Bronchopulmonary Dysplasia (BPD) and the Impact of Wildfire Smoke Exposure: Pediatric Vulnerability in a Changing Climate

by
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Abstract

Bronchopulmonary dysplasia (BPD), is a chronic lung disease of prematurity, representing a sentinel phenotype of pediatric vulnerability in the era of escalating wildfires and climate change. Wildfire smoke (WFS) exposure, driven by climate-associated increases in fire frequency, duration, and intensity, has emerged as a major public health threat across the United States and the world, with disproportionate impacts in California. Millions of children are routinely exposed to particulate matter ≤2.5 μm in aerodynamic diameter ($PM_{2.5}$) at levels exceeding the U.S. Environmental Protection Agency National Ambient Air Quality Standards during wildfire events.[1-4] Children with BPD, who have impaired alveolarization, abnormal pulmonary vascular development, and limited respiratory reserve, are uniquely susceptible to oxidative stress, inflammation, and respiratory decompensation triggered by wildfire-related pollutants.[5] This review synthesizes emerging epidemiologic and mechanistic evidence linking wildfire smoke exposure to respiratory morbidity among children with chronic lung disease, with a focus on BPD. We describe key exposure pathways, pathophysiologic mechanisms, psychosocial and health-equity considerations, and clinical mitigation strategies. A transdisciplinary, anticipatory approach that integrates environmental health, pediatric pulmonology, and community-engaged preparedness is essential to protect this high-risk population as climate-driven wildfire events continue to intensify.[1,4,8]

Introduction

In 2025, Southern California experienced another extreme wildfire season, especially in the wildland–urban interface (WUI) which are occurring more often, burning longer, and spreading farther; over the past decade, California has experienced some of its most destructive seasons on record. This was demonstrated with the Eaton and Palisades fires, which resulted in prolonged $PM_{2.5}$ elevations across large portions of Los Angeles County. Although comprehensive epidemiologic analyses of pediatric health outcomes related to these events are ongoing, early situational assessments from regional pediatric centers, including Children's Hospital Los Angeles (CHLA), raised substantial concerns for medically fragile children, particularly those with chronic respiratory disease such as bronchopulmonary dysplasia (BPD).

Wildfires are increasing in frequency, duration, and severity as a direct consequence of global climate change.[4,9] In the United States, the annual area burned has more than doubled since the 1990s, with California consistently accounting for the largest share of total acreage affected and

population exposure.[4] The 2020 California wildfire season alone emitted an estimated 112 million metric tons of carbon dioxide and produced $PM_{2.5}$ concentrations exceeding World Health Organization air quality guidelines for more than 40 consecutive days in multiple regions.[3,4] Nationally, more than 70 million individuals, including approximately 7 million children, were exposed to wildfire smoke during that year.[4] Pediatric populations, especially children with preexisting lung disease are disproportionately affected due to immature immune and antioxidant systems, higher minute ventilation per kilogram of body weight, and often limited physiologic reserve.[5,8]

Bronchopulmonary dysplasia remains one of the most common chronic respiratory conditions of childhood, affecting up to 40% of infants born before 28 weeks' gestation.[5] Survivors frequently experience persistent pulmonary morbidity characterized by alveolar simplification, airway hyperreactivity, impaired gas exchange, and abnormal pulmonary vascular development.[5] These features render children with BPD particularly vulnerable to wildfire-related air pollutants, including $PM_{2.5}$, ozone, nitrogen oxides, and volatile organic compounds.[1,3,8]

Wildfire Smoke Composition and Mechanisms of Injury

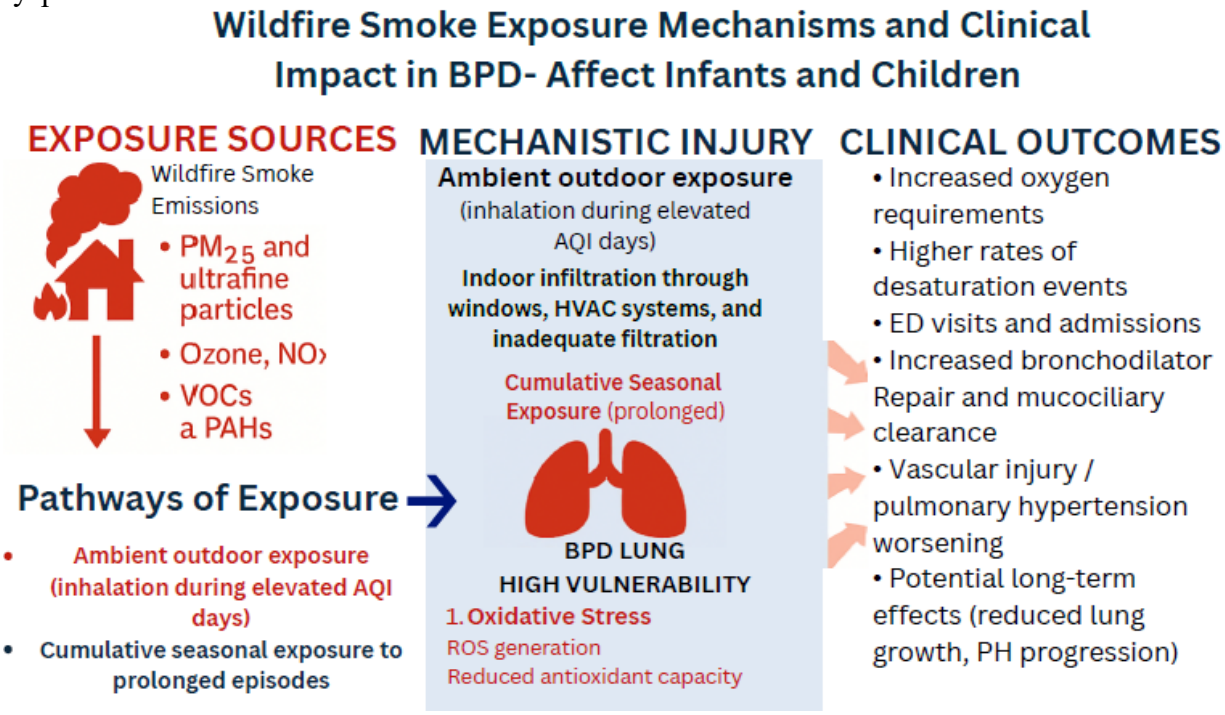
Wildfire smoke is a complex and dynamic mixture of gases and fine particulate matter generated by the combustion of vegetation, structures, and synthetic materials, particularly within wildland–urban interface (WUI) regions.[1,3] Major components of smoke include $PM_{2.5}$ and ultrafine particles, ozone (O_3), carbon monoxide (CO), nitrogen oxides (NO_x), volatile organic compounds (VOCs), in addition to polycyclic aromatic hydrocarbons (PAHs).[1] Structural fires common to WUI events may further introduce metals and other toxic combustion byproducts derived from building materials and household contents.[3,4]

Inhalation of wildfire smoke induces oxidative stress, airway inflammation, and impaired mucociliary clearance.[1,5] In infants and children with BPD, whose lungs already demonstrate dysregulated inflammatory signaling, impaired epithelial repair, and reduced antioxidant capacity, these exposures may exacerbate epithelial injury, amplify small airway remodeling, and worsen pulmonary vascular dysfunction.[5] These mechanisms provide biologic plausibility for the heightened clinical vulnerability observed in this population during wildfire smoke events.[5,8]

Clinical and Epidemiologic Impacts on Children With BPD

Evidence linking wildfire smoke exposure to acute respiratory morbidity in children is well established, although data specific to BPD remains limited. Multiple U.S. and international studies have demonstrated associations between $PM_{2.5}$ exposure and increased pediatric emergency department visits and hospitalizations for asthma, wheezing, and lower respiratory tract disease.[2,3,8,11] Among infants and children with chronic lung disease, wildfire periods have been associated with increased oxygen requirements, higher frequency of oxygen desaturation events, and greater bronchodilator utilization.[8,11]

Figure 1. Conceptual framework illustrating pathways linking wildfire smoke (WFS) exposure to pulmonary injury and clinical deterioration in infants and children with bronchopulmonary dysplasia.[1,5,8]

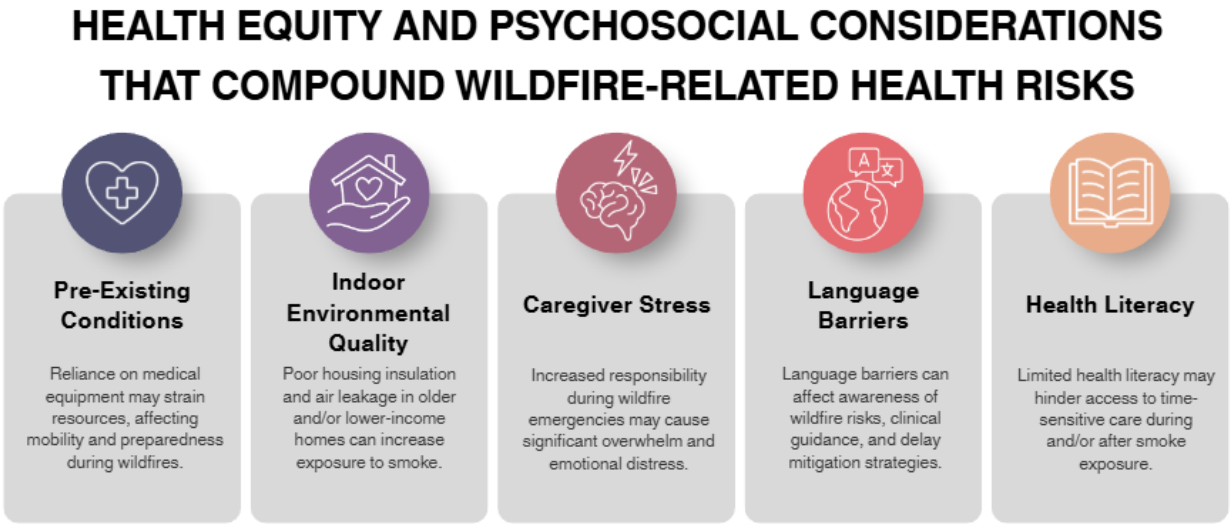


A population-based study from British Columbia reported a 1.6-fold increase in pediatric respiratory admissions during wildfire smoke days.[2] California-based analyses have similarly demonstrated marked increases in pediatric respiratory encounters during and following major wildfire events, including the Camp and Woolsey fires.[3,10,11] For children with BPD, these effects are likely magnified. Even short-term $PM_{2.5}$ exposure may destabilize baseline respiratory status, while repeated or cumulative exposures across wildfire seasons may contribute to accelerated lung function decline and increased risk of pulmonary hypertension, a common and serious comorbidity in severe BPD.[5,8]

Health Equity and Psychosocial Considerations

Socioeconomic and geographic disparities substantially compound wildfire-related health risks. Families of medically complex children, including those requiring home oxygen, noninvasive ventilation, or tracheostomy-dependent mechanical ventilation, often face limited resources for effective air filtration, relocation, or emergency preparedness.[9,12] Low-income, racially and ethnically minoritized communities are disproportionately represented in WUI regions, where wildfire smoke may contain additional toxicants from burned infrastructure, and/or leak into older housing units.[3,4,13] Caregiver stress, language barriers, and variable health literacy further impede timely action, mitigation, and access to care during prolonged smoke events.[8,11,14,15,16]

Figure 2. Illustration of health equity and psychosocial considerations that compound wildfire-related health risks.[3,4,8,9,12-16]



Mitigation and Clinical Recommendations

Pediatric healthcare providers play a critical role in anticipatory guidance and wildfire preparedness for children with BPD. Recommended strategies (**Table 1**) include integrating air quality education and smoke-specific counseling into routine BPD follow-up, ensuring updated emergency and equipment plans prior to wildfire season, facilitating access to high-efficiency particulate air (HEPA) filtration, optimizing controller medications, and coordinating closely with home health and respiratory therapy teams.[8,10] Telehealth-based monitoring and early intervention during smoke events may further reduce morbidity.[8]

At the systems level, healthcare institutions should adopt tiered wildfire response frameworks that include telemedicine triage protocols, surge planning for respiratory admissions, and partnerships with public health agencies to improve access to clean-air resources for high-risk families. [11]

Table 1. Recommended clinical mitigation strategies for infants and children with bronchopulmonary dysplasia during wildfire smoke events. [8,11]

Category	Mitigation Strategies
Environmental Controls	- Keep windows/doors closed during AQI >100. - Use indoor air recirculation mode (HVAC "recirculate" setting). - Use HEPA air purifiers in child's primary room and common living space. - Avoid indoor activities that worsen air quality (candles, vacuuming without HEPA, frying foods).
Exposure Reduction	- Avoid all outdoor activity during AQI >150. - For medically fragile older children who must travel, consider well-fitted pediatric N95 if age/fit appropriate. - Limit car trips; always use recirculate mode in vehicles.
Medical Management	- Ensure all controller medications (ICS, ICS/LABA, LTRA) are optimized before fire season. - Update BPD or chronic lung disease action plan with AQI-triggered steps. - Provide early refills for bronchodilators and steroids. - Reinforce adherence during smoke episodes.
Equipment Readiness	- Verify home oxygen supply, tubing, humidifier chambers, and ventilator filters prior to wildfire season. - Ensure availability of backup power sources for ventilator-dependent children. - Encourage families to keep a "go bag" with equipment and medications.
Monitoring	- Encourage daily symptom tracking and home oxygen saturation monitoring during wildfire alerts. - Provide clear thresholds for contacting the care team (e.g., SpO_2 < 92% despite baseline oxygen flow).
Telehealth & Care Access	- Offer telehealth check-ins during smoke events for early intervention. - Coordinate with home health services for urgent equipment or supply needs.
Family & Caregiver Education	- Educate caregivers on AQI interpretation (EPA AirNow, PurpleAir correction). - Share clear protective steps and emergency planning guidance. - Discuss risks of cumulative seasonal exposure and when to seek urgent care.
Community & Health System Supports	- Connect families to community "clean air shelters" during severe smoke days. - Coordinate with social work to address barriers to air purifiers/filters. - Encourage hospitals to develop wildfire-specific respiratory surge plans.

Future Directions and Research Gaps

Substantial knowledge gaps remain regarding the direct and cumulative effects of wildfire smoke exposure on infants and children with BPD. Research priorities include longitudinal cohort studies linking air quality metrics with clinical outcomes, mechanistic studies examining oxidative and vascular pathways in the developing lung, and intervention trials evaluating the effectiveness of filtration, telehealth, and community-based resilience strategies.[5,8,9] The 2025 Eaton and Palisades fires underscore the urgent need for real-time clinical surveillance and integrated satellite–electronic medical record analytic systems. Understanding and identifying specifics related to impacts on increased asthma statistics to respiratory impacts of debris exposure left behind and so many other components of WFS exposure have yet to be studied. Ongoing studies at CHLA and collaborating pediatric centers aim to characterize the burden of wildfire smoke exposure on children with BPD during these events.

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Figures and Tables

Figure 1. ***Conceptual framework*** illustrating pathways linking wildfire smoke (WFS) exposure to pulmonary injury and clinical deterioration in infants and children with bronchopulmonary dysplasia.[1,5,8]

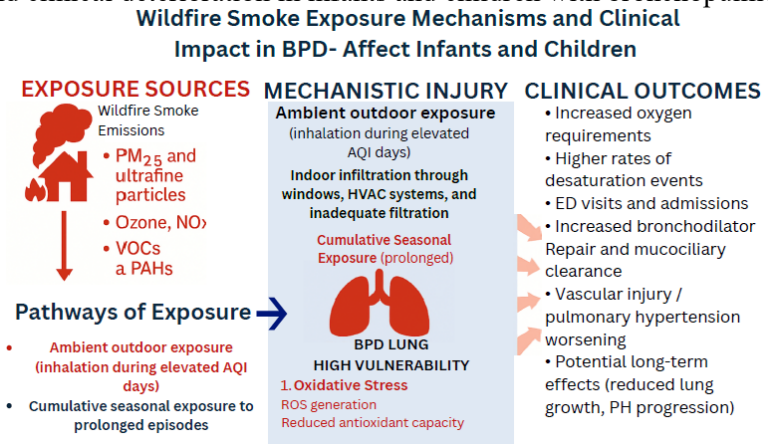

Figure 1. ***Multi-layered conceptual model*** of wildfire smoke–related clinical deterioration in infants and children with bronchopulmonary dysplasia (BPD).

From left to right; Exposure **Sources** depict wildfire smoke constituents, including **$PM_{2.5}$**, **ultrafine particles**, gaseous co-pollutants (**O_3, NO_x, CO**), and organic toxicants (**VOCs, PAHs**) with additional hazards from **burned structural materials in wildland–urban interface (WUI) zones** (metals and synthetic compounds).

Arrows indicate dispersion into **community outdoor air** and **indoor environments**.

In the **Pathways of Exposure** (center), infants encounter **ambient outdoor inhalation during elevated AQI days**, **indoor infiltration through windows, HVAC systems, and inadequate filtration**, **cumulative seasonal exposure** with prolonged/recurrent smoke episodes, and **caregiver vulnerabilities** that limit mitigation capacity, increasing exposure burden.

On the **Mechanistic Injury Pathways** (right), pollutants drive **oxidative stress** (ROS generation; reduced antioxidant capacity), **airway/alveolar inflammation** (IL-6, IL-8, TNF-α; neutrophil-mediated injury), **impaired epithelial repair and mucociliary clearance**, **vascular injury with worsening pulmonary hypertension** (endothelial dysfunction; increased pulmonary vascular resistance), and **small airway remodeling** (chronic inflammation; loss of pulmonary reserve). These converge on **"BPD Lung - High Vulnerability."**

The **Clinical Outcomes** (far right) include **increased oxygen requirements**, **higher rates of desaturation events**, **ED visits and admissions**, **greater bronchodilator and steroid use**, and **potential long-term effects** (reduced lung growth; **PH** progression).

Abbreviations: AQI, Air Quality Index; BPD, bronchopulmonary dysplasia; CO, carbon monoxide; ED, emergency department; HVAC, heating/ventilation/air conditioning; IL, interleukin; NO_x, nitrogen oxides; O_3, ozone; PAH(s), polycyclic aromatic hydrocarbon(s); PH, pulmonary hypertension; $PM_{2.5}$, particulate matter ≤2.5 µm; ROS, reactive oxygen species; VOC(s), volatile organic compound(s); WUI, wildland–urban interface.

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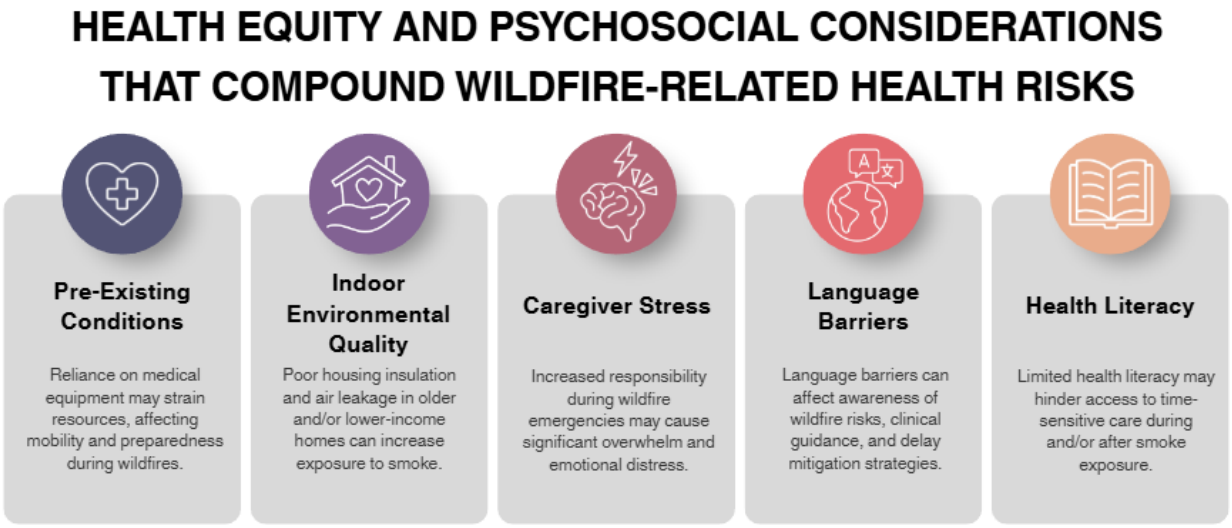

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