Health Effects of Wildfire Smoke
Need for Research and Application

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International Association of Wildland Fire
13th International Wildfire Safety Summit &
4th Human Dimensions of Wildland Fire

Boise Center
Boise, Idaho
April 20, 2015
Wildland Fire Smoke & Populations
Regional Impacts on At-Risk Populations

- Russia 2010
- Sydney Australia 1994
- Indonesia 1997
- Victoria Australia 2009
- California 2007
- Canada 2003
- Russia 2010
Health Effects of Wildland Fires
A Personal (Occupational) Issue

Whitewater-Baldy Complex, Gila National Forest, New Mexico, May, 2012

URL: lance-modis.eosdis.nasa.gov/cgi-bin/imagery/firemaps.cgi
Air pollution events from forest fires and emergency department attendances in Sydney, Australia 1996–2007: a case-crossover analysis

Fay H Johnston, Stuart Purdie, Bin Jalaludin, Kara L Martin, Sarah B Henderson and Geoffrey G Morgan
Constituents of Wildfire Smoke

- Particulate matter
- Trace gases
- VOCs
- Ozone
- CO
- Air toxics
- Hg

Cascade Complex, Idaho, 2007

doi:10.1038/ncomms3122

https://picasaweb.google.com/lh/photo/-ZF6POTn-Q0Dubaw7GCsktMTjNZETYmyPlvj0liipFm0?full-exif=true
• 10% increase in all-cause mortality/10 µg/m³ elevation in long-term average PM$_{2.5}$.
• Mortality risk due to CVD ≥ all-cause, ranging from 3% to 76%.

Increased Particulate Air Pollution and the Triggering of Myocardial Infarction

N= 772 patients with MI in Boston area during 1 yr (case-crossover)

**OR of 1.48:** with combined 20 µg/m³ ↑1 day prior

*Long-term PM Exposure*
(American Cancer Society II)

n=300-500,000 (16 yrs)

**Mortality CV > pulmonary**

1 day ↑10µg/m³ → ↑ mortality by 1%

1 death / day / 5 million people

**Long-term ↑10µg/m³ → ↑ mortality by 10%**

1-2 deaths / day / 1 million people

Risk similar to BMI 30-35 kg/m²

Brook RD, et al. AHA Scientific Statement: PM and CVD 2010
PM Triggers Heart Attacks
Lower exposure associated with lower risk

Population Attributable Fractions (PAF)
Related to: the strength of the association between exposure to a risk factor and the prevalence of this risk factor within the population

Where should “Wildland Fire Smoke” exposure be placed on this graph?

Modified from Nawrot et al. Lancet 2011
Health effects known or suspected to be caused by wildfire smoke:

- All-cause mortality
- Asthma & COPD exacerbations
- Bronchitis & pneumonia
- Childhood respiratory disease
- Cardiovascular outcomes
- Adverse birth outcomes
- Anxiety
- Symptoms such as: eye irritation, sore throat, wheeze and cough
**Epi Studies & Health Outcomes**

**Studies with Positive Associations (in %)**

Liu et al. A systematic review of the physical health impacts from non-occupational exposure to wildfire smoke. *Environmental Research 2015*
Susceptible populations include –

- Unborn children
- Children
- Adults 65 years of age and older
- Populations with pre-existing respiratory disease
- Populations with pre-existing cardiovascular disease
- Populations with lower socio-economic status

Populations suspected to be at greater risk –

- Populations with chronic inflammatory diseases (e.g., diabetes, obesity)
- Populations with specific genetic polymorphisms (e.g. GSTM1) that mediate physiologic response to air pollution
Health Effects of Inhaled PM
Proposed Mechanisms

Circulating Constituents
Blood
- PM or constituents in the circulation
  - UFP, soluble metals, organic compounds

Bronchioles/Alveoli
- Pulmonary oxidative stress & inflammation
- PM

Systemic Oxidative stress and Inflammation
- CELLS: ↑ activated WBCs, platelets, myeloperoxidase, Plt-MΦ
- CYTOKINES: ↑ IL-1β, IL-6, TNF-α
- OTHER: ↑ ET, histamine, ? Microparticles, ox-LDL, dysFx HDL

Direct actions
- Acute phase response
  - ↑ Clotting factors: Fibrinogen, CRP
  - Activated or Inflamed liver
  - ↑ Adipokines (PAI-1, Resistin)

ANS imbalance
- ↑SNS / √PSNS

Neural Response
- ANS
- "SPILL-OVER"

Blood Flow from Lung to Systemic Circulation
- Activation of lung ANS reflex arcs

ACUTE: Endothelial dysfunction, Vasoconstriction, Plaque instability, Coagulation, Thrombosis, Arrhythmias
CHRONIC: LV hypertrophy, Atherosclerosis, Arterial Stiffness, Metabolic Syndrome: HTN, Insulin resistance, Dyslipidemia
Subclinical Effects of Inhaled Wood Smoke
Controlled Human Exposure Studies

Healthy subjects. WSP vs FA for 2 hrs with exercise. PM 485 µg/m³. 20hr
• Increase in systemic & lung inflammation

Healthy subjects. 4-hr. PM ~260 µg/m³.
• NO & malondialdehyde levels in breath condensate
• urinary excretion of free 8-iso-PG2α
• Serum Clara cell protein
• serum amyloid A
• factor VIII in plasma factor VIII/vWF ratio

Unosson et al. *P&FT* 2013
Healthy subjects. Wood smoke (PM, 314 µg/m³) or filtered air for 3 hr intermittent exercise.
• Decrease in HRV
• Increase in central arterial stiffness

Pulmonary oxidative stress & inflammation
Bronchioles/Alveoli
PM
Pulmonary oxidative stress & inflammation
SYSTEMIC “SPILL-OVER”

Neural Response
ANS

ANS imbalance
↑SNS / ↓PSNS

Acute phase response
↑ Clotting factors
Fibrinogen, CRP

↑ Adipokines
(PAI-1, Resistin)

Activated or Inflamed fat

Activated or Inflamed liver

CARDIOVASCULAR INFLAMMATION + ROS/RNS

Unosson et al. *P&FT* 2014
No evidence of increased thrombus formation or impaired:
• vascular vasomotor function
• fibrinolytic function


Healthy subjects. WSP vs FA for 2 hrs with exercise. PM 485 µg/m³. 20hr
• Increase in systemic & lung inflammation
Adetona O, et al. *Inhal Tox* 2011

- No significant differences in the across workshift changes on burn days for all the spirometry measures
- Cumulative exposure to woodsmoke was associated with slight decrements in lung function among the wildland firefighters


- Exposure to PM$_{2.5}$ and CO was higher when firefighters performed ‘holding’ tasks compared with ‘lighting’ duties
- Exposures to CO and LG were higher when predominantly pine vegetation was burned
- Exposures to pollutants at prescribed burns may be impacted by non-woodsmoke sources
Subclinical Effects of Inhaled Wood Smoke Controlled Human Exposure Studies

Pulmonary oxidative stress & inflammation

Bronchioles/Alveoli

PM

Activation of lung ANS reflex arcs

PM

Systemic Oxidative stress and Inflammation

Acute phase response

Clotting factors

Fibrinogen, CRP

↑ Adipokines

(PAI-1, Resistin)

Activated or inflamed liver

CARDIOVASCULAR INFLAMMATION + ROS/RNS

Firefighters working at prescribed burns. Inflammatory biomarkers, PM$_{2.5}$, CO. Cross-work shift differences: IL-1β, IL-8, CRP, SAA, ICAM-1, and VCAM-1. Lighting fires had the largest cross-work shift increase in IL-8.
Health Effects of Wildland Fires

Health Threats & Mitigation

At-Risk Populations

Water and Land Pollution

Toxicology of Wildland Fire Smoke

Resources and Access

Communication

Health Effects

Finlay SE et al. PLOS Current Disasters 2012 18
Rationale for the public health messaging to decrease exposure to air pollution

Dose = Concentration x Ventilation rate x Time

C - be active outdoors when air quality is better
V - take it easier when active outdoors
T - spend less time being active outdoors
Public Health Recommendations

Exposure Reduction Measures

An individual can be advised to:

– Stay indoors
– Reduce outdoor physical activity
– Respirators (e.g., N-95) can help with short-term exposures
– Activate asthma/COPD action plans
– Use a home clean air shelter

A community can be advised to:

– Cancel outdoor events
– Provide community clean air shelters
– Increase air filtration in institutions
– Evacuate

Elliott CT. Guidance for BC Public Health Decision Makers During Wildfire Smoke Events. 2014
Protecting Public Health
Research Needs and Opportunities

• **Acquire a greater understanding of the toxicology of wildfire emissions alone and in combination with urban pollutants**
  - Differential toxicity of fuel types and combustion conditions
  - Relative contributions of the pollutant mix (PM, gases, VOCs, etc.)
  - Interaction with urban co-pollutants
  - Biological mechanisms for adverse health effects (Adverse Outcome Pathways)

• **Adapt new technologies to advance smoke surveillance, forecasting & exposure assessment**
  - Satellite-based models, chemical transport and dispersion models

• **Large-scale studies are needed to:**
  - establish more reliable estimates on health impact of wildfires
  - identify intrinsic factors that increase an individual’s susceptibility to wildfire smoke
  - Identify socio-demographic factors increasing a community’s susceptibility to wildfire smoke-related health responses
  - Identify at-risk communities and populations for policy assessment

Protecting Public Health
Research Needs and Opportunities

- **Multiple-episode fire events are needed to:**
  - identify consistency of an association over time
  - change in vulnerability or behavioral adaptation (e.g., remaining indoors) to wildfire smoke exposure.
  - If possible need long-term studies

- **Estimates of health effects of short-term exposures**
  - Health data from short-term exposure (<24 hrs) is needed to guide public health policy and decision-making

- **Estimates of future wildfire-related health impacts**
  - Integrate the probability and severity of wildfire events with health characteristics and resilience of communities likely to be affected
  - Develop models that include changes in parameters sensitive to climate-change
  - Less-developed regions (e.g. tropical regions)

• Studies in wildfire-affected but less-developed regions

• Develop, Harmonize, Implement and Evaluate Impact Public Health Communication on Health Effects
  - Link wildfire smoke forecasts to public health messaging to decrease exposure particularly among those that at greatest risk
  - Evaluate the effectiveness of interventions to decrease wildfire smoke exposures and associated adverse health outcomes
  - Evaluate the effectiveness of public service announcements (PSAs) and other communication methods
Health Effects of Wildfire Smoke
Aggregated effects at a Population Level

Subclinical Effects with No Symptoms
(e.g. asymptomatic decrease in lung function, heart rate variability or endothelial function)

Size of Population Affected by Exposure to Smoke

Deaths
Hospitalization
ED, Urgent Care, & Physician Office Visits
Restricted Activity Days
Respiratory, Cardiovascular, Other Symptoms, and/or Medication Use

Should we be concerned about this group?
Wayne Cascio, MD

- No conflicts of interest

- The presentation represents the opinions of the speaker and does not necessarily represent the policies of the US EPA
Thank you

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Wildfire Smoke & Health Outcomes
A Burden to Health and Wellbeing.

- Ophthalmic symptoms (N=5, 100%+)
- Respiratory
  - Asthma (N=5, 80%+); PEFR (N=2, 100%+)
  - Rescue medication use (N=3, 100%+)
  - Other respiratory diseases (N=37, 95%+)
- Mortality (N=13, 69%+)
- Birth weight (N=3, 67%+)
- Cardiovascular (N=14, 42%+)
- Bone marrow (N=1, 100%+)
- Blood marker concentration (N=1, 100%+)

Prevalence of Cardiovascular Disease
Adults ≥20 years old by Age and Sex

(National Health and Nutrition Examination Survey: 2009-2012)

Age is a strong determinant of cardiovascular disease

Mozaffarian et al. Circulation 2015;131:e29-e322
### PM$_{2.5}$ vs Wildfire Smoke

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<tr>
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*Sources: US EPA, IARC*
“Air pollution should be viewed as one of several major modifiable risk factors in the prevention and management of cardiovascular disease.”

“Health professionals, including cardiologists, have an important role to play in supporting educational and policy initiatives as well as counseling their patients.”
HEPA Filtration Improve Vascular Function

Wood smoke impacted community - British Columbia

Allen et al. AJRCCM 2011