Impact of Wildfire Smoke on Acute Illness

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Healthcare professionals need to better understand the health impacts of a warming planet. The frequency and severity of extreme weather events (and their health impacts) are rising (*e.g.*, heatwaves, droughts, floods, storms). So, too, are the frequency and severity of wildfires. For example, Australia's "Black Summer" fires in 2019 and 2020 were estimated to be 30% more likely due to human-caused increases in global surface temperatures.[1,2] Globally, 61% of countries experienced increased wildfires during 2018 to 2021 compared to 2001 to 2004, causing an additional 9.17 million person-days of smoke exposure annually.[3] Despite this growing public health risk, data on its perioperative impacts are lacking, as are guidelines on the management of exposed patients.

Air pollution from combustion contains fine particulate matter (PM) that is typically composed of carbon, sulfur, and nitrogen compounds. Exposure to this PM causes more death and disability worldwide than alcohol use, high-sodium diets, or fasting hyperglycemia.[4] Consequently, the World Health Organization (Geneva, Switzerland) recommends that exposure to mean 24-h concentrations of PM with a diameter of 2.5 μm or less ($PM_{2.5}$) not exceed 25 $\mu g/m^3$. Wildfire smoke composition is influenced by the fire area, intensity, and materials burned. In addition to PM, it contains gases, including carbon dioxide, sulfur dioxide, nitric oxide, ozone, polycyclic aromatic hydrocarbons, and volatile organic compounds (*e.g.*, aldehydes and alkanes).[5–7] The latter elements may make wildfire smoke inhalation even more harmful than fossil fuel–derived PM. Further, polyaromatic hydrocarbon components may be carcinogenic in the long term.[8]

Wildfire smoke elicits oxidative stress and inflammatory responses,[9] and a significant mortality impact results from short-term increases in PM exposure.[10] Additionally, certain sociodemographic groups (*e.g.*, young, elderly, obese, and pregnant persons and those with preexisting cardiopulmonary disease or of low socioeconomic status) are at greater risk than others.[11,12] In the United States, approximately 52% of all 24-h mean $PM_{2.5}$ levels greater than 35 $\mu g/m^3$ result from wildfire smoke exposure.[13] Between 2008 and 2012, 10.3 million U.S. residents were exposed to more than 10 days of wildfire smoke–induced pollution above the World Health Organization's 24-h mean $PM_{2.5}$ threshold of 25 $\mu g/m^3$.[14] Communities close to wildfires have even documented hourly $PM_{2.5}$ concentrations as high as 6,106 $\mu g/m^3$ and daily concentrations of 394 $\mu g/m^3$.[15,16] Such wildfire smoke is known to spread over large geographic regions; even populations thousands of miles downwind of the fires have experienced health impacts.[5,6] Given the broad cardio-respiratory impacts of wildfire smoke, it is likely that a significant unmeasured impact on perioperative and critical care morbidity is being caused by smoke exposure. Unrecognized subclinical exposure to smoke from distant wildfires could play a role in the development of major adverse cardiac events or postoperative respiratory compromise. The increased obstetric risk affects mothers and is then conferred to newborns through prematurity and low birth weight, with long-lasting health consequences, including increased perioperative risk among those children. As population exposure to wildfire smoke increases, there is a need for development of perioperative protocols to guide the management of patients with overt or subclinical wildfire smoke exposure.

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Mechanisms of Health Impacts

Once inhaled, wildfire smoke PM directly stimulates airway and alveolar inflammation. Beyond these directly exposed tissues, wildfire smoke impacts organs through three primary mechanisms: autonomic dysregulation, a cascade of inflammation and oxidative stress (from the pulmonary vasculature), and direct translocation of PM into the bloodstream. Collectively, this exposure results in systemic inflammation, platelet activation, and endothelial dysfunction (fig. 1).

Autonomic Dysfunction and Hypertension

Inhalation of PM stimulates respiratory C-nerve fibers and rapidly adapting pulmonary receptors in the bronchial tree, resulting in vagal afferent signaling. This increase in parasympathetic tone is contrasted by a similarly associated reduction in carotid baroreceptors' sensitivity.[17,18] The overall result appears to be sympathetic stimulation, with increased circulating concentrations of noradrenaline, renin, angiotensin II, and aldosterone.[19] Adults exposed to either ambient wildfire or wood smoke in a laboratory setting experience significant and reproducible increases in blood pressure.[20] Even transient $PM_{2.5}$ exposure increases systolic, diastolic, and mean blood pressure for 12 h or more.[20,21] Long-term PM exposure is associated with a 41% increased risk of hypertension.[22] Additionally, heart rate variability is reduced among healthy middle-aged subjects in response to controlled exposure to both fine ($PM_{2.5}$) and ultrafine PM (0.1 μm, or $PM_{0.1}$).[23,24]

Inflammation and Oxidative Stress

Wood smoke exposure causes inflammation of the pulmonary and systemic vascular endothelium, lipid peroxidation, and systemic oxidative stress. Wildfire smoke (especially through its metallic components) induces oxidative stress through redox cycling mechanisms[25] and triggers a vast inflammatory response in the pulmonary circulation, which initiates a systemic inflammatory response resulting in end-organ damage (table 1).

Translocation into the Bloodstream

Fire-derived PM (especially ultrafine particulate matter, or $PM_{0.1}$) and gases cross the alveolar–capillary membrane to enter the bloodstream and reach the heart, peripheral vessels, and other organs, where they induce endothelial activation and platelet aggregation.[34] Metallic PM components stimulate platelet activation and aggregation.[35] Such effects are accompanied by a fibrinolytic response characterized by increased plasminogen activator inhibitor-1 and decreased tissue plasminogen activator. When combined with endothelial cell damage, a prothrombotic state is promoted. In a laboratory setting, NADPH oxidase inhibitors alleviated $PM_{2.5}$-induced endothelial

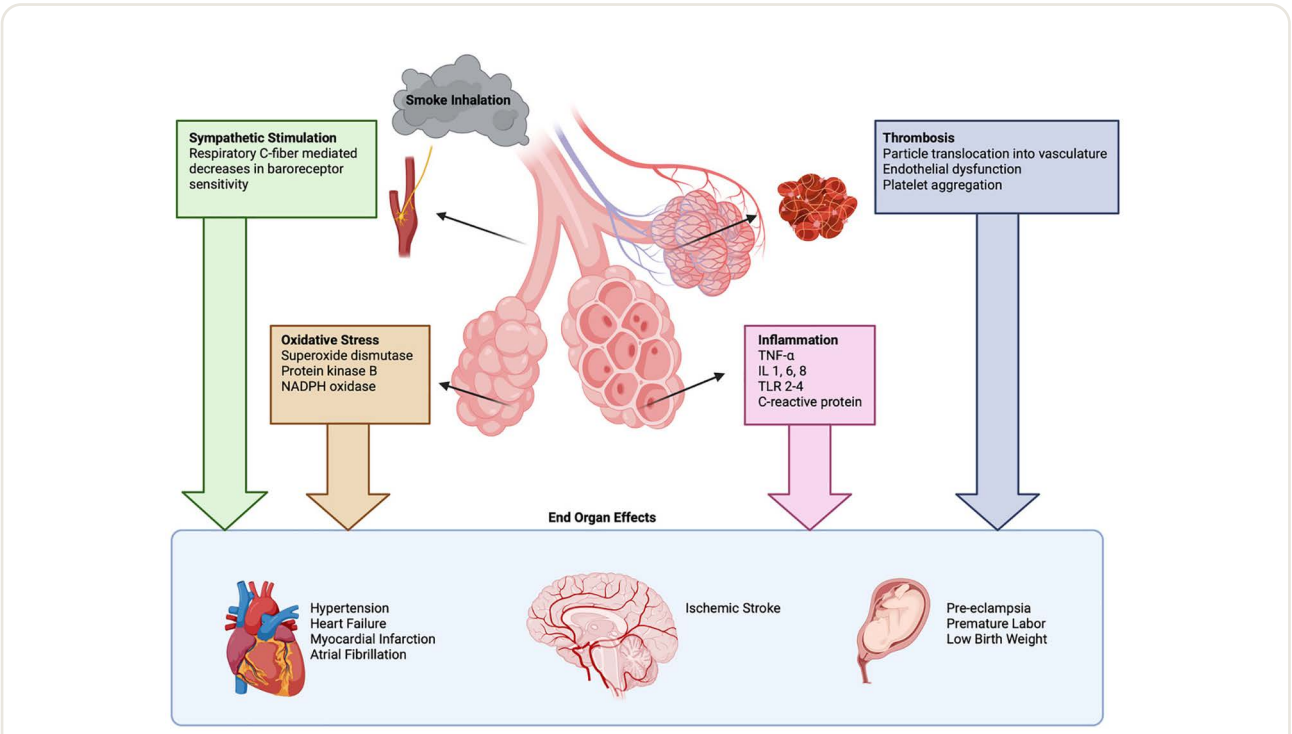

Fig. 1. The mechanisms by which wildfire smoke inhalation causes systemic health impacts. IL, interleukin; TNF-α, tumor necrosis factor-α; TLR, Toll-like receptor. Created with BioRender.com.

dysfunction.[27] Merely 3 h of exposure to wood smoke increases circulating concentrations of endothelial adhesion molecules, intracellular adhesion molecule-1, and vascular cell adhesion molecule-1 in healthy adults,[30] while its gaseous components induce a pro-atherosclerotic vascular response.[36]

Specific Disease Processes

Cardiovascular Disease

Myocardial Infarction. The impact of PM on cardiovascular disease is well established[37] (table 2). The combination of endothelial injury, platelet activation, and coagulation increases the risk of acute coronary syndromes. Similarly, elevations in sympathetic tone and systemic inflammation increase the risk of myocardial infarction. This association between the incidence of myocardial infarction and PM concentration has been described across age ranges and geographies.[53–55] Similarly, PM concentrations greater than 8 μg/m^3 resulting from forest fires increase cardiovascular mortality.[56]

Arrhythmias. Driven by circulating inflammatory cytokines, oxidative stress, and increased sympathetic tone, short-term increases in PM concentrations raise the risk of new-onset atrial fibrillation.[56,57] Over a longer time, PM-induced inflammatory fibrosis, progressive atrial remodeling, and increased pulmonary artery pressures lead to right ventricular dysfunction and right atrial stretch, causing intra-atrial conduction abnormalities.[58]

Heart Failure. Through high sympathetic tone, systemic inflammation, and endothelial injury, wildfire smoke may drive myocardial injury, resulting in decompensation of existing heart failure. Both pulmonary artery pressures and right ventricular afterload are increased by exposure to ambient PM, while increased arterial vasoconstriction elevates left ventricular afterload. When combined with PM-induced blunting of urinary sodium excretion, wildfire smoke markedly increases the demand on a failing heart and can precipitate decompensation.[59–62]

Stroke. Stroke is the second-leading cause of death and the third-leading cause of disability worldwide. PM-induced hypertension, atrial fibrillation, platelet aggregation, and oxidative stress increase stroke risk.[63] Ambient PM ranks fourth among 19 stroke risk factors summarized by the Global Burden of Diseases Study in 2019.[64]

Insulin Resistance. PM exposure is associated with insulin resistance and impaired pancreatic β-cell function, major risk factors for cardiac disease. With a 2-month lag, lower insulin sensitivity and higher fasting glucose concentrations were observed after high PM exposure in 1,000 Mexican-American patients.[65] In Eastern China, short-term PM

Table 1. Oxidative Stress and Inflammatory Response to Particulate Matter Exposure

Exposure	Cell Type or Population	Molecule	Biochemical Effect	Significance
Wood smoke[26]	Isolated pulmonary vascular endothelium	Superoxide dismutase 1 Heme oxygenase 1	Upregulation Upregulation	Antioxidant enzyme and heat shock protein that upregulate in response to high levels of oxidative stress
$PM_{2.5}$[27]	Human umbilical vein endothelial cells	Endothelin-1 NADPH oxidase Protein kinase B/ endothelial nitric oxide synthase pathway Inducible nitric oxide synthase Asymmetric dimethylarginine	Activation Upregulation Increase Increase Increase	Demonstrative of endothelial dysregulation; inducing processes of vasoconstriction, oxidative stress, thrombogenesis, and atherosclerosis
Wood smoke[28]	Human monocytes	Tumor necrosis factor-α Interleukin-1b Interleukin-8	Increase Increase Increase	Cytokines that upregulate inflammation through the innate immune response
PM_{10}[29]	Alveolar macrophages and airway epithelial cells	Toll-like receptors 2–4	Expression and activation	Activation leads to phagocytosis and immunomodulation in macrophages, and mast cell activation
Wood smoke[30]	Healthy volunteers	Intracellular adhesion molecule 1 L-selectin Interleukins 6 and 8	No change	Cellular adhesion molecules and cytokines involved in innate and adaptive immune responses
Occupational wood smoke[31]	Healthy firefighters	8-Isoprostane	Increase	Marker of lipid peroxidation in oxidative stress
Occupational wildfire smoke[32]	Healthy firefighters	Sputum granulocytes Circulating white blood cells Circulating band cells	Increase Increase Increase	Markers of acute inflammation
$PM_{2.5}$[33]	Healthy volunteers	Clara cell protein	Increase	Marker of lung epithelial injury

PM, particulate matter; $PM_{2.5}$, particulate matter with a diameter of 2.5 μm or less; PM_{10}, particulate matter with a diameter of 10 μm or less.

Table 2. Selected Studies Investigating Clinical Impacts of Wildfire Smoke and Particulate Matter Exposure

Disease Process	Region	Date	Population	Effect Size for $PM_{2.5}$ Exposure
Myocardial infarction[38]	International meta-analysis	2001–2018	2,250,473 patients	RR, 1.02 (95% CI, 1.01–1.03) per 10 μg/m^3
Atrial fibrillation[39]	Beijing, China	2013–2014	100 patients	RR, 1.038 (95% CI, 1.014–1.062) per 10 μg/m^3
Atrial fibrillation[40]	Yancheng, China	2015–2020	15,171 inpatients	RR, 1.028 (95% CI, 1.013–1.042) per 10 μg/m^3
Atrial fibrillation[41]	Seoul, South Korea	2007–2015	1137 emergency department visits	RR, 1.045 (95% CI, 1.002–1.089) per 10 μg/m^3
Cardiac arrhythmia[42]	South Korea	2002–2016	178,780 men	OR, 1.27 (95% CI, 1.15–1.40) per 10 μg/m^3
Heart failure[43]	International meta-analysis	1981–2005	1,520,099 hospital encounters	Attributable risk, 2.1% (admission) Attributable risk, 1.6% (mortality)
Ischemic stroke[44]	China	2013–2017	117,338,867 hospital admissions	0.20% per 10 μg/m^3
Transient ischemic attack[44]	China	2013–2017	117,338,867 hospital admissions	0.33% per 10 μg/m^3
Ischemic stroke[45]	Beijing, China	2014–2018	315,499 admissions	0.22% (95% CI, 0.12–0.33%) per 10 μg/m^3
Deep vein thrombosis[46]	USA	2000–2008	435,413 admissions	0.68% per 10 μg/m^3
Pulmonary embolism[46]	USA	2000–2008	435,413 admissions	0.59% per 10 μg/m^3
Asthma[47]	Colorado, USA	2011–2014	446,106 emergency department admission	OR, 1.081 (95% CI, 1.058–1.105) per 1 μg/m^3*
Combined respiratory disease[47]	Colorado, USA	2011–2014	446,106 emergency department admission	OR, 1.021 (95% CI, 1.012–1.031) per 1 μg/m^3*
Asthma[48]	Oregon, USA	5-month fire season in 2013	2,490 patients	OR, 1.089 (95% CI, 1.043–1.136) per 10 μg/m^3*
Asthma[49]	California, USA	2008	102,311 patients	RR, 1.07 (95% CI, 1.05–1.10) per 5 μg/m^3 (hospital admission) RR, 1.06 (95% CI, 1.05–1.07) per 5 μg/m^3 (ED presentation)
Asthma[50]	New South Wales, Australia	1994–2007	3,141,017 admissions	OR, 1.12 (95% CI, 1.05–1.19) when PM > 99th percentile
COPD[50]	New South Wales, Australia	1994–2007	3,141,017 admissions	OR, 1.13 (95% CI, 1.05–1.22) when PM > 99th percentile
Asthma[51]	California, USA	2003	40,856 admissions	OR, 1.34 (95% CI, 1.13–1.57) during smoke exposure
COPD mortality[52]	Washington state, USA	2006–2017	170,965 deaths	OR, 1.14 (95% CI, 1.02–1.26) when PM > 20.4
All-cause respiratory mortality (ages 45–64 yr)[52]	Washington state, USA	2006–2017	170,965 deaths	OR, 1.35 (95% CI, 1.09–1.67) when PM > 20.4

*Wildfire-specific.

COPD, chronic obstructive pulmonary disease; OR, odds ratio; PM, particulate matter; $PM_{2.5}$, particulate matter with a diameter of 2.5 μm or less; RR, relative risk.

exposure positively correlated with increased fasting blood glucose, especially in patients older than 65 yr.[66]

Respiratory Disease

Wildfire smoke causes lung inflammation, with alveolar macrophages and monocytes mounting a local immune response, resulting in direct tissue damage, impaired gas exchange, and airway irritation. Increases in both emergency department presentations and hospital admissions for asthma and chronic obstructive pulmonary disease result from exposure (table 2). Generally, finer particles are more toxic than coarser particles, and wildfire PM appears more toxic than urban PM.[67] Between 2013 and 2018 in Nevada, wildfire smoke resulted in a 6.1% higher incidence of asthma presentations compared to similar levels of PM originating from nonwildfire sources.[68] These effects are also modified by social factors. For example, North Carolina asthma emergency department visits stratified by socioeconomic status show that per 100 μg/m^3 of peat smoke $PM_{2.5}$ exposure, emergency department visits increased by 85% in richer counties but by 124% in poor counties.[69]

Obstetric and Neonatal Risk

Black carbon, a component of $PM_{2.5}$, translocates into the systemic circulation and deposits in placental tissue. A two-photon microscopy study assessing black carbon load in placental villous tissue demonstrated that the placental barrier is permeable to air pollution components, which would allow pollution to reach the fetal circulation.[70] There is concern that the same processes of inflammation, oxidative stress, and endothelial dysfunction may also affect the maternal–fetal unit. Evidence suggests a dose-dependent placental inflammatory response to wildfire smoke exposure.[71] Placental inflammation is strongly associated with preeclampsia and adverse birth outcomes, including preterm birth, low birth weight, and neonatal morbidity[72–74] (table 3). Likewise, oxidative stress–induced vascular and endothelial dysfunction are associated with preterm birth, hypertensive disorders of

pregnancy, preeclampsia, and restricted fetal growth.[87] In rabbit models, exposure to PM from fossil fuel combustion decreased placental blood flow and increased umbilical artery resistance.[88]

PM exposure may also impact endocrine signaling; in the second trimester, exposure among 222 mother–newborn pairs was associated with differences in the cord blood metabolites derived from the lipoxygenase pathways. These are key regulatory hormones for fetal metabolism and labor progression.[89] Premature birth negatively impacts health throughout a patient's life,[90] and wildfire smoke exposure is consistently associated with increased rates of preterm birth. A dose-dependent decrease in birth weight is observed with respect to proximity to wildfires and the severity of smoke exposure. *In vitro* experimentation also shows that organogenesis, as well as migration and differentiation of neural crest cells, are sensitive to oxidative stress.[91]

Pediatric Populations

Children are particularly vulnerable to PM pollution due to increased minute ventilation relative to body mass and reduced efficiency of nasopharyngeal particle deposition. This unique physiology results in a higher proportion of particles reaching lung tissue[92] (table 3). Since the mid-1990s, pediatric asthma presentations have been shown to increase during wildfire smoke exposure.[93] Furthermore, fire-derived ozone emissions account for more than 2,000 annual pediatric emergency department visits for asthma in the United States.[94]

Discussion

A host of negative cardiopulmonary, obstetric, and neonatal outcomes result from wildfire smoke exposure, driving a greater burden of comorbidities in the perioperative population. In addition, recent wildfire smoke exposure may directly increase perioperative risk: adverse respiratory events affected 37% of children with a past medical history of reactive airway disease during healthy air periods but affected 55% during periods with unhealthy air (1.50 [1.04 to 2.17]; $P = 0.032$).[95] This finding may well represent just the tip of the iceberg, with wildfire smoke exposure being responsible for a significant perioperative morbidity burden. Several key knowledge gaps are currently hampering our understanding of this perioperative risk factor.

Knowledge Gaps

Broader Perioperative Impacts

Data pertaining to perioperative risk from wildfires are sparse. The Californian data from Marsh *et al.*[95] were the first of their kind to bring this risk to light with a specific focus on pediatric respiratory complications. Given the paucity of perioperative data, some insight may be gained from tobacco literature. While the harm from vitamin E acetate exposure associated with electronic cigarette use is mechanistically different from PM exposure, perioperative guidelines on electronic cigarette smoke exposure may form a useful starting point. Guidelines recommend that patients are screened preoperatively for electronic cigarette smoke exposure and recommend focused investigations for health impacts when the exposure is suspected.[96] Such an approach could be taken in relation to wildfire smoke, screening patients for risk of exposure based on their location. Passive cigarette smoke exposure has been associated with increased acute respiratory distress syndrome development in septic patients.[97] Similarly, pediatric patients with chronic cigarette smoke exposure demonstrate increased rates of coughing, laryngospasm, and hypoxia when undergoing procedural sedation.[98] Linking wildfire smoke exposure data to perioperative outcomes datasets will likely elucidate this relationship. Linking critical care outcome registries or intensive care unit admissions datasets to smoke exposure data will be helpful to identify the proportion of patients whose critical illness is exacerbated by PM exposure.

Wildfire-specific Smoke

Many existing studies have determined exposure using total measured PM concentration, with some combining satellite images of smoke clouds or seasonal smoke patterns to attribute this measured PM to wildfire sources. Modern chemical transport models allow the impacts of wildfire PM to be studied in isolation.

Low-income Countries

Many of these existing data originate from relatively high-income countries. A substantial portion of global wildfire smoke exposure occurs in Sub-Saharan Africa and South Asia, where access to healthcare and healthcare data is limited. Data collected in low-resource settings would be highly valuable in producing better estimates of the true global burden of wildfire smoke impact.

Nonlinear Modeling of High Smoke Concentrations

Studies typically have included linear models reporting the odds ratio or relative risk of developing a clinical endpoint for a given change in PM concentration, typically 1 or 10 $\mu g/m^3$. PM concentrations can exceed 500 $\mu g/m^3$ in regions close to fires, and it is not known whether the exposure–response function is linear at these concentrations. Studies that explore the nonlinear effects of smoke exposure would allow for more complete estimates of current and future health impacts. Developing such an exposure response function will allow health systems to prepare for future impacts while providing vital data to guide policy decisions. Likewise, impacts of dynamic responses need to be studied (*i.e.*, might a sudden but small rise be more acutely harmful than a sustained higher concentration of toxicants?)

Table 3. Selected Studies Investigating Obstetric, Neonatal, and Pediatric Impacts of Wildfire Smoke, and Particulate Matter Exposure

Clinical Endpoint	Exposure	Region	Date	Population	Effect Size
Gestational hypertension[75]	Mean $PM_{2.5}$ across all three trimesters	Colorado, USA	2007–2015	535,895	OR, 1.204 (95% CI, 1.083–1.339) per 1 μg/m^3
Preeclampsia[76]	Mean $PM_{2.5}$ during weeks 10–14 of gestation	Southern Israel	2004–2016	133,197	HR, 1.04 (95% CI, 1.00–1.09) per 10 μg/m^3
Preeclampsia[76]	Mean $PM_{2.5}$ during first 25 weeks' gestation	Southern Israel	2004–2016	133,197	HR, 2.08 (95% CI, 1.10–3.94) per 10 μg/m^3
Premature birth (<37 weeks)[75]	Mean $PM_{2.5}$ during the second trimester	Colorado, USA	2007–2015	535,895	OR, 1.13 (95% CI, 1.09–1.18) per 1 μg/m^3
Premature birth (<37 weeks)[77]	Days of wildfire smoke during second trimester	California, USA	2006–2012	3,002,014	0.83% increase in risk per day
Premature birth (<37 weeks)[77]	Days of wildfire smoke during third trimester	California, USA	2006–2012	3,002,014	0.68% increase in risk per day
Premature birth (<37 weeks)[77]	Days of wildfire smoke during entire pregnancy	California, USA	2006–2012	3,002,014	0.49% increase in risk per day
Premature birth (<37 weeks)[78]	Smoke exposure from Camp Fire event	California, USA	2018	68,006	RR, 1.25 (95% CI, 1.22–1.28) per 1 μg/m^3
Premature birth (<37 weeks)[79]	Exposure to wildfire smoke wave during first trimester	Brazil	2001–2018	190,911	OR, 1.41 (95% CI, 1.31–1.51)
Premature birth (20–27 weeks)[80]	For 3 months following severe bushfires	Victoria, Australia	2009	73,831	50% increase
Premature birth (<37 weeks)[81]	$PM_{2.5}$ during entire pregnancy	Canada	2005–2012	818,400	OR, 1.04 (95% CI, 1.024–1.056) per interquartile range
Birth weight[75]	Mean $PM_{2.5}$ across all three trimesters	Colorado, USA	2007–2015	535,895	–5.7g per 1 ug/m^3 $PM_{2.5}$
Birth weight[82]	Exposure to smoke plume	Colorado, USA	2007–2013	135,676	3.8% lower
Birth weight <2,500 g[82]	Exposure to smoke plume	Colorado, USA	2007–2013	135,676	3.4% increased incidence
Birth weight <2,500 g[83]	Mean $PM_{2.5}$ during first trimester	Brazil	2001–2018	1,602,417	18.6% increased incidence
Birth weight <500 g[80]	For 3 months following severe bushfires	Victoria, Australia	2009	73,831	50% increased incidence
Congenital heart disease[84]	Mean $PM_{2.5}$ during the second month of pregnancy	Wuhan, China	2011–2013	105,988	OR, 1.10 (95% CI, 1.03–1.18) per 10 μg/m^3
Gastroschisis[85]	Second trimester wildfire smoke exposure	California, USA	2007–2010	844,384	RR, 1.28 (95% CI, 1.07–1.54)
Gastroschisis[85]	Third trimester wildfire smoke exposure	California, USA	2007–2010	844,384	RR, 2.17 (95% CI, 1.42–3.52)
Pediatric emergency department presentation for respiratory conditions[86]	Wildfire-exposed zip codes	San Diego, USA	2007	25,992	RR, 1.34 (95% CI, 1.18–1.52)
Pediatric emergency department presentation for asthma[86]	Wildfire-exposed zip codes	San Diego, USA	2007	25,992	RR, 2.12 (95% CI, 1.57–2.86)
Pediatric perioperative adverse respiratory events[95]	Elective surgery during periods of wildfire smoke exposure	California, USA	2018	625	RR, 1.5 (95% CI, 1.03–2.17)

HR, hazard ratio; OR, odds ratio; PM, particulate matter; $PM_{2.5}$, particulate matter with a diameter of 2.5 μm or less; RR, relative risk.

Perioperative Needs

Guidelines for Acute and Subclinical Exposure

Although a broad array of pathophysiologic mechanisms of smoke exposure has been described, it is challenging to identify a targeted intervention for exposed individuals, and thus there are no established treatments or practice guidelines. No randomized controlled trials have investigated treatment of PM or wildfire smoke exposure. Instead, care is typically supportive and defined by the presenting pathology. Emergency medicine guidelines do exist regarding smoke inhalation but are not specific to wildfires. These guidelines focus on supportive respiratory care with high-flow oxygen and bronchodilators as needed, testing for carbon monoxide toxicity, and administration of thromboprophylaxis. Perioperative wildfire smoke exposure protocols should consider the need for additional thromboprophylaxis, the need for antihypertensives in the acute setting, the role of additional hemodynamic monitoring among those with heart failure, the role of bronchodilators or other airway clearance interventions, and risk stratification for obstetric patients.

Procedure Timing to Mitigate Risk

Modern weather forecasting allows for accurate smoke plume spread prediction, which facilitates targeted air pollution warning systems. If research reveals increased perioperative complications after smoke exposure, then changes could be made in the scheduling of surgical cases in high-risk patients at times of high smoke exposure. Reactive airway disease or obstructive lung diseases typically present on lag days 0 to 2.[47] Hypertension develops within hours and can resolve after 12 h.[63] Downstream cardiac effects are variable in timing and peak between days 0 and 6 across different studies.[38] These risk peaks typically span 1 day, and in the few studies that employ longer time series, the effects were not significant during a 7-day window. While some patients present at lag day 0, within hours of smoke exposure, others will present several days into the exposure. As a period of PM exposure persists, the risk of health impacts increases. Beyond day 15, the exposure response curves for cardiovascular and respiratory admissions become nonlinear and appear to plateau.[99] During periods of heavy smoke, high hourly maximum PM concentration is highly predictive of elevated 24-h mean values.[100] As such, any evidence of high PM exposure should be treated with high suspicion for the presence of adverse effects suspected. Work is being undertaken by this research group to develop a nonlinear, time-dependent exposure–response function. It is envisaged that this work will better characterize the response at high PM levels. Detailed data on the time variance of the exposure–response function can form the basis for surgical scheduling around periods of wildfire smoke.

Conclusions

The health impact of wildfire smoke is just one symptom of a far broader systemic problem in the human ecosystem. Through changes in surface temperatures, rainfall patterns, wind speed, and relative humidity, the adverse effects of wildfires will increase as we continue warming the planet through the combustion of fossil fuels (*e.g.*, gasoline, natural gas, oil, coal, and diesel). As leaders in the perioperative space (the most carbon-intensive area of healthcare), anesthesiologists can lead the charge in rapidly reducing healthcare-related greenhouse gas emissions and those across society. Moreover, the healthcare industry has a global market more than three times the value of the fossil fuel industry. If leveraged effectively through coordinated action, the economic might of the healthcare community and academic institutions could reduce future damage to human health caused by the continued use of fossil fuels. As the window to limit global warming to 1.5° or 2°C closes, clinicians should act urgently and decisively to safeguard human health.

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Competing Interests

Dr. Montgomery is cochair of the Lancet Countdown on Health and Climate Change and is a member of other organizations that address this issue. Dr. Montgomery is cofounder of a nonprofit company to help decarbonize healthcare. Dr. Shindell is a consultant for the Institute for Governance and Sustainable Development. Dr. Levy has received funding from Merck, Octapharma, and Werfen unrelated to this work. The other authors declare no competing interests.

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