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Pediatric Cardiovascular Outcomes and the Wildfire Urban Interface (WUI): Pathways, Vulnerabilities, Mitigation, and Monitoring

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Introduction

Wildfire smoke (WFS) is a complex mixture of aerosolized gases that travels many miles and evolves chemically as it ages; increasingly blanketing populations in the wildland–urban interface (WUI). Beyond known respiratory effects, WFS components are dominated by fine particulate matter ($PM_{2.5}$), ultrafine particles (UFPs), nitrogen oxides (NO_x), carbon monoxide, volatile organic compounds (VOCs), and polycyclic aromatic hydrocarbons (PAHs) which have systemic cardiovascular impacts.[1-10] Children are a uniquely vulnerable population due to their higher minute ventilation per kilogram, developing organ systems, less efficient nasal filtration, and behavior patterns that increase exposure.[11-13] While pediatric studies have focused largely on respiratory outcomes, population-level evidence associates wildfire-related $PM_{2.5}$ with increased all-cause and cardiovascular mortality in mixed-age cohorts, demonstrating strong biologic plausibility for pediatric cardiovascular harm.[1,14-16]

Exposure Chemistry and Why It Matters to the Heart

WFS contains PM across size fractions (PM_{10}, $PM_{2.5}$, $PM_{0.1}$), with $PM_{2.5}$ typically comprising >90% of the particle mass; with ultrafine particles (UFPs) demonstrating greater tissue penetration.[4-6] Fresh smoke particles often measure ~100–150 nanometer, enlarging as they age (~200–300 nm) and oxidize, altering reactivity and toxic potency.[5]In WUI events, wildfire emissions can mix with urban/industrial sources, accelerating formation of secondary pollutants such as ozone (O_3) and secondary organic aerosols.[3,7]VOCs and PAHs vary by fuel and burn phase and have independent cardiometabolic relevance.[9-10]

Pathways Linking WFS to Pediatric Cardiovascular Harm

Wildfire smoke affects the cardiovascular system through several interconnected biological pathways. First, oxidative stress and inflammation play a central role as $PM_{2.5}$ and ultrafine particles generate reactive oxygen species that activate transcription factors such as NF-κB and AP-1, driving local and systemic inflammatory cascades with downstream vascular effects.[18-22]

Endothelial dysfunction and thrombosis represent a second pathway. As UFPs translocate across the alveolar barrier, they impair endothelial function, alter vasomotor tone, and promote a pro-thrombotic state that may be especially consequential for children with underlying congenital heart disease.[19,21]

A third pathway involves autonomic imbalance, in which irritant signaling from airway receptors triggers reflex neural responses that may alter heart-rate variability and

increase susceptibility to arrhythmias.[19] Toxicant receptor signaling also contributes, as PAH activation of the aryl hydrocarbon receptor (AhR) modulates immune and inflammatory gene expression in ways that intersect with vascular inflammation and remodeling pathways.[24-25]

Finally, genotoxic and epigenetic effects from PAHs and metals in wildfire smoke, including DNA damage, altered methylation patterns, histone changes, and shifts in microRNA expression, may influence cardiometabolic trajectories across the life span.[26-32] Together, these mechanisms provide strong biological plausibility for cardiovascular harm in children exposed to wildfire smoke, even though pediatric-specific cardiovascular studies remain limited.

What We Know and Don't Know in Children

Population-level data clearly link wildfire-related $PM_{2.5}$ exposure with increases in all-cause and cardiovascular mortality, but pediatric-specific cardiovascular endpoints remain largely under-reported.[1] Most pediatric literature has centered on respiratory outcomes such as increased emergency department visits and asthma exacerbations with cardiovascular effects noted as a critical evidence gap.[16]

Children who are most vulnerable include infants and young children (≤5 years), who experience higher pollutant doses per kilogram and have less mature physiologic defenses.[17] Additionally, children with congenital heart disease, pulmonary hypertension, cardiomyopathy or heart failure, in addition to other comorbidities such as obesity, diabetes, chronic kidney disease, or chronic inflammatory conditions may experience disproportionately higher cardiovascular risk during smoke events.[14-15,18-22,24-25] Mechanistic data from adult and ambient PM studies strongly support these elevated risks, underscoring the need for pediatric-focused research.

Emerging concern: toxic metals in urban-adjacent fires

During recent wildland-urban interface fire events in Los Angeles (**January 2025**), real-time monitors detected lead concentrations exceeding typical levels (>100 times), located many miles downwind from fire origins, and consistent with combustion of older building materials (pre-1970's paint, pipes). Similar spikes were noted during the 2018 Camp Fire. Aerosolized metals like lead can alter DNA activity, immune function and are linked to hypertension as well as other cardiovascular risks. Although long-term cardiac effects of brief but intense airborne lead exposures are not yet well defined, these metals add an additional cardiovascular hazard layer in WUI fires.

Pediatric Cardiovascular Mitigation and Monitoring

As pediatric providers caring for the most vulnerable of populations, the importance of educating and supporting our patients,families and medical subspecialists in preparation for WUI smoke exposures/risks is essential. **Table 1** summarizes evidence-informed environmental and health-system strategies to reduce wildfire smoke exposure and support physiologic stability in children with established or at-risk cardiovascular

disease. It highlights home and school-based air-quality controls, age-appropriate use of pediatric N95 respirators, hydration guidance, and systems-level preparedness measures, such as proactive medication refills, remote monitoring pathways, and tailored “smoke-season” action plans.[33-34,11,35]

Table 1. Practical Mitigation for Children With (or at Risk for) Cardiovascular Disease

Environmental Controls Stay indoors with windows closed when smoke intrudes; use recirculating HVAC and portable HEPA air cleaners to reduce indoor $PM_{2.5}$.[33] School/athletics policies should scale outdoor exertion to smoke severity in WUI communities.[33-34] **Respirators:** When age-appropriate/feasible, a well-fitted pediatric N95 reduces exposure far more than a surgical mask (~80% vs ~20% expected decreases), though fit/comfort/false-reassurance caveats do apply.[11] **Hydration:** Maintain adequate hydration to support mucociliary function and potentially reduce deeper contaminant penetration.[35]
Health-System & Community Readiness
Integrate AQI/$PM_{2.5}$ alerts into clinic messaging; distribute “smoke-season” action plans tailored for cardiac comorbidities.[33-34] Ensure medication refills, access to home monitors (BP cuffs, pulse oximeters where indicated), and telehealth pathways before peak smoke periods.[34]

Pediatric cardiovascular monitoring is an important consideration in the setting of WUI smoke exposure events. Structured approaches are outlined in **Table 2** for clinicians and families to monitor cardiovascular status during wildfire smoke episodes. It includes pre-season risk stratification, home vital-sign and symptom monitoring on smoke-affected days, recommended clinic/telehealth touchpoints, escalation criteria requiring urgent evaluation, and post-event reassessment to detect subacute physiologic changes in high-risk children.[33-34]

Table 2. Pediatric Cardiovascular Monitoring Considerations During Smoke Events

1. Pre-Season Risk Stratification[33-34]
● Flag high-risk diagnoses (e.g., CHD, pulmonary hypertension, cardiomyopathy/heart failure).

- Create individualized Smoke Health Action Plans (contact thresholds, device use, when to seek care); teach families how to follow AQI/PM dashboards and implement environmental controls.

2. Home Monitoring (smoke days)[33]

- **Vitals:** Twice-daily resting heart rate; blood pressure for hypertensive history or age-appropriate patients.
- **Oxygenation:** Spot SpO_2 checks for cyanotic CHD, pulmonary hypertension, or chronic cardiopulmonary disease (monitor for drops from baseline).
- **Symptoms diary:** Fatigue, exertional intolerance, chest discomfort, palpitations/syncope, edema or rapid weight gain (≥0.5–1 kg in 24–48 h may suggest fluid retention in older children with HF).
- **Activity scaling:** Avoid strenuous outdoor activity; prefer indoor light-to-moderate activity with HEPA-filtered air.

3. Clinic/Telehealth Touchpoints

- Tele-check high-risk patients during multi-day smoke episodes to review vitals/symptoms/adherence.

Medication optimization:

- Verify HF regimens (diuretics, ACEi, β-blockers), pulmonary vasodilators, antiarrhythmic adherence; ensure device (pacemaker/ICD) checks are current.

Selective testing:

- If concerning trends/symptoms, consider BNP/NT-pro-BNP, ECG, and targeted echocardiography (especially for HF or pulmonary hypertension risk patients).

4. Same-day Escalation Criteria

- New/worsening hypoxemia
- Syncope/near-syncope,
- Sustained tachycardia or palpitations,
- Chest pain,
- Rapid weight gain/edema
- Signs of respiratory distress in cardiac patients.

5. Post-Event Follow-up

- Reassess symptoms/vitals and, when indicated, cardiac function after prolonged smoke periods to detect subacute changes.

Action plans, which are often associated with disease processes such as asthma, should also be considered for pediatric cardiac or at-risk patients in relation to wildland urban-interface fires. **Table 3** provides a simplified, family-friendly action plan designed for rapid use during wildfire smoke events. It emphasizes key steps such as tracking air-quality metrics, sealing and filtering the indoor environment, selecting and fitting appropriate masks, maintaining hydration, monitoring symptoms and vital signs when instructed, and ensuring medication readiness throughout fire season.[33-34,11,35]

Table 3. Pediatric Cardiac Action Plan

Cardiac Action Plan (Essentials)	
Know the Numbers	Track local AQI/$PM_{2.5}$.[33]
	Activate the plan as thresholds (AQI >100-150) are crossed.[33]
Seal & Filter	Close windows.[33]
	HVAC on recirculate.[33]
	Run HEPA units in sleep and main living areas.[33]
Right Mask, Right Fit	Use pediatric N95 when feasible (>2yrs).[11]
	Avoid outdoor exertion on heavy smoke days.[11]
Hydrate & Rest	Maintain hydration.[35]
	Scale activity appropriately.[35]
Monitor & Message	Log HR/BP (as applicable), SpO_2 if instructed, and symptoms; contact the care team per the plan.
Medication Readiness	Keep 2–4 weeks of cardiac meds and needed supplies during fire season.[34]

Research and Policy Priorities

Key cardiovascular research priorities should include the development of prospective pediatric studies that examine wildfire smoke exposure in relation to blood pressure and heart rate variability, arrhythmias, cardiac biomarkers (BNP/NT-pro-BNP), and acute decompensations in congenital or acquired heart disease. A second priority is to advance pediatric exposure science, including personal exposure monitoring, particularly ultrafine particle measurements and evaluation of indoor air interventions such as HEPA filtration and HVAC upgrades in homes and schools located in WUI regions.[33]

Health-system preparedness also requires attention. Pediatric smoke-response playbooks should be implemented to support families of children with cardiac

comorbidities, incorporating early-warning communication systems, medication logistics, and telehealth capacity during prolonged smoke events.[34] Lastly, because urban-adjacent wildfires can aerosolize metals such as lead, further research is needed to characterize these exposures and clarify their cardiovascular implications for children who are already physiologically vulnerable.

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