Potential Long-Term Effects of Wildland Fire Chemical Exposure: Evidence and Mechanisms

NASEM WEBINAR–3 RECOVERY: Wildfire Recovery, Adaptation, and Long-Term Health Impacts

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Presentation Objectives

- Review the Reported Long-term Effects from Wildland Fire Chemical Exposure
- List the Potential Mechanisms for Toxicity
- Consider Evidence Based Interventions
- Discuss Gaps and Future Directions

Background

- **Changing landscape:** Global increase in wildland fire frequency, size, and intensity leads to more prolonged smoke events.
- **Complex exposures:** Wildland fires release smoke, ash, and resuspended dust. Toxic pollutants for both occupational groups and the public.
- **Landmark recognition:** In 2022, IARC classified occupational exposure as a firefighter (Group 1).
- **Disproportionate impacts:** Wildland firefighters, children, pregnant women, and socially vulnerable communities face the greatest risks.

The Chemical Composition of Wildfire Urban Interface (WUI) Emissions

Gaseous Pollutants & Particulate Matter

- **$PM_{2.5}$:** dominant pollutant; penetrates deep into lungs and bloodstream. Wildfire PM is more oxidative than other sources.
- **Gases:** carcinogens (benzene, formaldehyde), cellular asphyxiants (cyanide) and irritants (acrolein).
- **PAHs** : e.g., benzo[a]pyrene; formed during incomplete combustion. Biomonitoring confirms systemic uptake in firefighters.

- **Metals** — Lead (neurotoxin), Arsenic, Chromium, Cadmium, Mercury from paint, electronics, lumber and soil/vegetation.
- **Dioxins & Furans** — highly toxic byproducts of burning PVC/chlorine-containing materials.
- **Asbestos** — released from older building materials; risk of mesothelioma and asbestosis.

Biomonitoring: Quantifying the Internal Dose in Firefighters

Biomarkers of Exposure

- **PAHs:** Post-exposure urinary PAH metabolites (OH-PAHs) can increase up to 12-fold; a single work-shift causes a 3.5-fold increase. (Crit Rev Toxicol 2025;55(6):601-619.)
- **Chronic exposure:** Baseline OH-PAH levels in wildland firefighters are 2–23× higher than in the general population. (Front Public Health. 2024 Mar 6:12:1338435)
- **Metals:**), Post-shift increases documented: nickel (33–53%timony (45–56%), arsenic (up to 80% in smokers). (Science of The Total Environment 2024 Nov: 983:176105).

Biomarkers of Effect

- **Systemic Inflammation:** Significant increase in peripheral band neutrophils; elevated proinflammatory cytokines (IL-6, IL-8).
- **Oxidative Stress:** Increased free radicals and decreased antioxidants (a primary post-exposure finding.)
- **Genotoxicity:** Studies consistently show increased DNA damage markers after firefighting.

Healthcare (Basel). 2024 Jan 25;12(3):307

Long-Term Respiratory Effects: Firefighters and Children

Wildland Firefighters

- **Lung Cancer Mortality**: Career wildland firefighters face an estimated 8–43% increase in lung cancer mortality depending on career length and exposure (using modeling). Environmental Research. 2019. 173: 462-468.
- **Lung Function:** Accelerated age-related decline and immediate cross shift decreases in lung function; increased risk of COPD. (J Occup Environ Hyg. 2014;11(9):591-603)
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Children: A Highly Vulnerable Population

- Wildfire $PM_{2.5}$ may be up to 10× more harmful to children's respiratory health than $PM_{2.5}$ from other pollution sources. (Nat Commun 2021:12,1493).
- A 10 µg/m³ increase in wildfire $PM_{2.5}$ → 6% increase in asthma hospitalizations. (Environ Res 2019 Dec;179:Pt A:108777).
- A California study found the same increase associated with a 30% rise in pediatric respiratory visits. (Pediatrics. 2021 Apr;147(4):e2020027128)

Cardiovascular Effects & Mechanisms

Published Evidence

- **Firefighters:** Estimated 16–30% increase in CVD mortality for career wildland firefighters. Risk rises steeply early in career (using modeling). (Environmental Research. 2019. 173: 462-468).
- **US population:** Each 1 µg/m³ increase in 3-year average wildfire $PM_{2.5}$ → 1.3% increased stroke risk; potentially >17,000 additional cases/year in U.S. elderly. (European Heart Journal. June 2026, 47:21: 2673–2682)
- **US populaton:** Long-term exposure linked to >20,000 additional cases/year among U.S. older adults. (Journal of the American College of Cardiology. 2025. 85:25:2439-2451).

Pathophysiological Mechanisms (hypothesized)

1. Systemic Inflammation

$PM_{2.5}$ activates transcription factors→ releases IL-6, TNF-α → drives atherosclerosis.

2. Oxidative Stress

Wildfire $PM_{2.5}$ generates excessive ROS → promotes LDL oxidation and plaque formation.

3. Endothelial Dysfunction

Inflammation and oxidative stress impair vasodilation → prothrombotic state.

Neurological Effects & Mechanisms

Neurodegenerative disease is an emerging field of study after wildland fire exposure

Conflicting evidence: A study linked wildfire $PM_{2.5}$, potentially more neurotoxic than other sources, to dementia but was later retracted (JAMA Neurol. 2025;82(1):40–48).

Mechanisms of Neurotoxicity (hypothesized)

1. Increased Inflammatory Mediators: $PM_{2.5}$ activates microglia → releases secondary messengers→ increased inflammation in neurons.
2. Protein Aggregation: Chronic inflammation accelerates accumulation of β-amyloid plaques, hyperphosphorylated tau tangles (e.g. Alzheimer's disease)
3. Oxidative Stress & Neuronal Loss: $PM_{2.5}$ induces oxidative stress and mitochondrial dysfunction → impairs energy production → progressive neuronal cell death and cognitive decline.

J Toxicol. 2025 Dec 30;2025:5752904

Carcinogenic Potential for Wildland Fire Exposure

Sufficient Evidence (IARC Group 1) for occupational exposure as a firefighter

- **Mesothelioma**: 58% higher risk in firefighters vs. general population. Strongly linked to asbestos exposure in WUI fires.
- **Bladder Cancer:** Statistically significant increased risk. Mechanistically linked to dermal and inhalation absorption of PAHs in soot. Post-exposure urinary PAH metabolites increase up to 12-fold.

Limited Evidence in Other Cancers

- Colon, prostate, testicular cancer
- Melanoma, non-Hodgkin lymphoma
- One meta-analysis: 102% greater risk for testicular cancer; 53% greater risk for multiple cancers.

IARC. Monogr Identif Carcinog Hazards Hum. 2023:132:1–730.

Vulnerable Populations (High-Risk Groups)

- **Wildland Firefighters:** Highest risk due to repeated, high-intensity occupational exposures.
- **Children**: Higher respiratory rates, developing nervous system, and hand-to-mouth behaviors increase vulnerability to smoke and post-fire contaminants (e.g., lead).
- **Pregnant Women:** Linked to adverse birth outcomes; fetus especially vulnerable to chemicals with developmental toxicity.
- **Elderly & Those with Pre-existing Conditions:** Greater susceptibility to cardiovascular and respiratory impacts.
- **Socially Vulnerable Communities:** Greater heavy-smoke exposure; higher susceptibility to smoke-induced cardiovascular events.

Evidence-Based Interventions

- **Respiratory Protection:**
  - SCBA is the gold standard for structural firefighting, including overhaul. However, most wildland firefighters use bandanas and N95® (have no protection against gases)
  - For WFFs, N95 respirators can reduce particulate exposure by tenfold if fitted properly and have been shown to significantly reduce urinary levels of PAH metabolites (Ann Work Expo Health. 2023;67(3):354-365)
- **Decontamination:**
  - On-scene wet decontamination of turnout gear used by structural firefighters with soap and water can remove up to 85% of PAHs (J Occup Environ Hyg. 2019 Jan 31;16(3):199–205)
  - Using skin wipes on the head, neck, and hands immediately post-fire can potentially reduce skin concentration of PAHs by over 50% (J Occup Environ Hyg. 2017 Sep 14(10), 801–814)
- **HEPA air filtration:**
  - Portable air cleaners (PACs) with HEPA filters can reduce indoor $PM_{2.5}$ concentrations by a median of 71% (https://ncceh.ca/sites/default/files/202602/NCCEH%20%E2%80%93%20Wildfire%20Smoke%20Indoor%20Outdoor%20Evidence%20Review%2020260202.pdf: accessed 6/12/2026)

Research Gaps and Future Directions

- **WUI Fires**: Priority area due to unique chemical profiles and toxic legacy contamination in ash, soil, and dust — posing long-term risks to communities and cleanup crews.
- **Improved Exposure Assessment needed:** Biomonitoring, environmental sampling and exposure tracking
- **Longitudinal Cohort Studies:** Critical need for long-term studies like the Fire Fighter Cancer Cohort Study (FFCCS) and the National Firefighter Registry for Cancer to track outcomes over full careers and into retirement.
- **Vulnerable Population Studies:** A key gap is the lack of direct longitudinal studies quantifying pulmonary function changes in children following long-term wildfire smoke exposure.
- **Post-Fire Contamination:** Research needed on remediation phase hazards and re-entry exposures from contaminated gear, tools, and environment

Thank you!

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