TOXICITY OF WILDLAND FIRE SMOKE
EVIDENCE AND RESEARCH GAPS
Inhalation and Respiratory Specialty Section
Society of Toxicology
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Outline

• Acute Health Effects Associated with Wildland Fire Smoke

• Delayed Health Effects Associated with Wildland Fire Smoke

• Human Mechanistic and Toxicological Evidence

• Some Knowledge Gaps about Toxicity of Wildland Fire Smoke
Acute Pulmonary Effects: Observational Evidence among the General Population

• Different measures of exposure to wildfire smoke

• Outcome measures — ED visits, doctor visits, hospitalizations, medication use

• Mostly US and Australian studies
Acute Pulmonary Effects: Observational Evidence among the General Population

• Inconsistent evidence for respiratory mortality (Johnston et al., 2011; Magzamen et al., 2021; Morgan et al., 2010)

• Association with respiratory morbidity events (Alman et al., 2016; Cicretti et al., 2021; Delfino et al., 2008; Gan et al., 2019; Hahn et al., 2021; Magzamen et al., 2021; Morgan et al., 2010; Tinling et al., 2016 etc.)
  • Consistent evidence for all respiratory, asthma
  • Some evidence for respiratory infections, bronchitis, COPD

• Evidence of disparities in associations
Acute Pulmonary Effects: Observational Evidence among the General Population

- Same study regions, exposure, and outcome measures as for respiratory effects

- Inconsistent evidence across cardiovascular outcomes (Reid et al., 2016)

- Some evidence of association with specific outcomes (Gan et al., 2019; Hahn et al., 2021; Heaney et al., 2022; Johnston et al., 2011; Jones et al., 2020; Magzamen et al., 2021; Tinling et al., 2016, Wettstein et al., 2018)
  - All-cause cardiac, IHD, cardiac arrest, myocardial infarction, heart failure, and hypertension morbidity events
  - CVD mortality events

- Evidence of disparities
Evidence of Delayed/Long-Term Health Effects in Human Population Studies

• Sustained lung function decline in longitudinal and cross-sectional studies (Orr et al., 2020; Ontawong et al., 2020)

• Birth outcome – pre-term and low birth weight (Holstius et al., 2013; Abdo et al., 2019; Heft-Heal et al., 2022; Requia et al., 2022; Amjad et al., 2021)

• Infectious disease (Landguth et al., 2020; Prunicki et al., 2019; Brocke et al., 2022)
  • Supported by studies of immune dysregulation
Limitations of Observational Studies

• Mostly ecological by nature

• Prone to exposure misclassification – e.g., will not account for behavioral change and protective measures that might modify exposure

• Health outcomes most likely for the already vulnerable

• Lack of knowledge about contribution of exposure to disease pathogenesis
Evidence from Occupational Studies

Reinhardt TE and Ottmar RD, 2004

Adetona O et al., 2013

<table>
<thead>
<tr>
<th>Pollutant (2003 PEL)</th>
<th>Fire Type (No. of Samples)</th>
<th>Overall (Shift)</th>
<th>Mean (Shift)</th>
<th>Maximum (Shift)</th>
<th>Mean (Fireline)</th>
<th>Maximum (Fireline)</th>
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<tbody>
<tr>
<td>Acrolein (100 ppb)</td>
<td>Initial attack (n = 45)</td>
<td>1 ppb</td>
<td>11 ppb</td>
<td>5 ppb</td>
<td>37 ppb</td>
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<tr>
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<td>Project fires (n = 84)</td>
<td>1 ppb</td>
<td>15 ppb</td>
<td>2 ppb</td>
<td>16 ppb</td>
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<tr>
<td></td>
<td>Prescribed burns (n = 200)</td>
<td>9 ppb</td>
<td>60 ppb</td>
<td>15 ppb</td>
<td>98 ppb</td>
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<td>Acetic acid (1000 ppb)</td>
<td>Initial attack (n = 45)</td>
<td>3 ppb</td>
<td>24 ppb</td>
<td>14 ppb</td>
<td>43 ppb</td>
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<tr>
<td></td>
<td>Project fires (n = 84)</td>
<td>4 ppb</td>
<td>249 ppb</td>
<td>6 ppb</td>
<td>384 ppb</td>
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<tr>
<td></td>
<td>Prescribed burns (n = 200)</td>
<td>16 ppb</td>
<td>58 ppb</td>
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<td>Carbon dioxide (5000 ppm)</td>
<td>Initial attack (n = 24)</td>
<td>391 ppm</td>
<td>706 ppm</td>
<td>488 ppm</td>
<td>742 ppm</td>
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<td>Project fires (n = 31)</td>
<td>439 ppm</td>
<td>588 ppm</td>
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<td>Prescribed burns (n = 700)</td>
<td>440 ppm</td>
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<td>Carbon monoxide (50 ppm)</td>
<td>Initial attack (n = 45)</td>
<td>1.6 ppm</td>
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<td>4.0 ppm</td>
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<td>Prescribed burns (n = 200)</td>
<td>4.1 ppm</td>
<td>38 ppm</td>
<td>6.9 ppm</td>
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<td>Formaldehyde (750 ppm)</td>
<td>Initial attack (n = 45)</td>
<td>6 ppb</td>
<td>58 ppb</td>
<td>28 ppb</td>
<td>92 ppb</td>
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<td></td>
<td>Project fires (n = 84)</td>
<td>13 ppb</td>
<td>84 ppm</td>
<td>18 ppb</td>
<td>93 ppb</td>
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<td>Prescribed burns (n = 200)</td>
<td>47 ppb</td>
<td>390 ppb</td>
<td>75 ppb</td>
<td>600 ppb</td>
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<tr>
<td>Respirable particulate (5 mg/m³)</td>
<td>Initial attack (n = 45)</td>
<td>0.022 mg/m³</td>
<td>1.56 mg/m³</td>
<td>1.11 mg/m³</td>
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<td>Project fires (n = 84)</td>
<td>0.50 mg/m³</td>
<td>2.30 mg/m³</td>
<td>0.72 mg/m³</td>
<td>2.93 mg/m³</td>
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<td>Prescribed burns (n = 200)</td>
<td>0.63 mg/m³</td>
<td>6.9 mg/m³</td>
<td>1 mg/m³</td>
<td>10.5 mg/m³</td>
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<tr>
<td>Total particulate (15 mg/m³)</td>
<td>Initial attack (n = 15)</td>
<td>1.39 mg/m³</td>
<td>1.81 mg/m³</td>
<td>5.32 mg/m³</td>
<td>8.84 mg/m³</td>
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<tr>
<td></td>
<td>Project fires (n = 15)</td>
<td>1.47 mg/m³</td>
<td>4.17 mg/m³</td>
<td>1.72 mg/m³</td>
<td>4.38 mg/m³</td>
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<td>Prescribed burns</td>
<td>Not applicable</td>
<td>Not applicable</td>
<td>Not applicable</td>
<td>Not applicable</td>
<td></td>
</tr>
</tbody>
</table>

Table 1. Unadjusted geometric means for exposure components and burn characteristics (arithmetic mean for LG/PM_{10} ratio).
Evidence from Occupational Studies

Adetona O et al., 2017
Evidence from Occupational Studies

• Potential for studies on the level of the individual

• Caveats
  • Elevated exposure — intensity (might be order of magnitude higher), intake (breathing) rate, frequency
  • Healthy worker effect
  • Confounding exposures
  • Difficult population for long-term longitudinal study
Evidence from Occupational Studies

- Observation of acute responses aligning with mechanisms for adverse outcomes in the general population

  - Hejl et al., 2013

- Possibly, also decrements in lung function

  - Adetona et al., 2017

  - Wu et al., 2021
Evidence from Occupational Studies

• Indication of chronic effects of cumulative exposures

• Lung function decrements in association with continuing or cumulative exposure (e.g., Adetona et al., 2011)

• Increased oxidative stress markers in association with career length (Adetona et al., 2013)

• Increased odds/prevalence of self-reported physician-diagnosed hypertension, heart arrythmia, and hypercholesterolemia in association with career length (Semmens et al., 2016)
Evidence from Toxicology Studies

• Alteration of respiratory, cardiac, and vascular function from in-vivo and human chamber experiments (e.g., Kim et al., 2014; Martin et al., 2020; Thompson et al., 2018; Unoson et al., 2013)
  • Evidence of similar mechanisms (generation of reactive species, oxidative stress, inflammation, cytotoxicity, interaction with autonomic control)

• Biomass smoke (furnace-generated woodsmoke, wildfire smoke particles, woodsmoke particles)
  • Wildfire smoke exposure model difficult
Evidence from Toxicology Studies

• Differential PM effects
  • Wildland fire smoke vs. general ambient air or traffic PM
  • Fire condition
  • Vegetation type
  • PM size
  • Aging/airborne pollution transport

• Evidence for differential effects in epidemiological studies (Aguilera et al., 2021; Delfino et al., 2009; Magzamen et al., 2021)
  • Especially suggests more potency for respiratory outcomes vs. non-wildfire ambient PM
  • Also based on aging/airborne pollution transport
Toxicity Evidence from Whole Smoke In-Vivo Studies

• Cumulative exposure of rats to smoke plume induced neuroinflammatory responses (Scieszka et al., 2021)

• Behavioral deficits and cognitive impairment in offspring of macaques exposed to wildland fire (Camp Fire) smoke during pregnancy (Capitanio et al., 2022)

• Early-life infant macaque exposure to wildfire smoke associated with impaired immune and pulmonary functions in later life (Black et al., 2017).

• Support from biomass smoke and PM
Gaps in Knowledge

• Distinction of PM effect from other source PM

• Effect of non-PM component

• Effect of cumulative exposure

• Mitigation
References

- Erlandsson L et al. 2020. Exposure to wood smoke particles leads to inflammation, disrupted proliferation and damage to cellular structures in a human first trimester trophoblast cell line. Environ Pollut 264: 114790.


References

- Morgan G. 2010 et al. Effects of Bushfire Smoke on Daily Mortality and Hospital Admissions in Sydney, Australia. Epidemiology 21(1) 47-55.
References

Wildfires and the Urban Interface

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Office of Research and Development
7/28/2022

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Wildland fires are a major public health issue

- Estimated that 1,000s to 10,000s deaths each year attributable to wildland fire smoke
  - $11-20 billion/year for short-term exposures
  - $76-130 billion/year for long-term exposures

O'Dell et al. 2021 doi: 10.1029/2021GH000457
Fann et al. 2018 doi:10.1016/j.scitotenv.2017.08.024
Wildfire burned acres are increasing

Adapted from
https://www.nifc.gov/fireInfo/fireInfo_stats_totalFires.html
Climate change is making temperatures rise and increasing wildfire risk.

- Decades of fire suppression leading to overgrowth
- Drought and insect driven tree mortality

- It's Getting Warmer
  - Average annual temperatures around the world have increased by 1.9 °F

- Snow Melts Sooner
  - Winter snowpack melts up to 4 weeks earlier than in previous decades.

- Landscape is Drier
  - Conditions are primed and when wildfires ignite they spread quickly.
Wildland fires occur in every season, in every region

Wildland fires = wildfires, prescribed fires, and agricultural fires

Not a “Fire Season” but a “Fire Year”
Most of the health effects are attributed to fine particulate matter (PM$_{2.5}$), there is also a wide range of hazardous air pollutants:

- Volatile organic compounds (VOCs)
- Semi-volatile organic compounds (SVOCs)
Wildfires are an increasing slice of the PM$_{2.5}$ emissions in the U.S.

Anticipate wildland fires to make up 50% in 2020

Adapted from EPA's National Emissions Inventory for 2014 and 2017
Wildfire PM$_{2.5}$ is mostly carbon

**Average PM Composition**

- Elemental Carbon: 4.93%
- Organic O,N,S: 24.41%
- Organic Carbon: 62.88%
- SO$_4$: 0.66%
- Cl: 0.81%
- K: 0.96%
- All Other Elements: 0.25%

**PM Size Distributions Per Event**

- Regional smoke events (solid lines)
- Long range transport (dashed lines)

Laing et al. 2016 doi:10.5194/acp-16-15185-2016
Ash is another important fire emission

Adachi et al. 2022 doi:10.1029/2021JD035657
Wide range of VOCs emitted from wildfires

Wide range of everything in between VOC and PM

Composition of these intermediate organics (IVOCs)

Hatch et al. 2018 doi:10.5194/acp-18-17801-2018
Wildland Urban Interface (WUI)

“...exists where humans and their development meet or intermix with wildland fuel.”
Increasingly wildfires also burn in urban areas

2007 Greek Fires
2,100 structures

2016 Gatlinburg Wildfires
2,460 structures

2017 Thomas Fire
1,063 structures

2018 Camp Fire
18,804 structures

2020 Sonoma-Lake-Napa Unit
1,723 structures

2019 Australia Bushfires
8,208 structures
WUI areas have experienced rapid growth

WUI growth in the US from 1990 - 2010
WUI fires occur across the United States

Destructive WUI fires are not always the largest wildfires

In some WUI fires urban materials are the dominant “fuel”

Adapted from CalFire https://www.fire.ca.gov/media/5511/top20_destruction.pdf
Urban “fuels” are housing materials

- Lumber framing
- Insulation
- Surface finishes
Urban “fuel” includes the contents in the home

- Furniture
- Carpet
- Clothing
- Electronics
- Cleaning materials
- Pesticides
- Herbicides
- Fixtures
Wildfire emissions:
- PM$_{2.5}$
- VOCs
- SVOCs

Urban fire emissions:
- Metals
- Halogenated organics
- Cyanates
- What else?

WUI fire smoke is an even more complex mixture.
Emissions information derived from structure fire context

• Structure burns from inside out
• Focus on furnishings, not structural materials
• Focus on acutely toxic emissions (CO, HCN)
• Some data on VOCs
• Minimal data on PM
• Extremely limited data on metals
Some suspected emissions from WUI fires

Anticipate greater amounts of some species seen in laboratory studies:

- Hazardous organic pollutants (dioxins, furans, flame retardants, polychlorinated biphenyls)
- Toxic gases (HCl, HF, Phosgene, NH₄), Isocyanates
- Toxic metals (e.g., As, Pb, Sb, Cu, Zn, and Cr)
Metals might be a useful WUI fire “Fingerprint”

Carr Fire (2018)  
~ 500 structures  
7/28/18

Tubbs Fire (2017)  
~ 3,000 structures  
10/10/17

Camp Fire (2018)  
~18,000 structures  
11/10/18

Boaggio et al. in review
### Possible sources of metal emissions

<table>
<thead>
<tr>
<th>Metal</th>
<th>Category</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arsenic PM2.5 LC</td>
<td>Wood preservatives, pesticides</td>
</tr>
<tr>
<td>Chromium PM2.5 LC</td>
<td>Alloys, fixtures, tanning, pigments</td>
</tr>
<tr>
<td>Copper PM2.5 LC</td>
<td>Plumbing, electrical</td>
</tr>
<tr>
<td>Lead PM2.5 LC</td>
<td>Plumbing, solder, pigments, batteries</td>
</tr>
<tr>
<td>Magnesium PM2.5 LC</td>
<td>Alloys, pyrotechnic, fire retardant, fertilizers</td>
</tr>
<tr>
<td>Manganese PM2.5 LC</td>
<td>Alloys, fungicide, catalyst</td>
</tr>
<tr>
<td>Nickel PM2.5 LC</td>
<td>Alloys, batteries, coins, catalyst</td>
</tr>
<tr>
<td>Rubidium PM2.5 LC</td>
<td>Electronics/optics, pyrotechnics</td>
</tr>
<tr>
<td>Strontium PM2.5 LC</td>
<td>CRT glass, pyrotechnics</td>
</tr>
<tr>
<td>Titanium PM2.5 LC</td>
<td>Alloys, high temperature components, pigments</td>
</tr>
<tr>
<td>Vanadium PM2.5 LC</td>
<td>Alloys, pigments</td>
</tr>
<tr>
<td>Zirconium PM2.5 LC</td>
<td>Consumer products (cosmetics, anti-perspirants, plastics)</td>
</tr>
</tbody>
</table>

*Note: The color scheme represents the level of concern associated with each metal.*
In summary

• We have learned quite a lot about wildland fire smoke over the past few decades
• We have seen more frequent fires in the wildland urban interface
  o We know almost nothing about the “fuels” in these fires
  o We know very little about the emissions from these fires
• We expect these fires to continue to occur as the WUI continues to develop and fires continue to increase

More research is needed!
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  Katie Boaggio (EPA)
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