

# 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

TABLE II. Shift- and Fireline-Average Smoke Exposures Among Wildland Firefighters

Pollutant (2003 PEL)	Fire Type (No. of Samples)	Overall (Shift)		At Fires (Fireline)	
		Mean <sup>B</sup>	Maximum	Mean <sup>B</sup>	Maximum
Acrolein <sup>A</sup> (100 ppb)	Initial attack (n = 45)	1 ppb	11 ppb	5 ppb	37 ppb
	Project fires (n = 84)	1 ppb	15 ppb	2 ppb	16 ppb
	Prescribed burns (n = 200)	9 ppb	60 ppb	15 ppb	98 ppb
Benzene (1000 ppb)	Initial attack (n = 45)	3 ppb	24 ppb	14 ppb	43 ppb
	Project fires (n = 84)	4 ppb	249 ppb	6 ppb	384 ppb
	Prescribed burns (n = 200)	16 ppb	58 ppb	28 ppb	88 ppb
Carbon dioxide (5000 ppm)	Initial attack (n = 24) <sup>C</sup>	391 ppm	706 ppm	488 ppm	742 ppm
	Project fires (n = 31) <sup>D</sup>	439 ppm	588 ppm	465 ppm	668 ppm
	Prescribed burns (n = 200)	450 ppm	733 ppm	519 ppm	853 ppm
Carbon monoxide (50 ppm)	Initial attack (n = 45)	1.6 ppm	13.1 ppm	7.4 ppm	28.2 ppm
	Project fires (n = 84)	2.8 ppm	31 ppm	4.0 ppm	39 ppm
	Prescribed burns (n = 200)	4.1 ppm	38 ppm	6.9 ppm	58 ppm
Formaldehyde <sup>A</sup> (750 ppb)	Initial attack (n = 45)	6 ppb	58 ppb	28 ppb	92 ppb
	Project fires (n = 84)	13 ppb	84 ppb	18 ppb	93 ppb
	Prescribed burns (n = 200)	47 ppb	390 ppb	75 ppb	600 ppb
Respirable particulate <sup>A</sup> (5 mg/m <sup>3</sup> )	Initial attack (n = 45)	0.022 mg/m <sup>3</sup>	1.56 mg/m <sup>3</sup>	1.11 mg/m <sup>3</sup>	2.46 mg/m <sup>3</sup>
	Project fires (n = 84)	0.50 mg/m <sup>3</sup>	2.30 mg/m <sup>3</sup>	0.72 mg/m <sup>3</sup>	2.93 mg/m <sup>3</sup>
	Prescribed burns (n = 200)	0.63 mg/m <sup>3</sup>	6.9 mg/m <sup>3</sup>	1 mg/m <sup>3</sup>	10.5 mg/m <sup>3</sup>
Total particulate (15 mg/m <sup>3</sup> )	Initial attack (n = 7) <sup>E</sup>	1.39 mg/m <sup>3</sup>	1.81 mg/m <sup>3</sup>	5.32 mg/m <sup>3</sup>	8.64 mg/m <sup>3</sup>
	Project fires (n = 15) <sup>F</sup>	1.47 mg/m <sup>3</sup>	4.17 mg/m <sup>3</sup>	1.72 mg/m <sup>3</sup>	4.38 mg/m <sup>3</sup>
	Prescribed burns	Not applicable <sup>G</sup>	Not applicable <sup>G</sup>	Not applicable <sup>G</sup>	Not applicable <sup>G</sup>

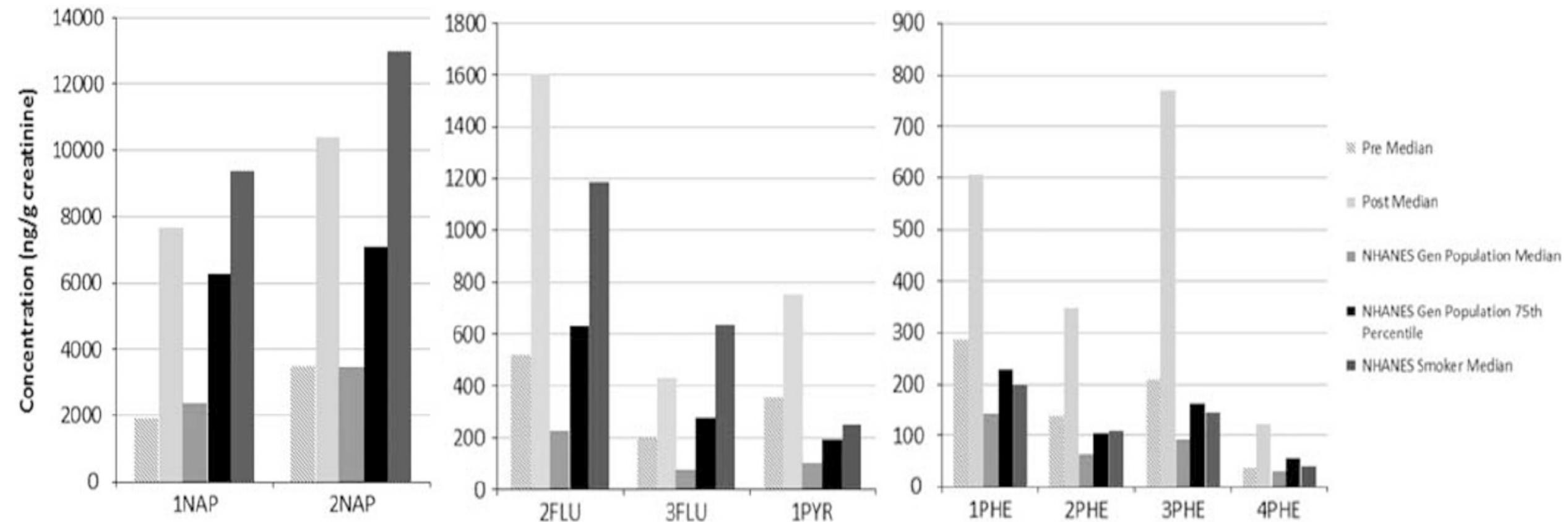
Table 1. Unadjusted geometric means for exposure components and burn characteristics (arithmetic mean for LG/PM<sub>2.5</sub> ratio).

Exposure	Average (95% lower, upper CLs)	Minimum	Maximum	N
<b>Overall</b>				
PM <sub>2.5</sub> (µg m <sup>-3</sup> ) <sup>a</sup>	530 (476, 591)	64	2068	130
8-h adjusted PM <sub>2.5</sub> (µg m <sup>-3</sup> ) <sup>a</sup>	525 (466, 590)	66	2456	130
CO (p.p.m.)	1.5 (1.3, 1.7)	0.02	8.2	140
LG (µg m <sup>-3</sup> )	20 (16, 29)	0.04	291	122
LG/PM <sub>2.5</sub> ratio (%)	6.9 (5.4, 8.5)	0.008	61	122
Size of burns (acres)	910	80	3300	
Duration of work shift (h)	7.9	3.0	13	

Reinhardt TE and Ottmar RD, 2004

Adetona O et al., 2013

# Evidence from Occupational Studies



Adetona O et al., 2017

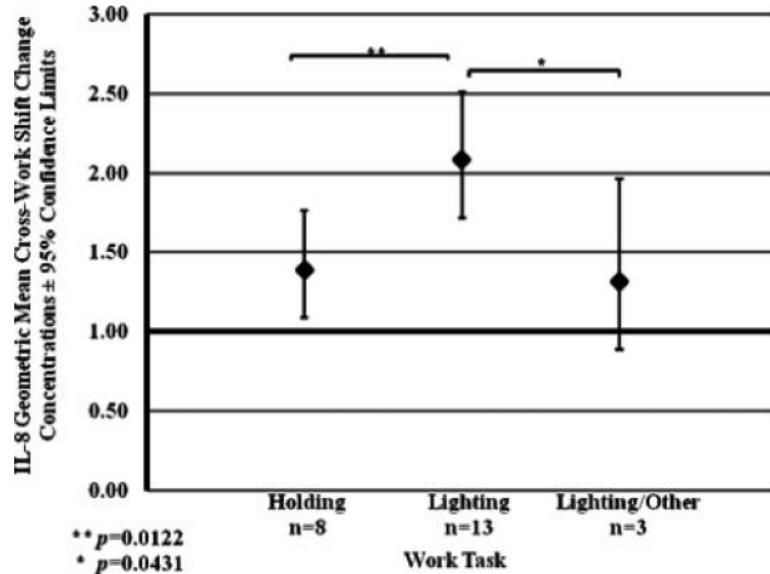
# Evidence from Occupational Studies

- 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

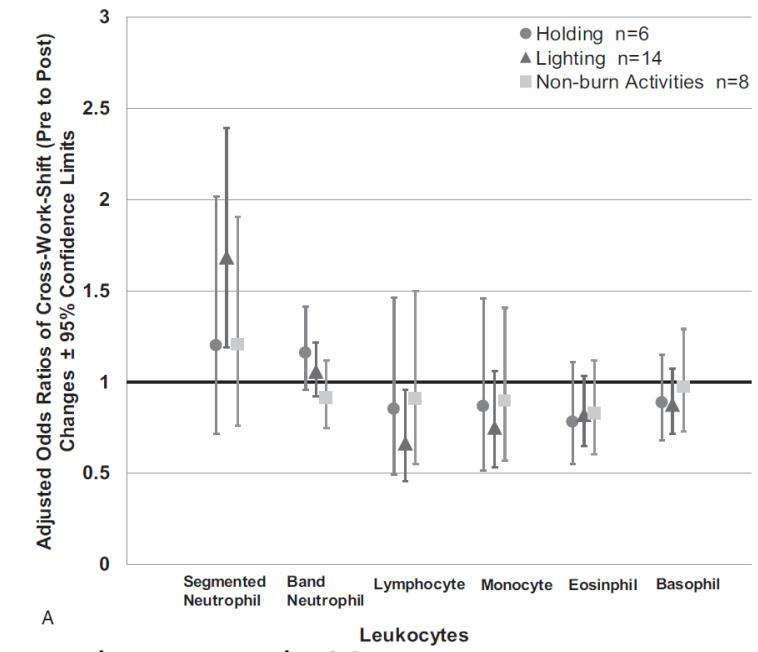
# Evidence from Occupational Studies

- Observation of acute responses aligning with mechanisms for adverse outcomes in the general population
- Possibly, also decrements in lung function

Wu et al., 2021



Hejl et al., 2013



Adetona et al., 2017

Table 2 Cross-shift changes (post-shift or next-morning vs pre-shift) in crude and creatinine-corrected values of urinary biomarkers in WLFFs on prescribed burn days or regular work days using linear mixed-effect models

	Prescribed burn		Regular work	
	Ratio (95% CI)	P value	Ratio (95% CI)	P value
<b>Crude values</b>				
Pre-shift to post-shift*				
Mutagenicity	2.56 (1.49–4.40)	<0.01	0.92 (0.49–1.73)	0.77
<b>Creatinine-corrected values</b>				
Pre-shift to post-shift*				
Mutagenicity	1.16 (0.98–1.39)	0.09	0.93 (0.74–1.18)	0.52

# 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 arrhythmia, 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

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# Wildfires and the Urban Interface

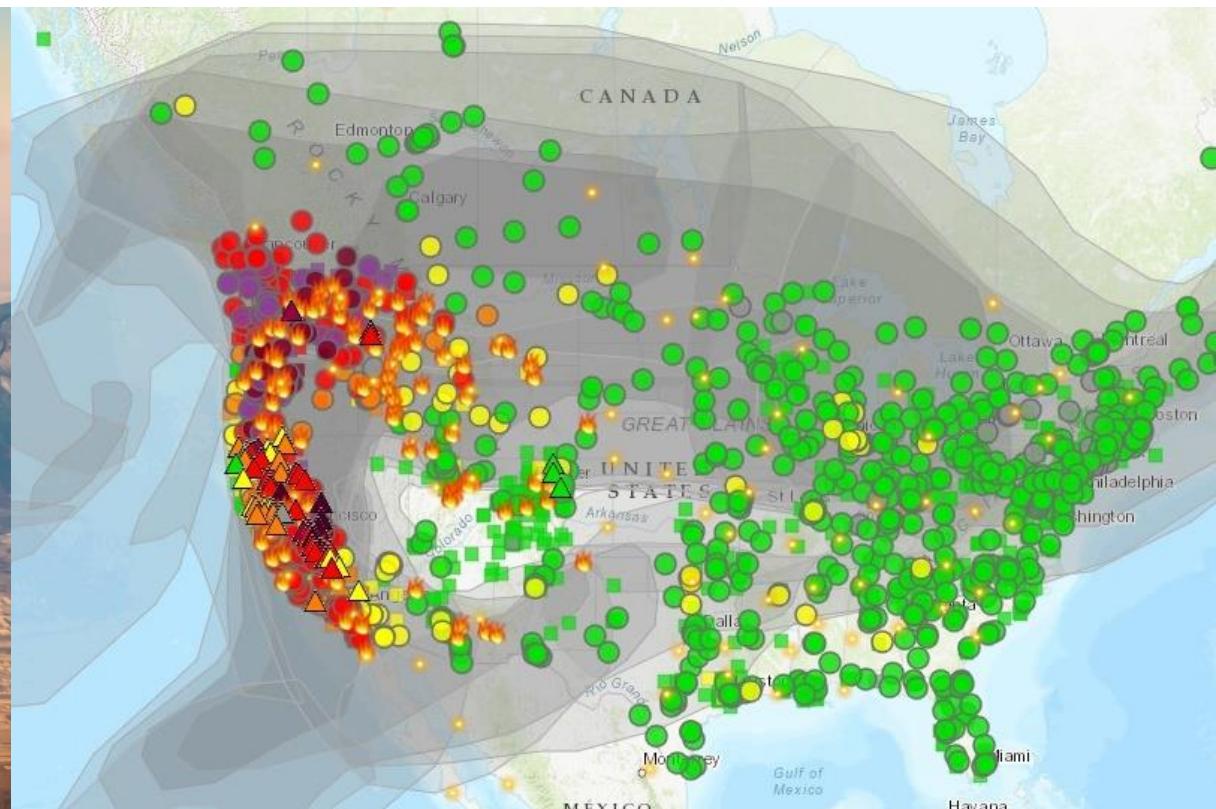
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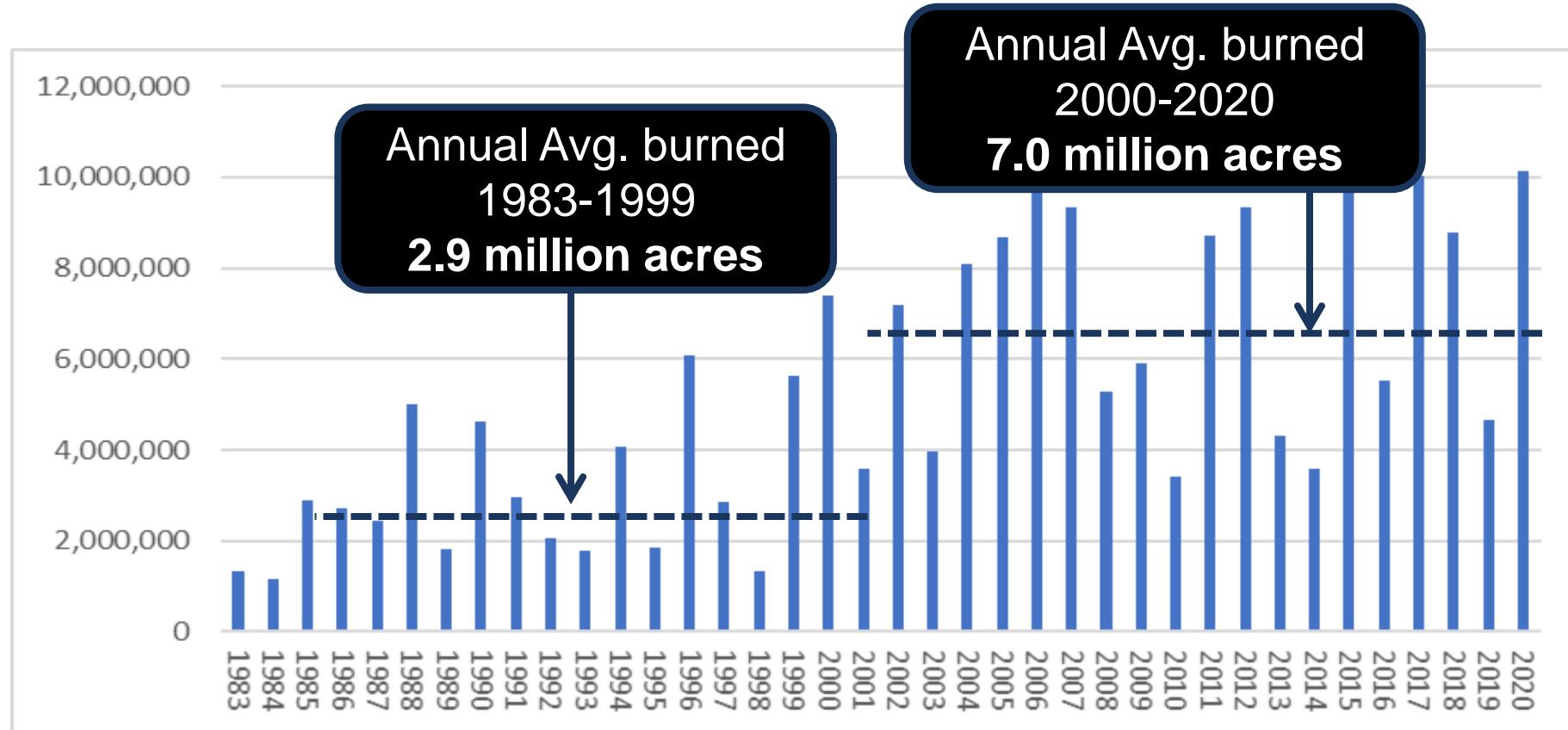
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# Wildland fires are a major public health issue



- 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

# Wildfire burned acres are increasing



Adapted from  
[https://www.nifc.gov/fireInfo/fireInfo\\_stats\\_totalFires.html](https://www.nifc.gov/fireInfo/fireInfo_stats_totalFires.html)

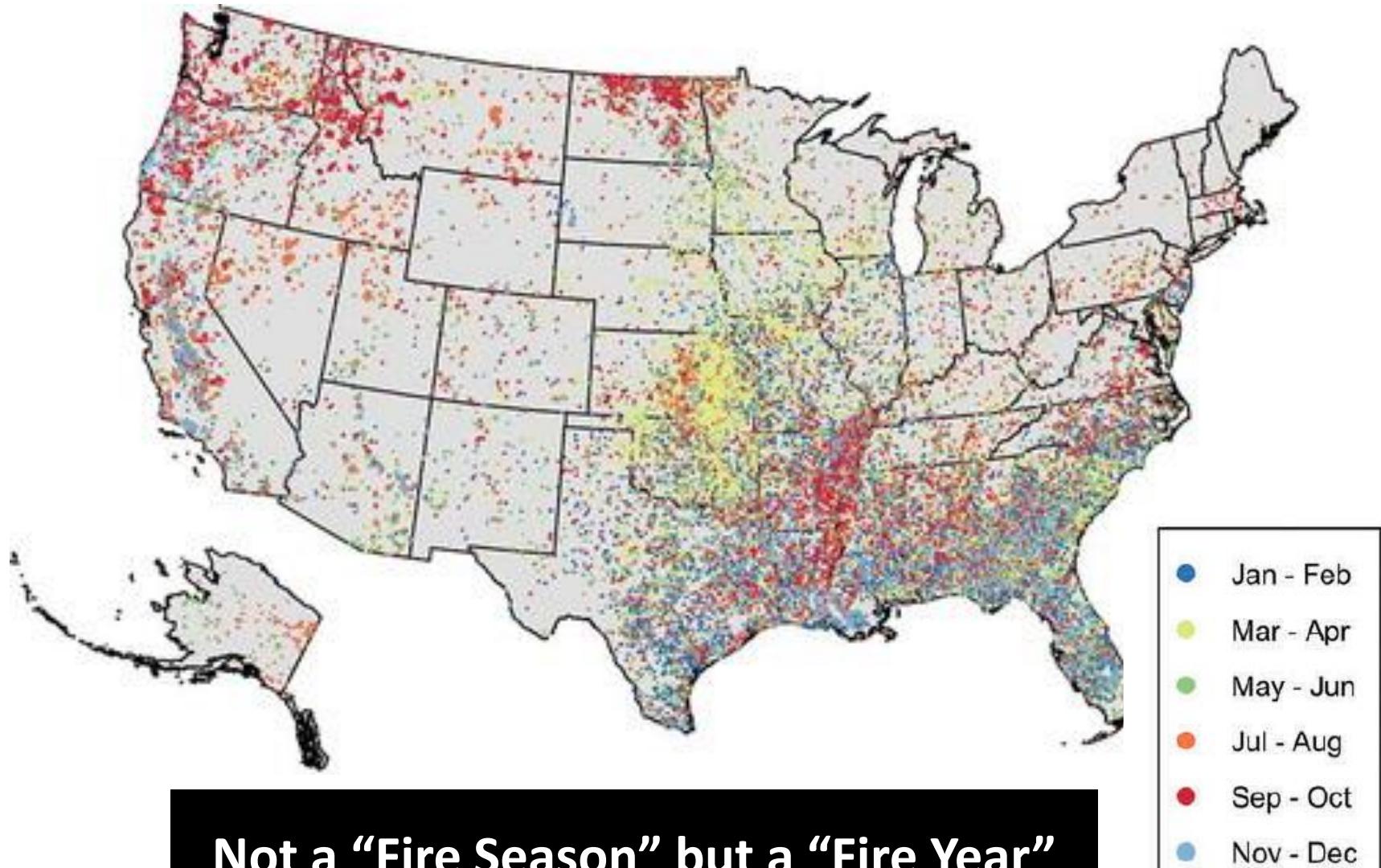
# Climate change is making temperatures rise and increasing wildfire risk.



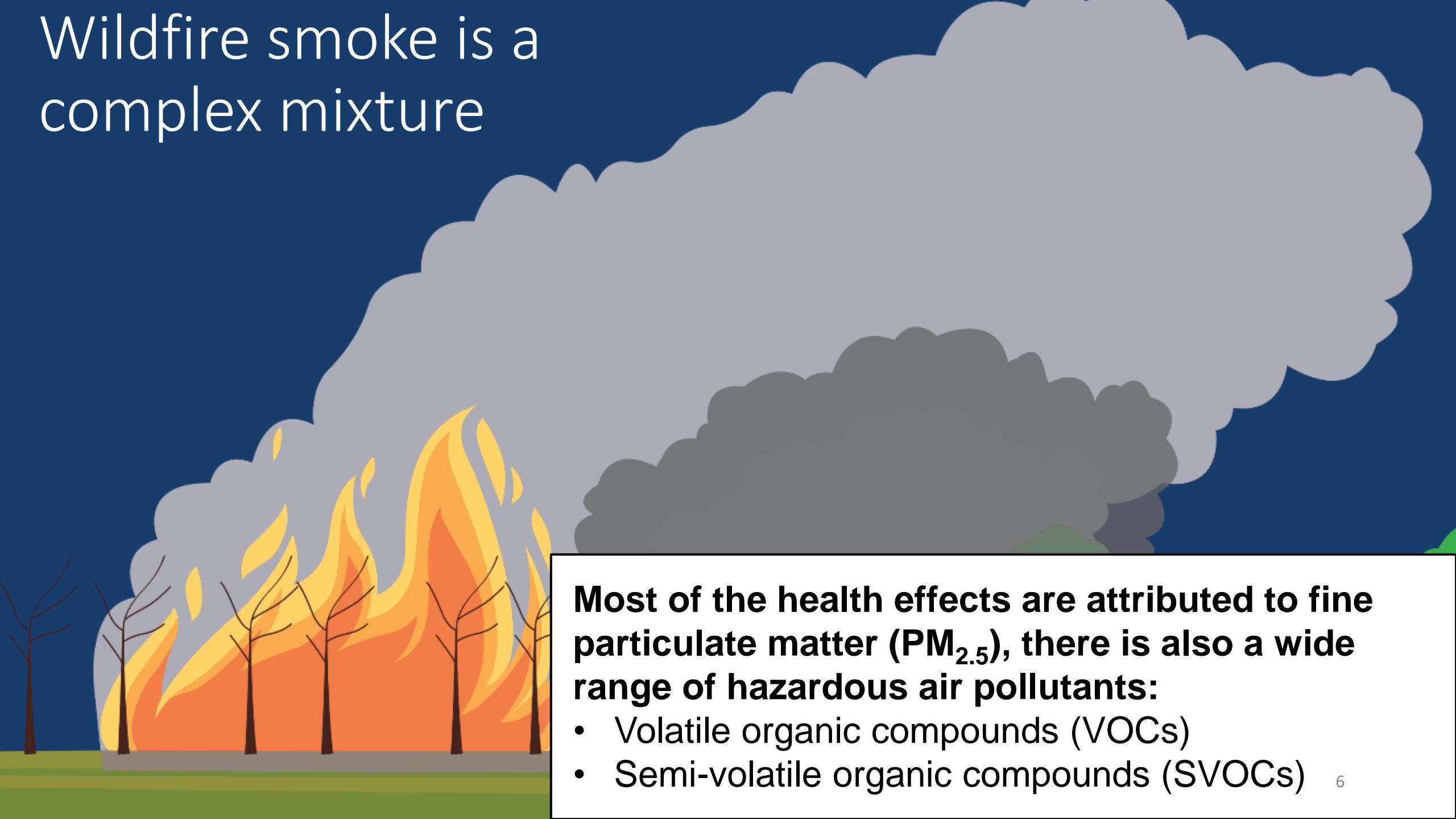
- Decades of fire suppression leading to overgrowth
- Drought and insect driven tree mortality

# Wildland fires occur in every season, in every region

Wildland fires = wildfires,  
prescribed fires, and  
agricultural fires



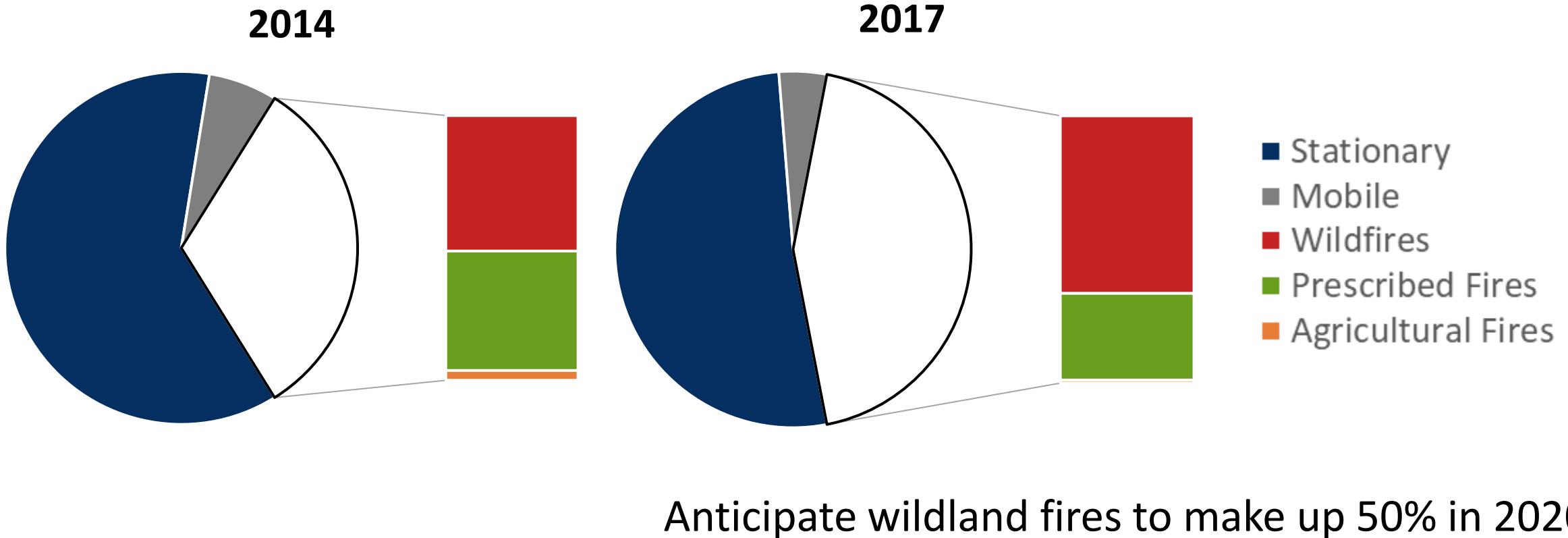
# Wildfire smoke is a complex mixture



**Most of the health effects are attributed to fine particulate matter (PM<sub>2.5</sub>), there is also a wide range of hazardous air pollutants:**

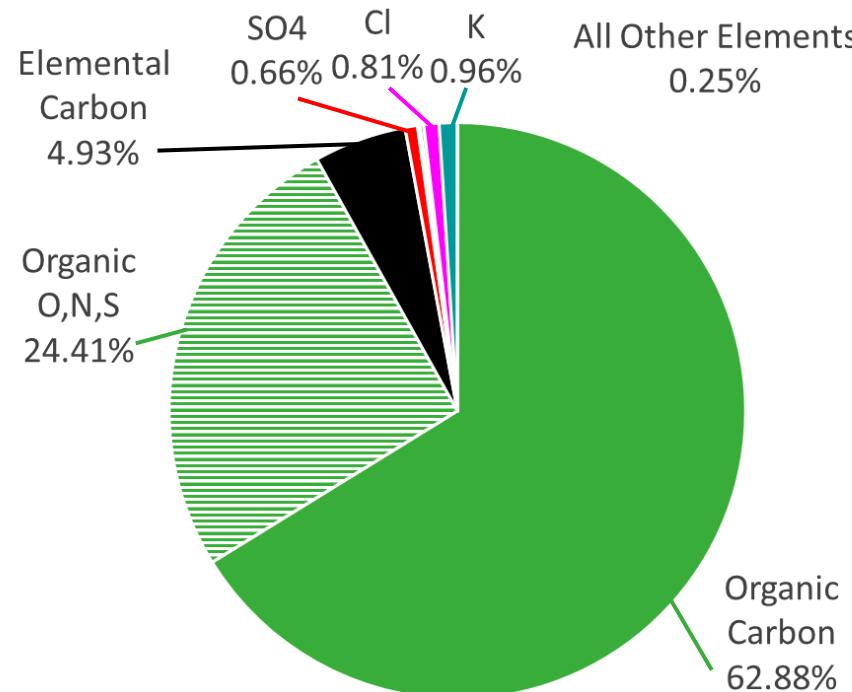
- Volatile organic compounds (VOCs)
- Semi-volatile organic compounds (SVOCs)

# Wildfires are an increasing slice of the PM<sub>2.5</sub> emissions in the U.S.

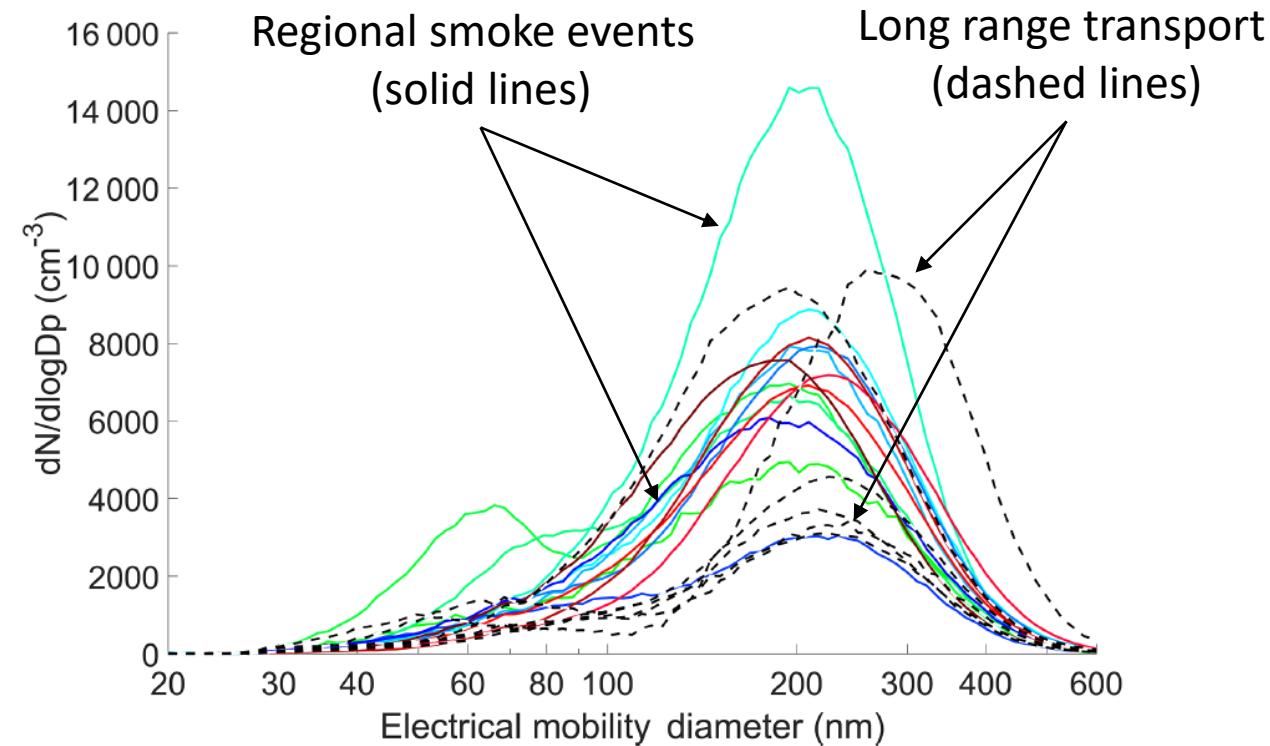


# Wildfire PM<sub>2.5</sub> is mostly carbon

## Average PM Composition

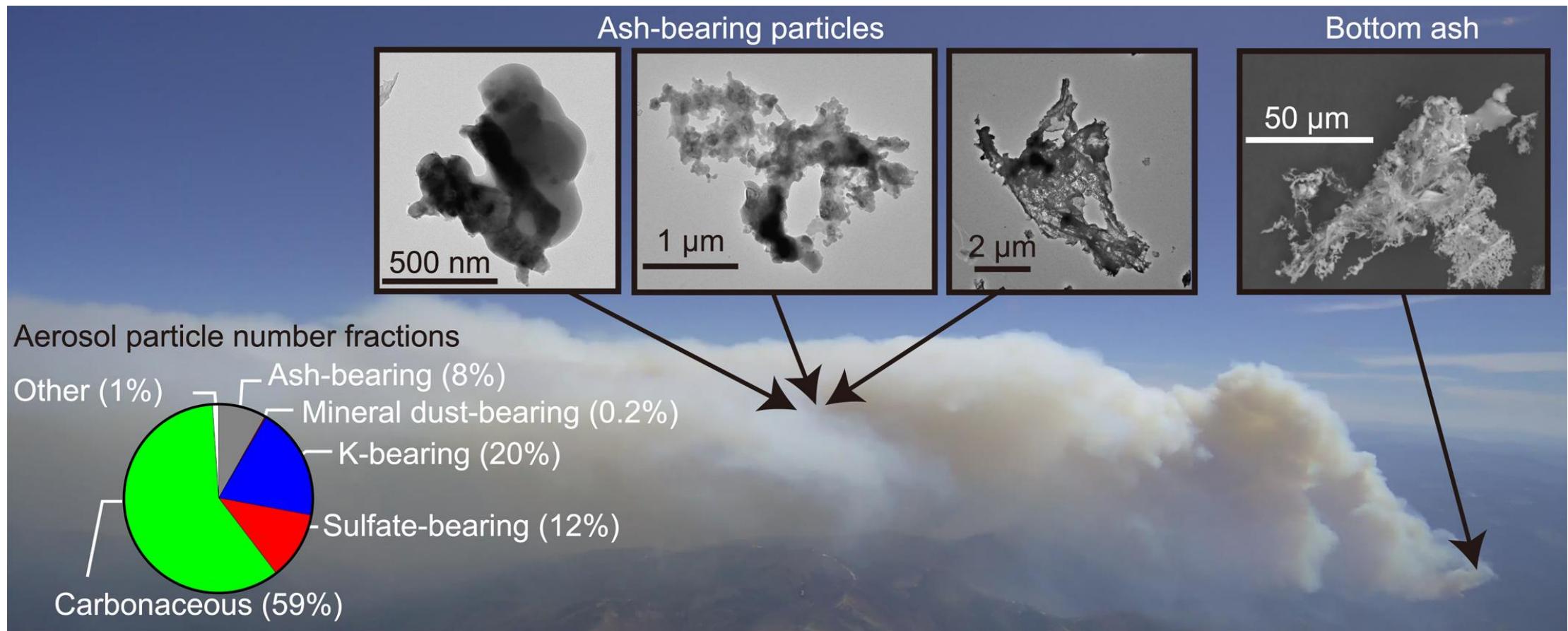


## PM Size Distributions Per Event

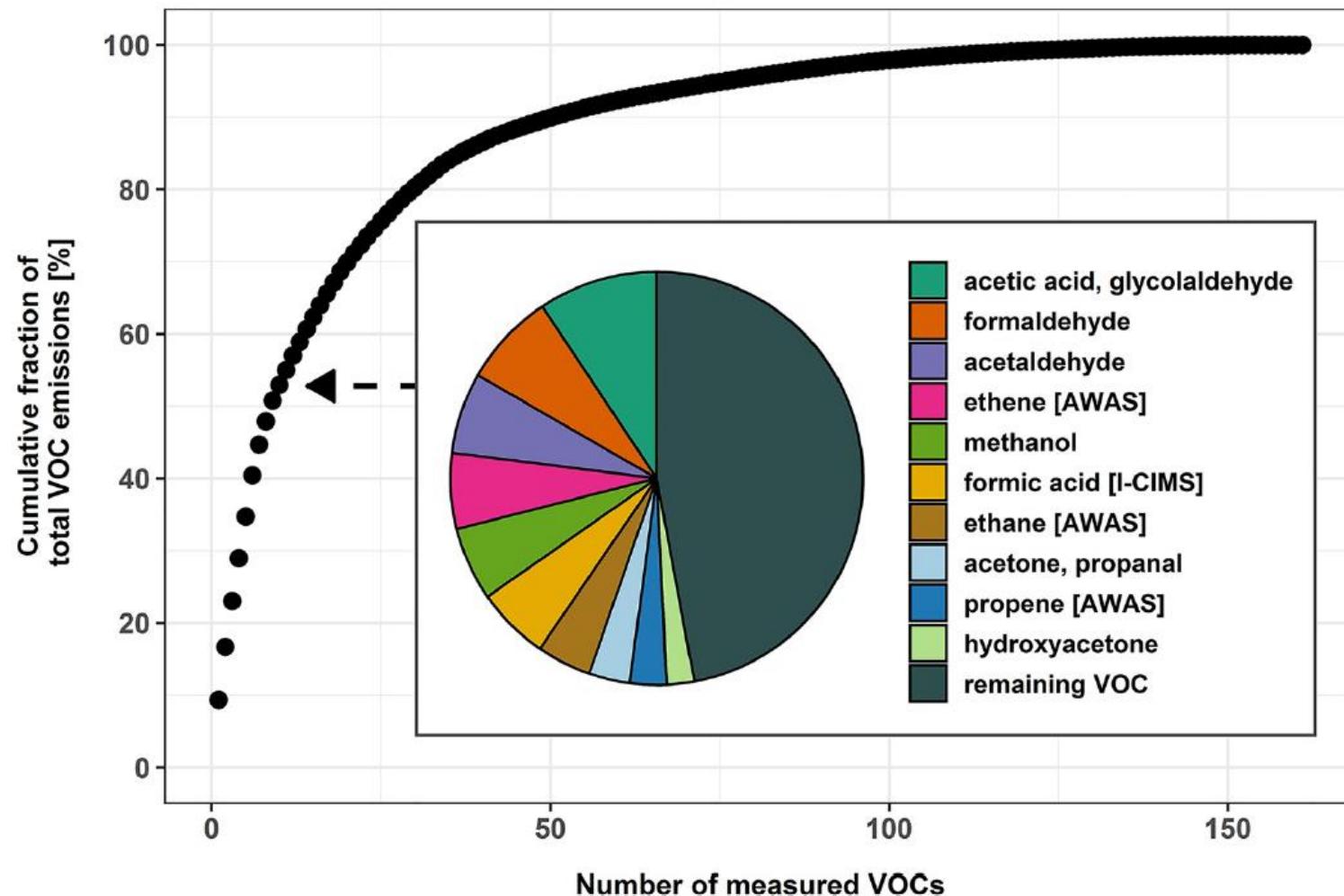


Laing et al. 2016 doi:10.5194/acp-16-15185-2016

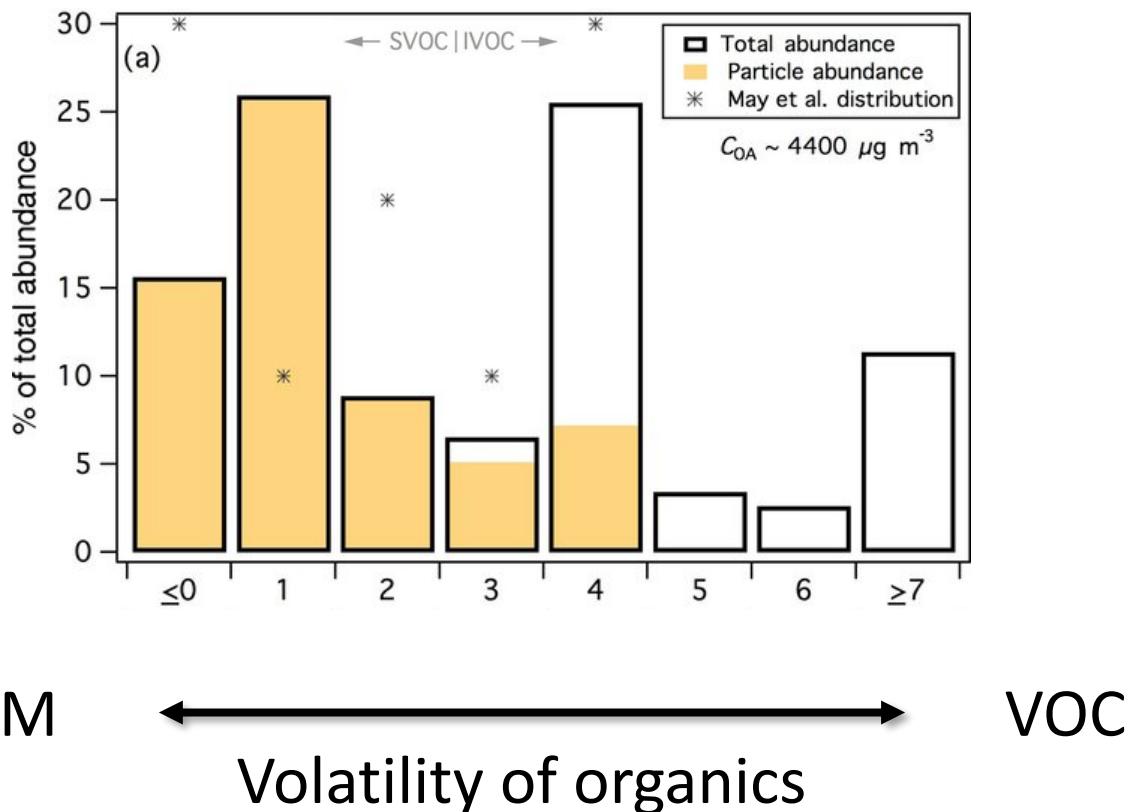
# Ash is another important fire emission



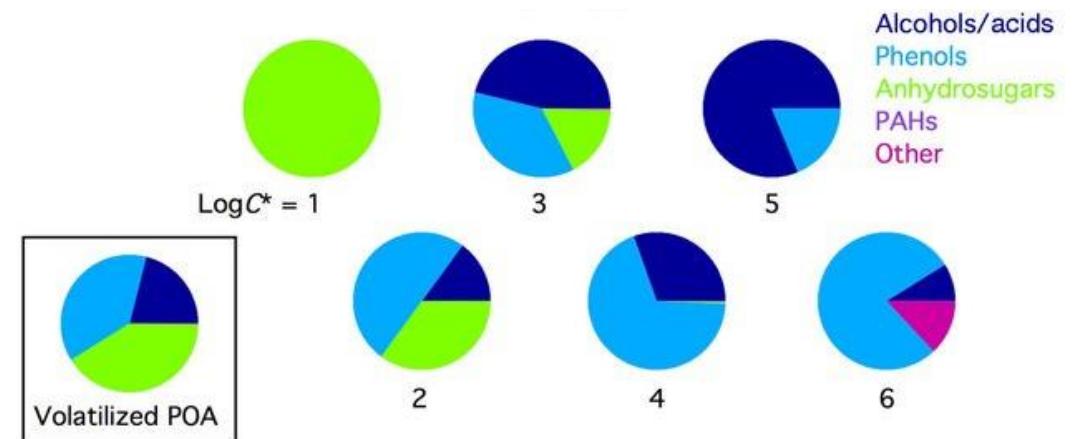
# Wide range of VOCs emitted from wildfires



# Wide range of everything in between VOC and PM



Composition of these intermediate organics (IVOCs)



# Wildland Urban Interface (WUI)

**“...exists where humans and their development meet or intermix with wildland fuel.”**



# Increasingly wildfires also burn in urban areas



(Wikipedia Nick-D)  
2019 Australia Bushfires 8,208 structures

2007 Greek Fires  
2,100 structures

2016 Gatlinburg Wildfires  
2,460 structures

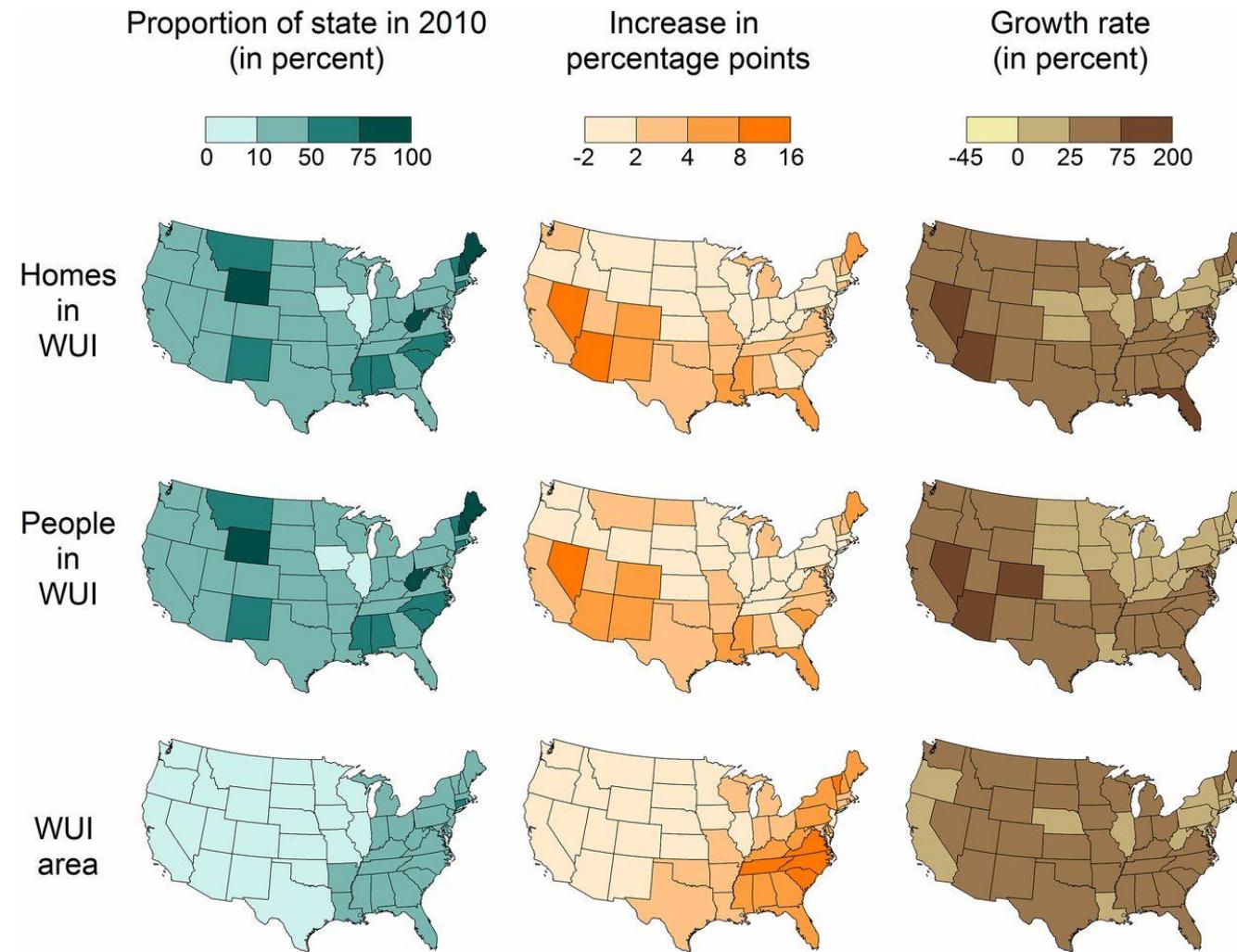
2017 Thomas Fire  
1,063 structures

2018 Camp Fire  
18,804 structures

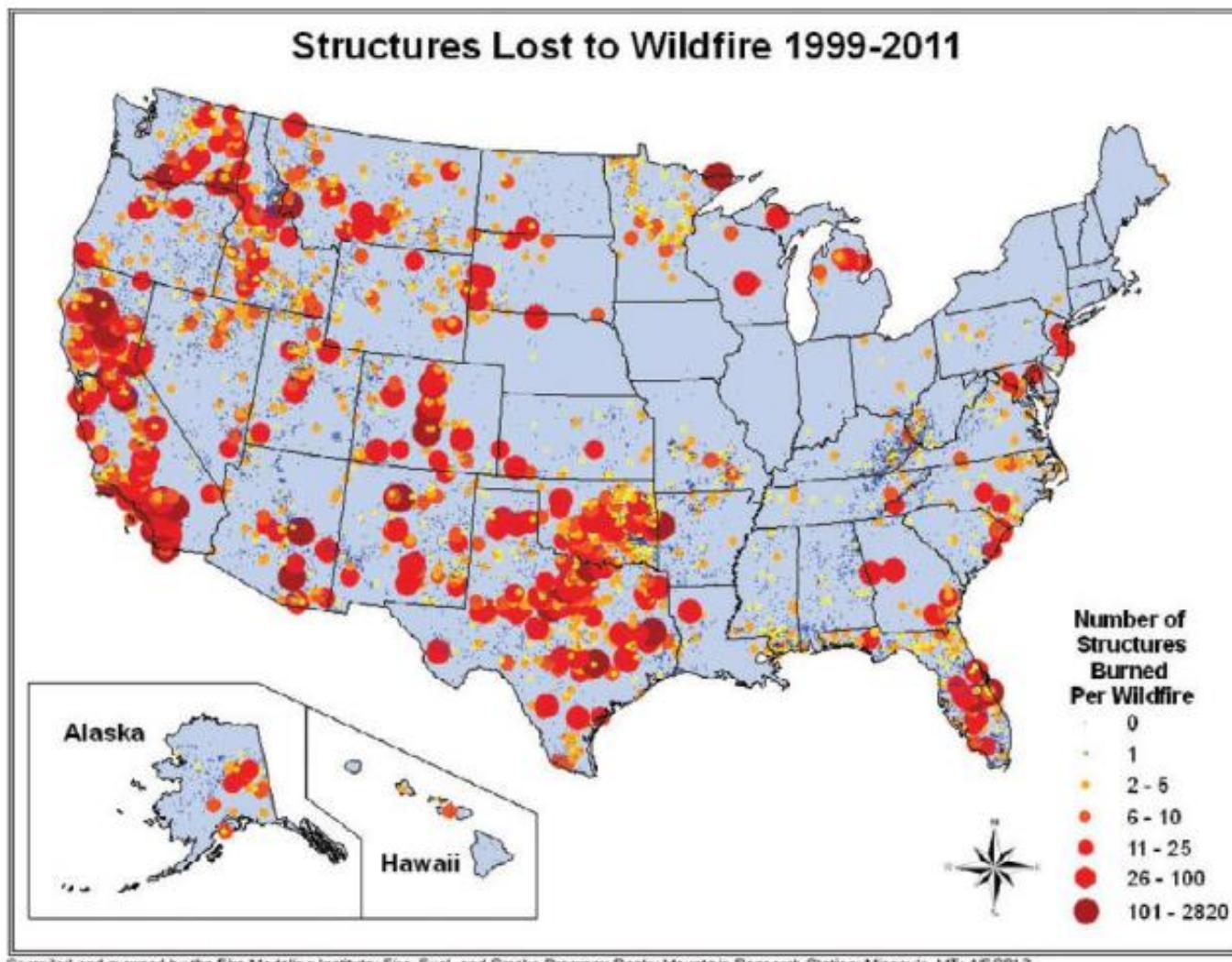
2020 Sonoma-Lake-Napa  
Unit 1,723 structures

# WUI areas have experienced rapid growth

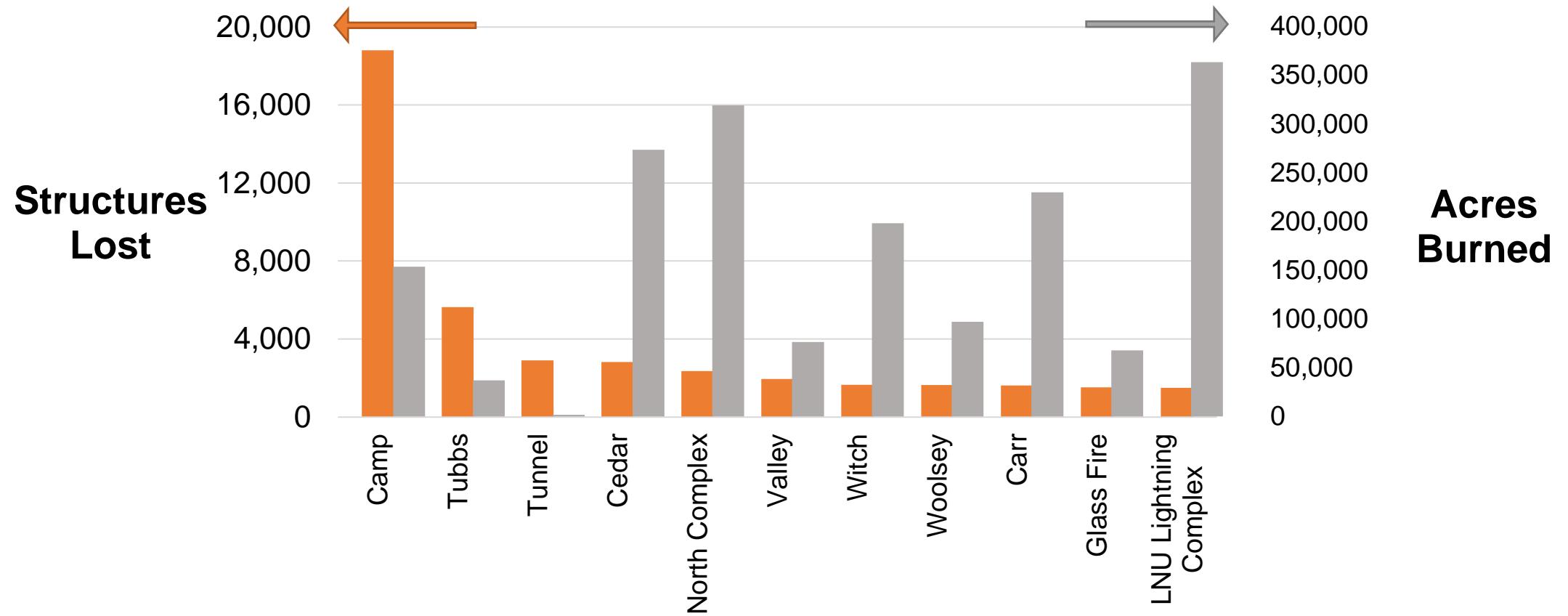
**WUI growth in  
the US from  
1990 - 2010**



# 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”

# 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

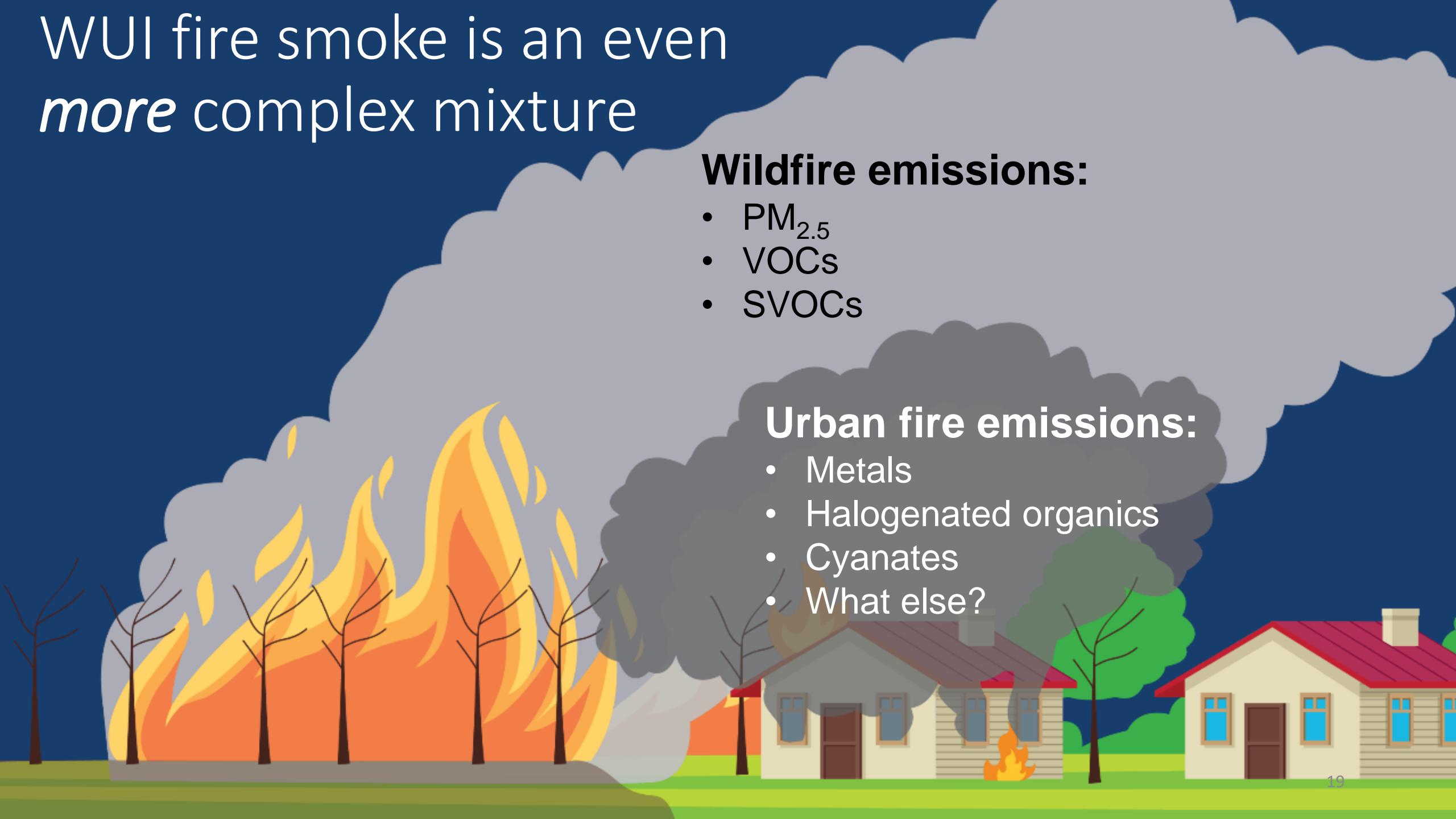
# WUI fire smoke is an even *more* complex mixture

## Wildfire emissions:

- PM<sub>2.5</sub>
- VOCs
- SVOCs

## Urban fire emissions:

- Metals
- Halogenated organics
- Cyanates
- What else?



# 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



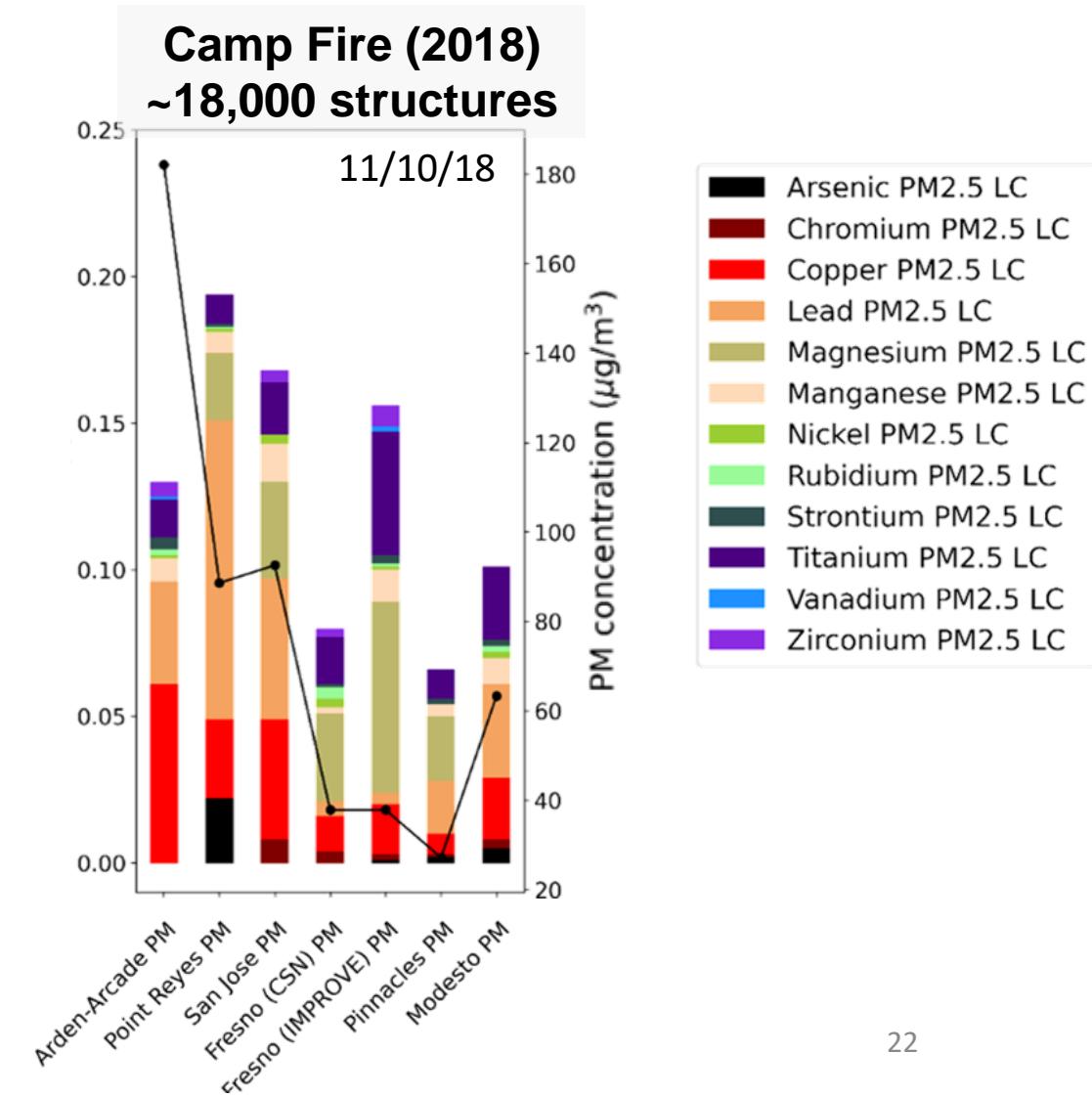
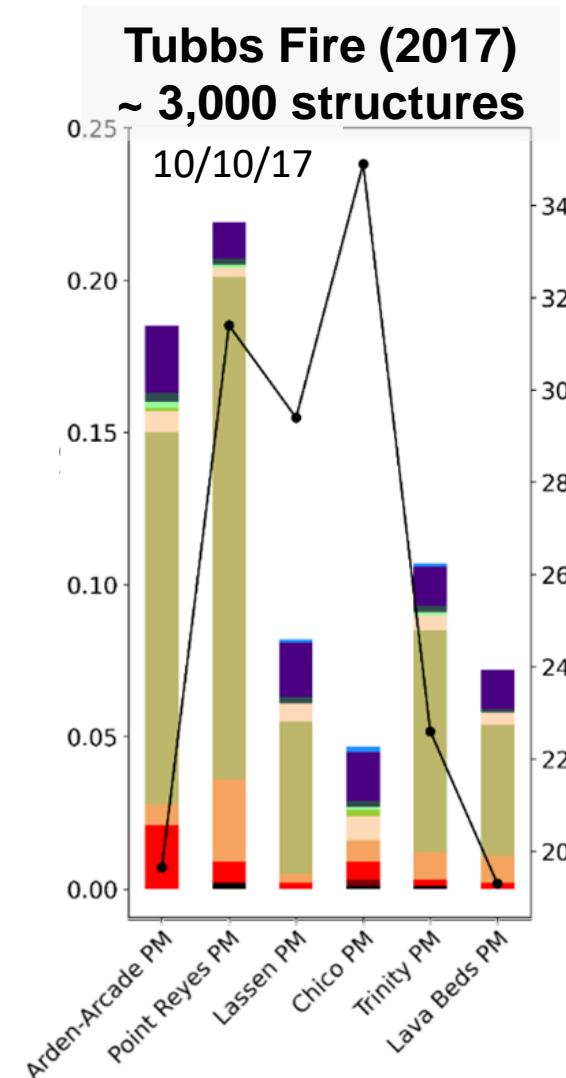
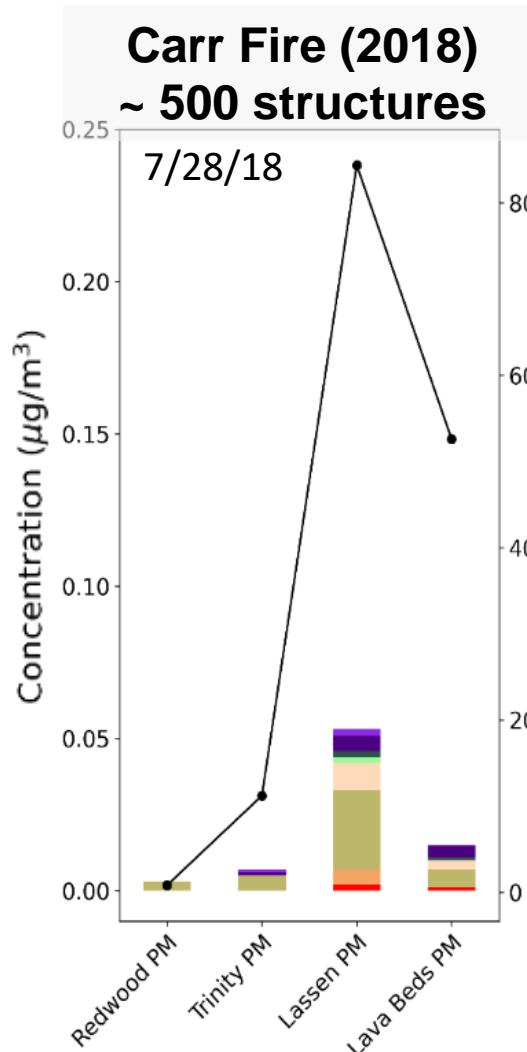
# Some suspected emissions from WUI fires



Anticipate greater amounts of some species seen in laboratory studies:

- Hazardous organic pollutants (dioxins, furans, flame retardants, polychlorinated biphenyls)
- Toxic gases (HCl, HF, Phosgene,  $\text{NH}_4$ ), Isocyanates
- Toxic metals (e.g., As, Pb, Sb, Cu, Zn, and Cr)

# Metals might be a useful WUI fire “Fingerprint”



# Possible sources of metal emissions

	Arsenic PM2.5 LC
	Chromium PM2.5 LC
	Copper PM2.5 LC
	Lead PM2.5 LC
	Magnesium PM2.5 LC
	Manganese PM2.5 LC
	Nickel PM2.5 LC
	Rubidium PM2.5 LC
	Strontium PM2.5 LC
	Titanium PM2.5 LC
	Vanadium PM2.5 LC
	Zirconium PM2.5 LC

Wood preservatives, pesticides  
Alloys, fixtures, tanning, pigments  
Plumbing, electrical  
Plumbing, solder, pigments, batteries  
Alloys, pyrotechnic, fire retardant, fertilizers  
Alloys, fungicide, catalyst  
Alloys, batteries, coins, catalyst  
Electronics/optics, pyrotechnics  
CRT glass, pyrotechnics  
Alloys, high temperature components, pigments  
Alloys, pigments  
Consumer products (cosmetics, anti-perspirants, plastics)

# 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
  - We know almost nothing about the “fuels” in these fires
  - 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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