbon Monoxide

2 **NIOSH Method 6604: Carbon Monoxide** [NIOSH 2016a] is a fully evaluated NMAM 5th
3 edition method. It uses a portable, direct-reading carbon monoxide monitor. It can be used
4 for both personal and area monitoring. The method's instrumental LOD (1 part per million,
5 ppm) is above what could be expected on smoke days. However, newer monitors may have
6 improved sensitivity. It is important to determine each monitor's sensitivity ahead of use.

7 C1.3 Hydrogen Cyanide

8 **NIOSH Method 6010: Hydrogen Cyanide** [NIOSH 2017] is an active sampling method. Air
9 samples are collected on a sorbent tube containing soda lime. A glass fiber pre-filter is
10 placed in front of the sorbent tube during sampling to collect particulate cyanides (the pre-
11 filter is then discarded and not analyzed). Sorbent samples are extracted using water and
12 further processed to complete color development before analysis by a UV/Vis
13 spectrophotometer. A high concentration of hydrogen sulfide may cause a negative
14 interference in the measurement of hydrogen cyanide.

15 **OSHA Method 1015: Hydrogen Cyanide** [OSHA 2010] is a passive sampling method where
16 a diffusive sample is collected onto a soda lime sampling medium, extracted with water and
17 the extract analyzed using IC with electrochemical detection. The reliable quantitation limit
18 is 0.48 mg/m³.

19 C1.4 Aldehydes

20 Aldehydes are a class of compounds that may be found in wildland fire smoke. Some
21 examples of more prevalent aldehydes in this exposure situation include formaldehyde,
22 acrolein, and acetaldehyde. Various U.S. federal agencies have published methods for
23 measuring these compounds in the workplace and ambient air.

24 **NIOSH Method 2016: Formaldehyde** [NIOSH 2016b] is a fully evaluated NMAM 5th
25 edition method. Samples are collected on a cartridge containing silica-gel-coated with 2,4-
26 dinitrophenylhydrazine, extracted with acetonitrile, and analyzed by liquid
27 chromatography with ultraviolet detection (HPLC-UV). The method can measure at 0.015
28 mg/m³ (in a 15-L sample).

29 **EPA Method TO-11A: Determination of Formaldehyde in Ambient Air Using**
30 **Adsorbent Cartridge Followed by High-Performance Liquid Chromatography (HPLC)**
31 [EPA 1999b] is a U.S. Environmental Protection Agency (EPA) Compendium Method for the
32 Determination of Toxic Organic Compounds in Ambient Air. Air samples are collected on
33 2,4-dinitrophenylhydrazine (DNPH)-coated adsorbent cartridges, extracted with
34 acetonitrile, and analyzed by HPLC-UV. This method also includes other carbonyl
35 compounds (e.g., aldehydes and ketones) that also react with DNPH to form a stable
36 derivative, including acetaldehyde. This method can be used for sampling up to 24-hr when
37 formaldehyde is at low parts per billion (ppb) levels and for short-term sampling (<60 min)

1 when formaldehyde is at ppm levels. This method has the necessary sensitivity to measure
2 aldehydes in wildland fire smoke.

3 **NIOSH Method 2501: Acrolein** [NIOSH 1994c] is a partially evaluated NMAM 4th edition
4 method for the measurement of acrolein. Air samples are collected on 2-(hydroxymethyl)
5 piperidine-coated XAD-2 tubes, desorbed in toluene, and analyzed by gas chromatography-
6 nitrogen phosphorous detector (GC-NPD). The overall method accuracy was determined to
7 be 29%.

8 **OSHA Method 52: Acrolein and Formaldehyde** [OSHA 1989] is an evaluated OSHA
9 sampling and analytical method. Air samples are collected on sorbent tubes containing XAD-
10 2 that has been coated with 2-(hydroxymethyl) piperidine. Samples are desorbed in toluene
11 and analyzed by GC-NPD. Reported reliable quantitation limits (at TWA) are 6.1 µg/m³ for
12 acrolein and 20 µg/m³ for formaldehyde.

13 **EPA TO-15A: Determination of Volatile Organic Compounds (VOCs) in Air Collected in**
14 **Specially Prepared Canisters and Analyzed by Gas Chromatography–Mass**
15 **Spectrometry (GC-MS)** lists over 70 volatile organic compounds (VOCs) that are
16 quantifiable by the method, including acrolein [EPA 1999d]. Samples are analyzed by GC-
17 MS. The ability to preconcentrate samples collected in the canisters allows for very low
18 levels to be measured, including parts per trillion (ppt) by volume for some chemicals. The
19 method has the sensitivity required to measure acrolein in wildland urban interface (WUI)
20 smoke.

21 **NIOSH Method 2018: Aliphatic Aldehydes** is a partially evaluated NMAM 4th edition
22 method for four aldehydes: acetaldehyde, propionaldehyde, valeraldehyde, and
23 isovaleraldehyde [NIOSH 2003d]. The collection and analysis are similar to that listed for
24 NIOSH 2016 and EPA TO-11A.

25 For all samples collected using DNPH-coated samplers, the presence of ozone in the air
26 sample may consume the DNPH, affecting its availability to derivatize the aldehyde
27 compounds [Kleindienst et al. 1998; Sirju and Shepson 1995]. In such situations, ozone
28 must be removed before it can react with DNPH. A potassium iodide ozone scrubber
29 attached to the sampler inlet is one way to remove it. Additionally, unsaturated carbonyls
30 may form byproducts over storage time that complicate accurate measurements [Schulte-
31 Ladbeck et al. 2001].

32 **C1.5 Volatile Organic Compounds (VOCs)**

33 **NIOSH 1501: Aromatic Hydrocarbons** is an NMAM 4th edition method that can be used to
34 measure VOCs [NIOSH 2003e]. The method involves the collection of an air sample on a
35 charcoal sorbent tube, desorption using carbon disulfide, and analysis by gas
36 chromatography-flame ionization detection (GC-FID). The method specifically evaluates the
37 following compounds: benzene, toluene, ethylbenzene, xylenes, cumene, p-tert-
38 butyltoluene, and alpha- and beta-methylstyrene. An LOD range from 0.4–1.1 µg/sample is
39 reported. This method may have limited utility away from the source of a wildland fire due

1 to its sensitivity, selectivity, and potential analytical interferences when other volatile
2 organic compounds are present.

3 More sensitive and selective methods to measure VOCs include those that use gas
4 chromatography-mass spectrometry (GC-MS) for analysis. **NIOSH Method 2549: Volatile**
5 **Organic Compounds (Screening)** [NIOSH 1996] is one such method.

6 This method involves air sample collection on a multi-bed thermal desorption tube and GC-
7 MS analysis. Compounds are identified based on mass spectra interpretation and
8 computerized library searches. This method is commonly used to screen the composition of
9 air samples but can be used for quantification with few modifications, namely the inclusion
10 of a calibration curve.

11 **EPA TO-17: Determination of Volatile Organic Compounds in Ambient Air Using**
12 **Active Sampling onto Sorbent Tubes** [EPA 1999c] is an EPA Compendium Method for the
13 Determination of Toxic Organic Compounds in Ambient Air. This is a quantitative method
14 and includes many additional VOCs beyond those listed within the NIOSH methods.

15 Air samples are collected on sorbent tubes, thermally desorbed, and analyzed by GC-MS.
16 The choice of sorbent material to use in the sampling tubes depends upon the analytes of
17 interest. The method includes information to assist the user in choosing the appropriate
18 sorbent material for the analytes to be collected and measured. Reported method detection
19 limits are ≤ 0.5 ppb.

20 **NIOSH 3900: Volatile Organic Compounds** [NIOSH 2018] is an NMAM 5th edition method
21 for whole air sampling using evacuated canisters. Samples are analyzed by GC-MS. It was
22 tested for over 15 C1 to C10 VOCs, including methyl methacrylate, benzene, di- and tri-
23 chloromethane, and ethylbenzene. The working range of the method spans several orders of
24 magnitude (0.24 ppb to 2.0 ppm), depending upon the analyte. Sample preconcentration
25 allows for low levels of VOCs to be measured. This method is expected to have the
26 sensitivity required to measure VOCs in WUI smoke.

27 **EPA TO-15A: Determination of Volatile Organic Compounds (VOCs) in Air Collected in**
28 **Specially Prepared Canisters and Analyzed by Gas Chromatography-Mass**
29 **Spectrometry (GC-MS)** [EPA 1999d] lists over 70 VOCs that are quantifiable by the
30 method, including chlorinated VOCs, styrene, benzenes, and methyl methacrylate. Whole air
31 samples collected in evacuated canister and are analyzed by GC-MS. The ability to
32 preconcentrate samples collected in the canisters allows for very low levels to be measured,
33 including parts per trillion (ppt) by volume for some chemicals. This method is expected to
34 have the sensitivity required to measure VOCs in WUI smoke.

35 **C2 Particulate Gravimetric Methods**

36 Occupational air sampling methods are generally focused on the worker. For these, air
37 samples are collected within the worker's breathing zone during working hours.

38 **NIOSH Method 0500: Particulates Not Otherwise Regulated (NOR), Total, NIOSH 0501:**
39 **Particulates NOR, Total, OSHA Method PV2121: Gravimetric Determination, and**

1 **NIOSH Method 0600: Particulates NOR, Respirable** are validated occupational methods
2 that use gravimetric particulate mass as the basis for measurement [NIOSH 2003f,g, 2015b;
3 OSHA 2003]. For these methods, a microbalance is used for gravimetric weighing of the
4 filter or capsule media. The reported LODs for these methods range from <30-75
5 µg/sample, but these values could be improved with the use of a more sensitive balance. In
6 atmospheres with low particulate matter (PM) concentrations, collection of a larger air
7 volume will increase the quantity of PM collected and likely the number of reportable
8 results above the LOD. A common respirable sampler can operate at a flow rate of 4.2 L/min
9 [NIOSH 2016d]. Over an 8-hr working shift, a 2016 L air sample would be collected and for a
10 10-hr working shift, 2520 L would be collected.

11 Clean collection media are pre-weighed under controlled conditions. For each of these
12 methods, contaminated air is drawn through the air sampler inlet, the particle sizing device
13 (if present), and then through the filter media or capsule with a personal air sampling
14 pump. The collection media is subsequently post-weighed, following air sampling, under
15 similar controlled conditions. The PM mass gain (µg or mg) for the collection media is
16 divided by the sampled air volume (m³) and provided as an averaged air concentration.
17 Gravimetric analysis is a non-destructive technique that allows collection media to be
18 further utilized for chemical analyses.

19 Applied to outdoor occupational smoke exposure, gravimetric measurements could provide
20 a definitive particle mass concentration collected within the personal breathing zone of the
21 worker. A further advantage to a gravimetric measurement is that smoke is potentially
22 made up of a complex mixture of components. The overall quantity of PM collected is useful
23 as individual components that make up the smoke may then be compared to the overall
24 mass.

25 **C3 Particulate Metals**

26 **NIOSH 7300 series methods (7300, 7301, 7302, 7303, 7304, 7306):** Elements are
27 published within NMAM 4th and 5th editions [NIOSH 2003a–c, 2014a,b, 2015a]. For all the
28 methods, air samples are collected on a filter (NIOSH 7300–7304) or internal capsule
29 (NIOSH 7306 only), digested using concentrated acids, applied heat (using a hot plate, hot
30 block, or closed vessel microwave digestion system), and analyzed using an inductively
31 coupled plasma-optical emission spectroscopy (ICP-OES or ICP-AES).

32 These methods allow for the simultaneous determination of dozens of elements, including
33 those noted as possibly being present in fire smoke (aluminum, arsenic, cadmium,
34 chromium, cobalt, copper, iron, lead, manganese, nickel, potassium, titanium, and zinc) (see
35 Section 2.1). These methods may be able to measure some elements in this exposure
36 situation with sufficient sensitivity. For example, the minimum detectable concentration for
37 Mn in NIOSH 7300 is 0.025 µg/m³. This order of magnitude is below the maximum Mn level
38 detected in smoke in downwind communities [Rice et al. 2023]. However, the ubiquitous
39 nature of some of the metals (e.g., aluminum) prohibits low-level determinations using
40 these methods.

1 **OSHA Method 5003: Metal Sampling Group 1** [OSHA 2019] is validated for arsenic,
2 cadmium, and lead. Air samples are collected on MCE filters, prepared using microwave-
3 assisted acid digestion, and analyzed using ICP-MS. Listed reliable quantitation limits range
4 from 0.0365 $\mu\text{g}/\text{m}^3$ (^{114}Cd) to 0.240 $\mu\text{g}/\text{m}^3$ (^{206}Pb).

5 **OSHA Method 125G: Metal and Metalloid Particulates in Workplace Atmospheres**
6 [OSHA 2002] includes air, wipe, and bulk sampling with ICP-AES sample measurement. It is
7 validated for 13 elements, including Cd, Co, Cr, Cu, Fe, Mn, Ni, Pb, and Zn, all which may be
8 present in wildland fire smoke. As with other multi-elemental methods, additional elements
9 can be added to the method depending on instrument capability and element solubility and
10 stability in the acid matrix used for sample digestion. Quantitative detection limits listed
11 vary by element and range from 0.20 μg (Mn) to 30 μg (Fe).

12 **EPA Compendium Method IO-3.2: Determination of Metals in Ambient PM using**
13 **Atomic Absorption (AA) Spectroscopy** [EPA 1999a] can be used for the determination of
14 inorganic compounds in ambient air. Unlike other methods listed in this section, this
15 method is intended to measure an air sample that is collected over a 24-hr period on glass
16 fiber filters using a high-volume sampler. After a portion of the filter is extracted, samples
17 are analyzed using AA. The method includes many of the elements potentially present in
18 wildland fire smoke. With method detection limits ranging from 0.0001 ng/m^3 (Zn) to 4.4
19 ng/m^3 (Al), the method may have the sensitivity necessary to measure some metals in
20 wildland fire smoke.

21 **C4 Polycyclic Aromatic Hydrocarbons**

22 **NIOSH Method 5528: Polynuclear Aromatic Hydrocarbons in Air by GC-MS SIM** [NIOSH
23 2021] is a fully evaluated NMAM 5th edition method. The chemicals included in this method
24 are acenaphthene, acenaphthylene, anthracene, benz[a]anthracene, benzo[b]fluoranthene,
25 benzo[k]fluoranthene, benzo[ghi]perylene, benzo[a]pyrene, chrysene,
26 dibenz[a,h]anthracene, fluoranthene, fluorene, indeno[1,2,3-cd]pyrene, naphthalene,
27 phenanthrene, and pyrene. Samples are collected using OVS-7 tubes with glass fiber filters,
28 desorbed in methylene chloride, and analyzed by GC-MS with selected ion monitoring (SIM).

29 **NIOSH Method 5506: Polynuclear Aromatic Hydrocarbons by HPLC** [NIOSH 1998] is a
30 partially evaluated NMAM 4th edition method. The chemicals included in this method are
31 acenaphthene, acenaphthylene, anthracene, benz[a]anthracene, benzo[b]fluoranthene,
32 benzo[k]fluoranthene, benzo[ghi]perylene, benzo[a]pyrene, benzo[e]pyrene, chrysene,
33 dibenz[a,h]anthracene, fluoranthene, fluorene, indeno[1,2,3-cd]pyrene, naphthalene,
34 phenanthrene, and pyrene. Samples are collected using a sampling device consisting of a
35 polytetrafluoroethylene (PTFE) filter and XAD-2 sorbent tube. A common modification of
36 this method is to replace the sampling device with an OVS-7 sampler. Samples are extracted
37 with acetonitrile and analyzed by high-performance liquid chromatography (HPLC) with a
38 fluorescence detector and ultraviolet or photodiode array detector coupled in series. This
39 method may have limited utility away from the source of a wildland fire, due to selectivity
40 and potential analytical interferences.

1 **EPA Method TO-13A:** Determination of Polycyclic Aromatic Hydrocarbons (PAHs) in
2 Ambient Air Using Gas Chromatography/Mass Spectrometry (GC/MS) [EPA 1999e] can be
3 used to determine polycyclic aromatic hydrocarbons (PAHs) involving three member rings
4 or higher, including anthracene, benz[a]anthracene, benzo[a]pyrene, benzo[e]pyrene,
5 benzo[g,h,i]perylene, benzo[k]fluoranthene, chrysene, coronene, dibenz[a,h]anthracene,
6 fluoranthene, fluorene, benzo[b]fluoranthene, indeno[1,2,3-cd] pyrene, phenanthrene,
7 pyrene, and perylene.

8 Air samples are collected on a 102-mm quartz fiber filter and sorbent (polyurethane
9 foam [PUF] or XAD-2 resin) cartridge using a high-volume flow rate air sampler.
10 Samples are extracted and concentrated and analyzed by GC-MS. Detection limits range
11 from 10 picograms to 1 nanogram; therefore, the method should have necessary
12 sensitivity to measure PAHs in WUI smoke. Acenaphthene, acenaphthylene, and
13 naphthalene showed low collection efficiency when using PUF sorbent; therefore,
14 method users are encouraged to use XAD-2 if these analytes are of interest.

15 **Other PAHs:** Additional PAHs could be added to these methods with appropriate testing
16 and validation. For example, Navarro, et al. [2019] included the compound retene when
17 analyzing samples for PAHs by GC-MS/SIM.

18 **C5 Particulate Carbon**

19 **NIOSH Method 5040: Diesel Particulate Matter (as Elemental Carbon)** [NIOSH 2016c]
20 is a fully evaluated NMAM 5th edition method. Air samples are collected on quartz fiber
21 filters and analyzed using a thermal-optical analyzer. The thermal-optical method is
22 applicable to nonvolatile carbon species (i.e., particulate organic carbon, calcium carbonate,
23 and elemental carbon). As noted earlier, PM_{2.5} from the smoke particles contains a
24 significant carbonaceous component.

25 **C6 Other Hazardous Air Pollutants**

26 Beyond what is described above, a wide range of hazardous air pollutants could be present
27 in wildfire smoke, including dioxins, furans, and flame retardants. Their presence is
28 dependent upon the nature of the fire, the materials consumed, and the distance from the
29 fire.

30 **EPA Method 23: Determination of Polychlorinated Dibenzop-Dioxins,**
31 **Polychlorinated Dibenzofurans, Polychlorinated Biphenyls, and Polycyclic Aromatic**
32 **Hydrocarbons from Stationary Sources** details the methodology to determine chlorinated
33 dioxins and furans, chlorinated biphenyls, and PAHs from stationary sources [EPA 2023]. As
34 written, air samples are collected using a sample probe, on a glass fiber or quartz filter, and
35 on a packed column of XAD-2 sorbent. After extraction, samples are analyzed by high
36 resolution GC with high resolution MS detection.

37 A method developed by La Guardia and Hale [2015] can measure brominated and
38 organophosphate flame-retardants. Air samples are collected using a stainless steel
39 Institute of Occupational Medicine inhalable sampler containing a foam disc followed by a

1 25-mm glass fiber filter. Samples are extracted using methylene chloride and further
2 purified before analysis by ultra-performance liquid chromatography with atmospheric
3 pressure photoionization tandem MS. The reported detection limit is 0.1 ng/m³.

4 Both methods have been modified to use an OVS-2 tube containing a glass fiber filter and
5 XAD-2 sorbent as the sampler [Fent et al. 2020]. In that study, LODs for flame retardants
6 were reported as low as 0.008 µg/m³. Although no detection limits are specified, median
7 values of chlorinated dioxins and furans were measured as low as 0.18 ng/m³. Stored
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2 **Appendix D — Key Considerations** 3 **for a Medical Surveillance** 4 **or Monitoring Program** 5 **for Wildland Fire Smoke**

6 While not broadly recommended, medical monitoring or surveillance could currently serve
7 as a tool in a comprehensive risk management plan for specific indications. Several
8 elements should be considered for a wildland fire smoke medical surveillance or monitoring
9 program. How elements are used in a program can be flexible and adaptable to the situation
10 and goals of the program. For example, baseline exams can be incorporated into existing
11 surveillance programs for other hazards in the outdoor worker population (e.g., heat), or a
12 medical monitoring program can be planned for more “emergency” situations in which
13 wildland fire smoke is a rare, short-term exposure (e.g., before a planned, prescribed
14 wildland fire or on days with severe Air Quality Index). Whichever approach is chosen, it is
15 vital to ensure that the information captured will inform specific actions in the program. For
16 example, an emergency medical monitoring program may identify workers that can be
17 prioritized for voluntary use of respirators or relocated indoors on days of high wildland
18 fire exposure.

19 The following sections provide overviews of key components that should be discussed
20 when designing a medical surveillance or monitoring program for wildland fire smoke.

1 **Worker Identification**

2 Not all workers may need to be included in a medical surveillance or monitoring program
3 for wildland fire smoke. Job descriptions are a useful tool in risk stratification to identify job
4 tasks with continued exposure despite occupational engineering or administrative controls.
5 For example, within an agricultural company, there may be workers that primarily work
6 outside in the fields, versus management who work inside a ventilated, indoor enclosure.
7 Participation in the program should be voluntary and workers should be encouraged to ask
8 questions and be included in the development process.

9 **Medical Examinations and Testing (Baseline, Recurrent, 10 and Symptom-Based)**

11 Baseline medical exams serve two purposes. They establish baseline characteristics of the
12 worker population and can identify individual risk factors for adverse health outcomes due
13 to wildland fire smoke. On the population level, baseline data is used to adjust for
14 confounders during investigations of causal relationships between wildland fire smoke and
15 job tasks. For the individual worker, identification of risk factors during the baseline exam
16 can empower the worker to advocate for occupational controls to protect their health from
17 wildland fire smoke at work and give workers important information to protect themselves
18 outside the workplace. Risk factor identification could additionally be used by employers to
19 triage implementation of new occupational controls. In designing a program for wildland
20 fire smoke exposure, it is acceptable to use data from baseline medical exams conducted
21 through other programs such as respiratory protection programs. There is no need to have
22 separate or duplicate baseline exams for separate hazards assuming that all the necessary
23 baseline medical testing is included.

24 Specific medical testing included in the baseline medical examination should be correlated
25 with screening for anticipated health effects due to PM_{2.5} and include screening for
26 cardiovascular health and respiratory health as determined by qualified healthcare
27 professionals. As research expands, other health effects that have associations with PM_{2.5}
28 may need to be added (e.g., neurologic effects, metabolic effects, reproductive and
29 developmental effects, infectious disease, cancer).

30 The frequency of recurrent exams may be determined by a qualified healthcare
31 professional, although at this time there is no agreed upon exam frequency for wildland fire
32 smoke. Often, recurrent exams occur annually, unless identified health effects precipitate
33 more frequent exams. An example of a respiratory hazard that led to an altered recurrent
34 exam frequency is diacetyl, 2,3-pentanedione. NIOSH recommends 6-month recurrent
35 evaluations for workers exposed to diacetyl and 2,3-pentanedione (agents unrelated to
36 wildland fire smoke), with follow-ups every 3 months if suspected lung disease is found.
37 This recommendation follows research showing that lung disease can rapidly develop after
38 inhalation exposure to these compounds [NIOSH 2016]. As research in wildfire smoke
39 exposure expands, future recommendations on the frequency of recurrent exams may be

1 able to be provided. In addition, recurrent exams may need to be organized and
2 implemented for the workforce after a significant wildland fire smoke event.

3 At recurrent exams, additional medical testing to the baseline exam may be added as
4 deemed appropriate by a healthcare professional. Any instance in which a worker
5 experiences an acute health event believed to be precipitated by wildland fire smoke should
6 be evaluated at the time of the event in a symptom-based exam. The data from symptom
7 based medical exams should be included in surveillance data analysis.

8 It is important to mention that some medical surveillance programs can incorporate
9 physical exam data from medical monitoring programs or baseline work capacity testing
10 that is often used for occupations that require high levels of physical fitness.

11 **Worker Training**

12 All programs should include an element of worker training. Key components of the training
13 should include risk factors for severe health effects from wildfire smoke, common
14 symptoms, ways to reduce exposure through occupational controls, and what to do if
15 serious health events occur while working.

16 **Data Analysis**

17 Systemic, longitudinal data analysis is the keystone of a medical surveillance program to
18 identify trends and relationships between occupational tasks, hazards, and health effects.
19 The data is generally de-identified and compiled from medical examinations, any
20 Occupational Safety and Health Administration (OSHA) 300 logs of events that occurred
21 related to wildland fire smoke, and any relevant alternate sources (e.g., public health
22 surveillance systems). Often, this requires an individual trained in epidemiology or
23 surveillance.

24 Medical monitoring programs are more often used to detect early manifestations of clinical
25 disease in the exposed worker population and allow for an individual worker to get access
26 to secondary prevention from a qualified healthcare professional.

27 **Respiratory Protection Program**

28 If a respiratory protection program is needed due to the level of exposure to wildland fire
29 smoke or other respiratory hazards, a medical questionnaire, appropriate fit testing, and
30 training on respirator use will need to be conducted in accordance with OSHA's Respiratory
31 Protection standard, 29 CFR 1910.134 [OSHA 1998].

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1 **Attribution Statement**

2 N95, P100, and NIOSH Approved are certification marks of the U.S. Department of Health
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