Assessing the exposure of high mountain lakes to ammonia from wildfire smoke as a source of reactive nitrogen

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Abstract:
Wildfire smoke is a major source of ammonia (NH_3), and smoke is becoming increasingly abundant as fires burn larger areas. Nitrogen deposition provides an important source of nutrients to high elevation watersheds, but alongside increased temperatures, can result in harmful levels of algal primary production and eutrophication. In this study, we examine warm-season (March-October) smoke exposure and NH_3 concentrations from 2013-2023 across the U.S. Rocky Mountain (RM) region (Idaho, Colorado, Montana, Wyoming, Utah, and New Mexico) with a focus on high elevation lakes. We identify lakes larger than 2 ha and above 2000 m using the NHDPlus High Resolution dataset and then assess smoke and NH_3 exposure. We identify smoke-impacted lakes and plume concentrations using the National Oceanic and Atmospheric Administration's Hazard Mapping System (HMS) smoke product. To determine surface smoke exposure, we couple the HMS data with in situ fine particulate organic matter concentrations from the Interagency Monitoring of Protected Visual Environments (IMPROVE) network. We also combine the HMS smoke data with daytime NH_3 retrievals from the Cross-track Infrared Sounder (CrIS) satellite instrument and compute oversampled seasonal NH_3 concentrations that include and exclude smoke. On average, from 2013-2023, high elevation RM lakes were exposed to smoke in the column (surface) on 10-24% (5-17%) of warm-season days and the percentage of lake smoke days has increased by 2.26% per year. Smoke's contribution to seasonal NH_3 concentrations can be substantial along primary smoke transport pathways (e.g., high latitudes) in high smoke years–rivaling local agricultural emissions.

Air Quality Perception and Improvement Strategies in Congregate Living Facilities

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Background: Poor indoor air quality from wildfire smoke exposure in the Rocky Mountain Region threatens public health for vulnerable populations such as older adults in congregate living facilities and the staff that work there. Indoor air filtration can be an effective tool to improve air quality, but little research exists on factors for successful implementation in congregate living facilities.
Methods: We surveyed staff from congregate living facilities in Colorado, Idaho, and Montana to assess perceptions of air quality risks, current strategies for managing indoor air, and the feasibility of using an air quality monitoring website and high-efficiency particulate air filters. The online survey included up to 25 questions and was distributed through existing professional networks. Descriptive statistics and simple cross-tabulations were used to analyze the data.
Results: Among 36 participants, 86% viewed air quality as a health concern. Common strategies to improve air quality during days with poor air quality included closing windows (75%) and limiting outdoor time (69%). Half of the facilities (50%) reported having no emergency plan for poor air quality. Most participants (74%) indicated that an air quality website would be "very helpful," and 53% said the same for air filters. Cost was the most frequently cited barrier to implementation. Staff who perceived air quality as a "big" health issue were the most likely to respond that they would use these tools.
Conclusions: Staff in congregate living facilities are willing to use air quality tools, but cost and lack of awareness and resources remain significant barriers.

Assessing Wildfire Smoke Particulate-Induced Alterations in Reproductive Neuropeptide Expression: An In Vitro Model

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Wildfires have become increasingly intense and frequent due to climate change. Epidemiologic studies have linked wildfire smoke exposure to negative reproductive health outcomes, but the mechanisms remain unclear. This project uses cell culture to investigate how wildfire smoke may influence neuroendocrine function as a precursor to adverse reproduction. We hypothesize that exposure to wildfire smoke particulate matter will disrupt the normal function of the neuroendocrine system as indicated by altered production of neuropeptides kisspeptin, neurokinin B, and dynorphin in KNDy neurons.

To assess the effects of wildfire smoke, we leverage an immortalized murine KNDy neuron. These cells are then exposed to extracted wildfire smoke particulate at varying concentrations and timepoints. Following exposure, RNA is extracted and analyzed using quantitative polymerase chain reaction (qPCR) to evaluate gene expression. We are using qPCR to examine the expression of three genes/neuropeptides involved in regulating reproductive function, which include Tac2/neurkinin B, Kiss1/kisspeptin and PDyn/prodynorphin.

By quantifying the mRNA expression levels of these genes, we aim to determine how KNDy neurons respond to varied doses and durations of wildfire smoke exposure. This will help clarify the pathways through which smoke disrupts reproductive health and expand our understanding of environmental influences on the hypothalamic-pituitary-gonadal axis.

By investigating this novel connection, we hope to expand the field's understanding of how wildfire smoke interferes with neuroendocrine control of reproductive function. This knowledge could ultimately support future efforts to develop therapies to protect reproductive health in the face of increasing wildfire smoke exposure.

Kevin Ridgway

With nearly 50 million U.S. houses located at the wildland-urban interface (WUI) and wildfire activity increasing, there is growing concern that structure fires may be an important but underexplored source of air pollutants. Aerosol emissions from such fires remain poorly characterized. The Burning Homes And Structural Materials (BHASMA) campaign investigated emissions from a wide range of structural fuels under different combustion modes (pyrolysis, flaming) and fire scales (0.5–250 kW) to quantify fuel-specific emission factors.

Nearly 100 experiments were conducted with over 17 fuels, including lumber (southern yellow pine), engineered wood products (oriented strand board), insulation (polyurethane foam), textiles (polyester carpet), conduit (polyvinyl chloride), and siding (cement fiberboard). Mixed-fuel burns, designed to mimic residential structures, were compared with individual fuels.

Emission factors varied strongly with fuel type and combustion efficiency, often by 1–2 orders of magnitude for bulk pollutants such as PM2.5 and organic aerosol (OA). Incorporating non-lumber materials at 30% of structural mass approximately doubled PM2.5 emissions, tripled OA, and increased elemental carbon (EC, soot) nearly fivefold. Ongoing work is identifying fuel-specific chemical markers through analysis of ions, saccharides, and elemental species (e.g., ammonium, levoglucosan, zinc, chromium, iron) to distinguish biomass from non-biomass fuels. Comparisons with published emission data will provide further context.

These findings highlight that structural materials can substantially amplify air pollutant emissions from WUI fires. Improved understanding of fuel-specific emissions and chemical signatures will support more accurate assessments of exposure risks for first responders, evacuees, and downwind communities.

Smoke3d – Wildfire Smoke Transport Modeling with Adaptive Mesh Refinement

Patricia Azike

We focus on the use of representers to decouple the Euler–Lagrange equations that arise when errors are present in the dynamics, initial conditions, and observational data during the simulation of wildfire smoke transport. The representer method is chosen for its ability to reduce computational cost by transforming the problem from the high-dimensional state space to the typically much smaller data space. This reduction yields two equations: a forward equation and a backward (adjoint) equation. Our approach leverages adaptive mesh refinement (AMR), enabling the forward and adjoint equations to be simulated on different adaptive meshes. In particular, the adjoint equation benefits from AMR because its source term, a delta function, requires only localized refinement. This work forms part of a larger wildfire smoke modeling framework, Smoke3d, which we will use for our simulation and estimation of PM2.5 concentrations.

Simulated wildfire smoke exposure elicits an inflammatory and oxidative response in Raw264.7 macrophage lung model of the lung-brain axis

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Background
Wildfire smoke (WFS) is a major source of ambient particulate matter (PM) exposure and has been associated with adverse respiratory and neurological outcomes, including increased inflammation. However, the cellular mechanisms linking WFS exposure to inflammatory signaling within the lungs and periphery and how this in turn affects the blood-brain-barrier and finally the brain itself remains unclear.

Methods
Here, we established our simplified lung exposure model of the lung-brain axis and evaluated the effects of $PM_{2.5}$ generated from a 50:50 mixture of Eucalyptus:Manzanita (E/M)—a common wildfire smoke source in California and Australia, on macrophage activation using the murine RAW264.7 cell line. These macrophages were exposed to a concentration range of 0.01-10 ug/ml WFS $PM_{2.5}$, and outcomes include mitochondrial superoxide, cytokine gene profiling (IL-1β, TNF-α, IL-6) via qPCR, NLRP3 inflammasome expression and p65 phosphorylation through western blotting. Data were analyzed using one-way ANOVA with appropriate multiple-test corrections.

Results
Exposure to E/M WFS $PM_{2.5}$ induced a dose and time dependent increase in proinflammatory cytokines, with IL-1β showing the most consistent and significant upregulation across replicates. Importantly, cell viability remained largely intact at relevant exposure concentrations, indicating that the inflammatory activation was not driven by overt cytotoxicity.

Conclusion
These findings demonstrate that WFS $PM_{2.5}$ robustly activates macrophage inflammatory pathways, with IL-1β potentially being a key mediator. By establishing RAW264 macrophages as a tractable model for WFS-induced inflammation, this work lays the foundation for future studies investigating downstream effects on the blood–brain barrier and neuroinflammatory signaling.

Clear the Air: Respiratory Disease and Air Pollution in Dairy Calves

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Background

Bovine respiratory disease (BRD) is a leading cause of morbidity and mortality in the cattle industry, affecting approximately 22% of dairy calves. Air pollution, particularly particulate matter (PM2.5), is a well-known risk factor for human respiratory disease, yet its impact on calf health is poorly understood. With climate change increasing air pollution events, including wildfire smoke in the western U.S., identifying sensitive models to study respiratory effects is critical.

Methods

We retrospectively analyzed three cohorts of dairy calves over a 10-week period, combining clinical assessments (Wisconsin Calf Health Scoring App) and point-of-care lung ultrasound with local PM2.5 data from EPA Air Quality System monitors. Cumulative exposure and high-exposure days were calculated and analyzed relative to pneumonia risk.

Results

Cumulative PM2.5 exposure varied significantly across cohorts ($F(1, 43) = 212.8$, $p < 0.001$). Higher-than-average exposure increased the risk of pneumonia in the following week (RR = 1.29, CI = 1.024–1.626). Calves with subclinical pneumonia three days before a high-exposure day were more likely to develop clinical pneumonia within three days (RR = 1.64, CI = 1.13–2.38).

Conclusion

PM2.5 exposure, especially in calves with pre-existing lung pathology, contributes to BRD development. Because calves share daily air exposures with agricultural workers, they provide a unique and accessible model for investigating how air pollution affects human respiratory health, offering opportunities for preventive strategies and mechanistic research.

Evaluation of Sperm Parameters in Mice Following a 5-Day Simulated Wildfire Smoke Particulate Exposure.

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Background: Intensifying wildfires are driving increased wildfire smoke (WFS) exposures annually across the globe. Extensive work has documented the cardiopulmonary health disruption of WFS and its subcomponent, particulate matter (PM2.5); however, novel epidemiologic data suggest that WFS exposure is associated with dysfunction of the reproductive and nervous systems. Concentrated ambient PM2.5 has been linked to suppression of the hypothalamic-pituitary-gonadal (HPG) axis in male mice via inflammatory signaling, however, the effect of smoke-derived PM2.5 on the HPG axis has been relatively understudied.

Methods: Adult male C57BL/6 mice were exposed to simulated WFS in two cohorts to assess impacts on the reproductive system. Animals received 50 µL per day of vehicle control, Douglas-fir smoke extract, or eucalyptus/manzanita smoke extract (1 mg/mL) via intranasal installations (n = 6 per group) and sacrificed 24 hours post-exposure.

Results: Sperm motility was significantly decreased by both smoke types. Cell morphology was also significantly impacted by smoke exposure: Total, midpiece and tail defect rates increased significantly across smoke types compared to controls, although the rate of head defects decreased significantly in EM PM2.5-exposed males. Sperm concentrations were not affected by treatment. This data indicates acute WFS exposure is linked to significant decreases in caudal sperm motility, possibly via direct exposure to stored sperm population by WFS or secondary inflammatory mediators.

Conclusion: These preliminary findings motivate studies with a longer exposure duration that is more likely to engage the HPG axis and a more physiologically relevant exposure route (e.g., whole body rather than intranasal) to aid translatability to human exposure levels.

This project has been funded by MAP ERC funding grant #T42OH009229. The authors have no conflicts of interest to disclose.

Astrocytic STING signaling mediates wildfire smoke-induced neurotoxicity *in vitro*

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Concurrent with climate change, global shifts in the frequency and duration of wildfire smoke (WFS) generating events necessitate an assessment of the potential for this toxicant to induce neurologic consequences, motivated by a growing body of literature from our group and others. Additionally, glial cGAS-STING signaling has recently been implicated in the pathogenesis of neurodegenerative disorders given its ability to modulate neuroinflammation in the brain parenchyma. Together, this existing work motivated our present interrogation of STING activation in primary glial cells in the context of WFS exposure. Simulated WFS was generated using a laboratory furnace. Particulate matter was collected in-line during burns and then extracted to characterize particle size and shape, ensuring the relevance of this exposure in a neurotoxic context. Primary murine mixed glial cultures were subsequently liquid suspension dosed with WFS extract and assessed for viability, changes in transcriptomic profiles, secreted protein expression, and STING activation using high-content imaging and fully automated deep-learning based image analysis pipelines. These assays demonstrated significant STING-dependent cytotoxicity, signal transduction, and downstream release of inflammatory cytokines and chemokines into the glial conditioned media (GCM). Primary neurons were further exposed to GCM from WFS-treated glia, demonstrating substantial STING-mediated neurotoxicity. This work motivates subsequent assessment of the role that WFS plays in glial-mediated neuroinflammation and neuroimmune modulation in vivo to further parse out effects of these phenomena in the context of age-related neurodegeneration.

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The extreme wildfire events of 2020 caused widespread elevated fine particulate matter ($PM_{2.5}$) concentrations across the western US. The Colorado Front Range experienced significant air quality deterioration, due to both smoke from local fires and smoke transported from hundreds of kilometers away. The objective of this work is to use the 2020 wildfire smoke events to 1) quantify the spacing between $PM_{2.5}$ monitors needed to resolve smoke plumes in real time, and 2) evaluate the ability of an operational smoke forecast system to warn the public in the context of local versus transported smoke. These are key questions needed to protect vulnerable groups, including outdoor workers. Multiple episodes with elevated $PM_{2.5}$ concentrations along the Front Range occurred over the course of the 2020 smoke season. We leverage EPA regulatory and community low-cost PurpleAir monitoring networks, and the operational High-Resolution Rapid Refresh Smoke (HRRR-Smoke) forecast for our assessment. The PurpleAir network in the region in 2020 indicates that during smoke events the $PM_{2.5}$ concentration in many communities can deviate significantly from the nearest regulatory monitor. We find that PurpleAir sensors need to be spaced within 6 km to capture concentration gradients in smoke when the fires are local. The HRRR-Smoke forecasts perform best when the smoke impacting the Front Range has been transported from further away. For all smoke types, we find that the HRRR-Smoke predicts > 84% of the elevated hourly concentrations to within one $PM_{2.5}$ health effect category.

Scientifically Informed & Socially Responsive: Co-Creation of Relevant and Actionable Materials to Support Decision Making during Poor Air Quality Episodes

Authors: Milena Guajardo, Cassie Archuleta, Lea Schneider, Mindy Hill, Carolina Mendez Torres, Lena Low, Jeff Collett, and Emily Fischer.

Northern Colorado faces three persistent air quality challenges: ozone, wildfire smoke, and air toxics. Our team partnered directly with concerned community members, including those disproportionately impacted by these issues to co-create relevant and actionable materials to support protective decision making. Together, we created materials that include 1) an air quality decision tree, 2) tailored text message alerts, and 3) infographics highlighting nearby sources of air toxics. While these tools are important, the relationships built through this work are equally vital. We've learned that meaningful engagement requires more than information sharing—it demands listening, mutual respect, and recognizing the systemic barriers that prevent communities from simply "making better choices." Academic research teams often enter these community spaces without staying long enough to support lasting change. We aimed to center community voices, acknowledge lived realities, and work alongside local leaders to deepen their knowledge. As a result, we have continued to support the ability of local leaders to serve as trusted sources of information and connect their neighbors to reputable resources.