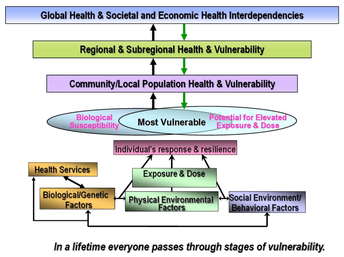
In and Beyond the Smoke: Human Health Considerations for Wildfire Risk, Mitigation and Adaptation Strategies

Helene G. Margolis, M.A., Ph.D.,[1] Jerrold A. Last, Ph.D., [1] Kent E. Pinkerton, Ph.D. [1, 2]
NIEHS T32 Fellows[3]: Doug Banda, Sara Carratt, Mary Corrigan, Samantha Faber, Cameron Flayer, Michelle Kossack, Kyla Walter, Rui Zhang
University of California, Davis Schools of [1] Medicine and [2] Veterinary Medicine, [3] Graduate Groups in Chemistry, Genetics, Pharmacology and Toxicology

Introduction

Human populations having to navigate the hazards of wildfires is not new. The degree to which wildfires pose a hazard to human populations and the ecosystems upon which they depend ***is*** new and unprecedented. Climate change and variability and the resultant environmental sequelae, coupled with human activities, are increasing the frequency, intensity, duration, locations and size of wildfires in California (and across the U.S. and globally).

To evaluate the potential health impacts of wildfires, the factors that may contribute to individual and population risk, and alternate solutions and points of intervention to diminish the risk it is helpful to frame the problem holistically in the context of an environmental health model of vulnerability (Figure 1). Vulnerability factors and their relative importance may differ at the individual and population levels and at different geographical scales or geopolitical domains, and there can be cross-scale interactions among factors. Furthermore, the presence and importance of a given factor or factors can change over time, affecting one or more scales differently. Importantly, this health/disease system interacts (including feedbacks) with multiple climate and earth systems.



In a lifetime everyone passes through stages of vulnerability.

Figure 1 *Environmental Health Multiple-Determinants Model of Vulnerability.* Individual and population vulnerability for occurrence and severity of acute or chronic health outcomes is a function of complex interrelationships among *biologic factors,* including those that confer innate biologic sensitivity and/or resilience to an environmental insult (e.g., sex, race/ethnicity, obesity/overweight, oxidative stress, nutritional status, comorbidities and related treatments, and genetics/epigenetics); *physical environment and exposure characteristics* (e.g., physical/chemical nature of the exposure, duration and dose, coincident environmental stressors (such as water and/or food scarcity, air pollution)); and the *behavioral/lifestyle, social and economic factors* that may influence (or be associated with) both biologic response and exposure (e.g., physical activity, social isolation, disparate neighborhood exposure levels, access to healthcare).

Objective: To evaluate from a transdisciplinary perspective climate change-related increased occurrences of wildfires in California and the implications for the independent and joint effects on public health and ecological health. Key questions evaluated included:

- Do the health effects of wildfire smoke differ from those due to other combustion source emissions? If so, in what ways and why?
- Is toxicity of wildfire smoke different from that of other combustion sources?
- What are the implications, efficacy, and potential unintended adverse consequences of practices, policies, and adaptation and mitigation strategies in place or proposed to manage the increased risk of wildland fires and the 'downstream' consequences on human and ecosystem health.
- What are the gaps in knowledge?

Methods

Setting: Under the auspices of the UC Davis NIEHS predoctoral training program in environmental health sciences, in early summer 2015 an interdisciplinary team of UC Davis faculty and graduate student Fellows tackled the issue.

Approach: Review of *(a)* scientific literature on the health effects of wildfires and toxicity of effluent constituents, *(b)* strategies and policies for fire prevention and suppression, and *(c)* strategies for public health protection.

Results/Discussion

- Wildfire smoke-associated pollution exposures:
  - Are often dispersed across large geographic areas (Figure 2), posing a threat to human health in rural and urban locations.
  - Differ depending on fuels, location (wildland vs neighborhood) and burn conditions.
  - Are typically reported as an increase in mass of particulate matter (PM) 2.5 microns (or 10 microns) in diameter ($PM_{2.5}$ or PM_{10}). PM mass concentrations do not provide complete information on potential toxicity.
    - In mouse model PM from wildfire smoke more toxic than PM from ambient air.[1]
    - Combustion and pyrolysis of biomass and anthropogenic materials in a wildfire can generate chemical products that can cause irritation, asphyxiation, incapacitation, and/or systemic toxicity. In addition to inorganic gases, e.g., carbon monoxide (CO), and oxides of nitrogen (NOx), chemicals of priority concern are: oxygenated organics, e.g., formaldehyde and acrolein; hydrocarbons, e.g., Polycyclic aromatic hydrocarbons (PAHs); Polychlorinated dibenzo-p-dioxins and furans (PCDD/F); cellulose breakdown products, e.g., anhydrous monosaccharides; and a wide array of chemicals from anthropogenic sources.

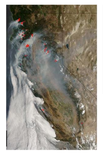


Figure 2

Figure 3

- Wildfire emissions contribute to formation of secondary pollutants, e.g., ozone (O_3).
- Imminent risk to human health and property of a raging wildfire (Figure 4) justifies use of chemical fire retardants and wetting agents (Figure 5). However, the potential human health and ecosystem hazards posed by those chemicals are understudied.

Figure 4

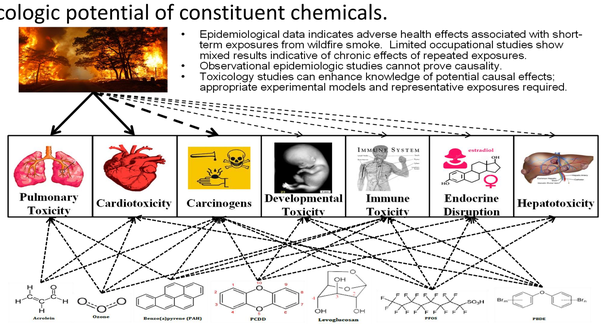
Figure 5

  - Fertilizer-based retardants (nitrates, phosphates, ammonium, ferrocyanide) can lead to eutrophication and other habitat damage, fish kills and other impacts on fauna and flora, contamination of aquifers, and loss of resources and ecosystem services.
  - Common surfactants in wetting agents are perfluoroalkylated compounds. Toxicological profiles of perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) have been most studied. Their potential as developmental, immuno-, neuro-, and hepato-toxicants, as well as carcinogens and endocrine disruptors have been investigated in animal models. [2, 3]
  - Epidemiologic evidence linking these chemicals to human health outcomes is inconsistent.[2] PFOS has been included in the Stockholm Convention on Persistent Organic Pollutants,[4] and PFOA has been recommended by the USEPA to be considered as a likely human carcinogen.[5]
  - Their high-volume usage in California when combating wildfires increases their liability in terms of human health and ecological impact. The associated risks need to be further studied and appropriate actions taken. The federal government has adopted a policy to limit use of fire retardants near waterways and other avoidance areas in consideration of their impacts on habitats and protected species. California has not.
- Epidemiologic studies have focused on acute effects of transient short-term exposures (e.g., non-accidental deaths,[6] emergency room visits,[7] respiratory and cardiovascular events[8]). Most studies have relied on routine community monitors and have not characterized chemical constituents of exposures. Chronic health effects studies have been conducted in occupational cohorts. For the general population chronic effects due to wildfire-related repeated short-term or longer-term exposures have not been studied. Chronic respiratory effects in children have been associated with ambient criteria pollutants (combustion and O_3).[9, 10] It is likely more frequent and longer duration exposures to wildfire smoke will contribute to those and yet to be defined chronic effects in children and adults.
- Exposures to wildfire effluents and to chemicals used to combat wildfires can occur via multiple media (e.g., air, water, soil, food) and multiple routes (e.g., inhalation, ingestion, dermal).

Conclusions

The direct and indirect impacts of wildfires on human health[11] and the societal impacts may be underappreciated.

There are notable data gaps on the potential short-term or chronic health effects associated with one or repeated exposures to wildfire smoke, as well as exposures to the chemical agents used to fight fires. A robust characterization of human exposures (via multiple media and routes) and the resultant health impacts of the complex pollutant mixtures associated with wildfire events does not exist, despite the toxicologic potential of constituent chemicals.



Cross-sector strategies to reduce occurrence and intensity of wildfires, and to reduce wildfire-related exposures should be emphasized. Strategies should factor in the environmental fate of chemicals and 'downstream' consequences on human and natural systems, and the forward and feedback processes that interconnect those systems.

Efficacy of strategies is dependent on multi-directional communications among diverse stakeholders and the public. Enhanced communications across geopolitical scales and domains, and adequate transfer of resources to local government, trusted community-based (e.g., faith-based) organizations can help insure vulnerable populations are protected and/or have the information and resources to protect themselves. In addition to toxicant exposure effects, the severe psychosocial and related health impacts of temporary or permanent population displacement must be proactively addressed.

Acknowledgments

Special note of appreciation to Dr. Pamela Lein and Dr. Michael Denison for their support and leadership.

This work was supported by the National Institute of Environmental Health Sciences T32 Training Grant (T32 ES007059; PI: P. Lein).

The NIEHS training program at UC Davis offers interdisciplinary predoctoral training in environmental health science. Areas of research focus in this training program are: (1) Cancer; (2) Endocrine and Metabolic Mechanisms of Toxicity; (3) Genotoxicity and Epigenetics; (4) Neurotoxicology; and (5) Respiratory Toxicology. Trainees are recruited from several graduate groups that provide disciplinary training relevant to environmental health sciences: toxicology, exposure assessment, epidemiology, cell and molecular biology, neuroscience and pathophysiology.
Additional information is available at: http://niehs.etox.ucdavis.edu/

References (*Selected*)

1. Wegesser TC, Pinkerton KE, and Last JA (2009). California wildfires of 2008: coarse and fine particulate matter toxicity. Environmental Health Perspectives 117(6): 893-897.
2. Lau C, Anitole K, Hodes C, Lai D, Pfahles-Hutchens A, and Seed J (2007). Perfluoroalkyl acids: a review of monitoring and toxicological findings. Toxicological sciences: an official journal of the Society of Toxicology 99(2): 366-394.
3. Mariussen E (2012). Neurotoxic effects of perfluoroalkylated compounds: mechanisms of action and environmental relevance. Archives of toxicology 86(9): 1349-1367.
4. Secretariat of the Stockholm Convention (2009). Governments unite to step-up regulation on global DDT reliance and add nine new chemicals under international treaty. Press Release.
5. Renner R and Christen K (2006). Scientists hail PFOA reduction plan. Environmental science & technology 40(7): 2083.
6 Johnston F, Hanigan I, Henderson S, Morgan G, and Bowman D (2011). Extreme air pollution events from bushfires and dust storms and their association with mortality in Sydney, Australia 1994–2007. Environ Res 111(6) 811-816.
7 Johnston FH, Bailie RS, Pilotto LS, and Hanigan IC (2007). Ambient biomass smoke and cardiorespiratory hospital admissions in Darwin, Australia. BMC Public Health 7:240.
8. Delfino RJ, Brummel S, Wu J, Stern H, Ostro B, Lipsett M, Winer A, Street DH, Zhang L, and Tjoa T (2008). The relationship of respiratory and cardiovascular hospital admissions to the southern California wildfires of 2003. Occupational and Environmental Medicine.
9. Gauderman WJ, Avol E, Gilliland GF, Vora H, Thomas D, Berhane K, McConnell R, Kuenzli N, Lurmann F, Rappaport E, Margolis HG, Bates D, Peters JM. The effect of air pollution on lung function development in children aged 10 to 18 years. New Engl. J. Med. 351(11):1057-67, 2004.
10. McConnell R, Berhane K, Gilliland F, Islam T, London SJ, Gauderman WJ, Avol E, Margolis HG, Peters JM. Sports and asthma in children exposed to ozone. Lancet 359 (February 2): 386-391, 2002.
11. Weinhold B (2011). Fields and Forests in Flames: Vegetation Smoke and Human Health. Environmental Health Perspectives 119 (9):A386-A393.