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Review

Respiratory Emergencies in Disasters: A Narrative Review

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ABSTRACT

Disasters cause a wide spectrum of respiratory injuries and affect hundreds of millions of people each year. Because the respiratory system is in continuous contact with the external environment, it is among the most vulnerable systems in disaster settings. Silica- and asbestos-containing dust released from earthquake debris, fine particulate matter from wildfire smoke, mold spores in flood-damaged interiors, ash and acidic gases from volcanic eruptions, toxic industrial chemicals released during chemical incidents, barotrauma from explosions, and biological or pandemic threats each trigger acute and chronic respiratory diseases through distinct mechanisms. This narrative review addresses respiratory emergencies associated with natural and human-made disasters, the underlying pathophysiological mechanisms, diagnostic and management approaches, vulnerable populations, and the influence of climate change on the rising burden of disaster-related respiratory disease. Evidence was synthesized from PubMed/MEDLINE, Web of Science, and Scopus, supplemented by official reports from the Emergency Events Database, the World Health Organization, and the United Nations Office for Disaster Risk Reduction. Available data indicate that acute respiratory infections are major causes of morbidity and mortality in disaster settings across all age groups. Furthermore, patients with chronic respiratory diseases face an increased risk of exacerbations, while the management of acute respiratory distress syndrome remains a significant challenge, particularly in resource-limited environments. Disaster preparedness planning must prioritize respiratory emergencies and strengthen response protocols for vulnerable groups.

KEYWORDS: Disasters, acute respiratory distress syndrome, earthquakes, wildfires, pandemics, climate change

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INTRODUCTION

Disasters are events that exceed a community's coping capacity, require external assistance, and produce severe consequences for human life and health systems.[1] According to the Emergency Events Database (EM-DAT) classification, disasters are grouped into natural disasters (including geophysical, hydrological, meteorological, climatological, and biological subtypes) and technological (human-made) disasters.[2] Each year, recorded disaster events affect millions of people, cause substantial mortality and morbidity, and impose a heavy burden on healthcare systems.[3]

Between 1995 and 2022, a mean of 398 natural disasters per year was recorded, with a cumulative death toll of 918,198.[4] In 2023 alone, disaster-related mortality reached 86,473, exceeding the two-decade average, largely driven by the February 6, 2023 Kahramanmaraş earthquake sequence in Türkiye and Syria, which claimed 56,683 lives, affected 18 million people, and caused USD 42.9 billion in economic losses.[5]

The respiratory system is uniquely exposed in disaster settings. The lungs maintain continuous contact with the environment and serve as the primary surface for inhaled particulates, toxic gases, and biological agents.[6] Silica- and asbestos-containing dust from collapsed structures, fine particulate matter (PM2.5) in wildfire smoke, mold spores proliferating in flood-

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damaged interiors, irritant gases released in chemical incidents, and pressure waves generated by explosions cause upper and lower airway injuries, acute bronchospasm, pneumonia, and acute respiratory distress syndrome (ARDS) through distinct mechanisms.[7] A systematic review of 47 studies on earthquake-induced tsunamis demonstrated that crowded evacuation centers increase the incidence of acute respiratory infections, which rank among the most frequently reported post-disaster illnesses.[8]

The Sendai Framework for Disaster Risk Reduction 2015–2030, endorsed by 187 UN member states, explicitly identifies protection of health infrastructure and continuity of health services as integral to disaster preparedness.[9] However, disaster protocols specific to respiratory diseases remain underdeveloped. This review addresses respiratory emergencies associated with natural and human-made disasters, the underlying pathophysiological mechanisms, diagnostic and management approaches, vulnerable populations, and the influence of climate change.

METHODS-LITERATURE SEARCH STRATEGY

This narrative review draws on a comprehensive literature search of PubMed/MEDLINE, Web of Science, and Scopus, completed in March 2026. Search terms combined respiratory- and disaster-related vocabulary using Boolean operators, including respiratory emergencies, inhalation injury, ARDS, chronic obstructive pulmonary disease (COPD) exacerbation, asthma exacerbation, mechanical ventilation, mass casualty, earthquake, wildfire, flood, hurricane, volcanic eruption, tsunami, chemical disaster, blast lung, pandemic, displaced populations, and climate change. The review focused on publications from 2015-2026, with selected seminal references retained from earlier periods. Official reports from EM-DAT, the World Health Organization, and the United Nations Office for Disaster Risk Reduction were consulted as primary sources for disaster epidemiology.

Main Points

- The respiratory system is a primary target across virtually all disaster types, with mechanisms ranging from particulate inhalation and toxic gas exposure to barotrauma and viral aerosol transmission.
- Crowded evacuation centers and disrupted health infrastructure substantially increase the burden of acute respiratory infections and tuberculosis after natural disasters.
- Wildfire-derived fine particulate matter (PM2.5) appears to be more toxic to the respiratory system than urban PM2.5 of equivalent concentration.
- Point-of-care ultrasound, pulse oximetry, and capnography are pragmatic, field-deployable tools that should be embedded in disaster respiratory triage protocols.
- Children, the elderly, pregnant women, patients with chronic respiratory disease, and displaced populations bear a disproportionate share of disaster-related respiratory burden.

CLASSIFICATION OF DISASTERS AND GENERAL RESPIRATORY EFFECTS

The EM-DAT database contains more than 27,000 recorded disaster events, the majority of which are of natural origin, with floods and storms appearing most frequently.[2] Biological disasters—including epidemics and pandemics—are classified within the natural disaster category in EM-DAT.

Each disaster type affects the respiratory system through distinct mechanisms, making disaster-specific clinical planning necessary. Earthquakes generate acute inhalation injury through dust containing silica and asbestos released during structural collapse. Wildfires produce complex, PM2.5-dominated smoke that directly damages the airway epithelium. Floods and hurricanes create conditions for biological contamination, particularly mold, which triggers exacerbations of asthma and COPD. Industrial chemical disasters release highly toxic gases, such as chlorine, ammonia, and phosgene, causing mass inhalation injuries.[7]

The post-disaster respiratory burden is substantial. The systematic review of 47 earthquake-induced tsunami studies cited above identified acute respiratory infections as the most frequently reported post-disaster illness group, with crowded evacuation centers, exposure to high pathogen loads, adverse climate, and inadequate vaccination coverage as the principal risk factors.[8] Recent evaluations following the Kahramanmaraş earthquake have demonstrated that dust clouds rising from collapsed buildings can spread across wide geographic areas within hours, with silica and asbestos content posing both acute inhalation hazards and long-term pneumoconiosis risk.[10,11] A contemporary review of natural disasters and respiratory health similarly highlights that heatwaves, wildfires, hurricanes, floods, earthquakes, and volcanic eruptions each gives rise to distinct but serious respiratory complications.[7]

RESPIRATORY EMERGENCIES BY DISASTER TYPE

Earthquakes

Earthquakes cause abrupt and widespread damage to the built environment and rapidly generate large volumes of airborne particulate matter. The respiratory consequences span a wide clinical spectrum, ranging from the acute phase to long-term sequelae, and include inhalation of dust and particulates, acute respiratory infections, exacerbation of pre-existing respiratory disease, thoracic trauma, and pulmonary thromboembolism.[12]

Dust released from collapsed structures may contain high concentrations of silica, asbestos, and other toxic constituents. Measurements performed in Hatay after the February 6, 2023, Kahramanmaraş earthquakes—conducted during the active demolition phase and representing peak rather than time-weighted average exposures—recorded mean respirable and total dust concentrations of 30.84 mg/m^3 and 33.66 mg/m^3, respectively, far exceeding international occupational exposure limits. Scanning electron microscopy (SEM) of debris samples identified fibrous particles consistent with the morphology of asbestos; however, definitive mineralogical confirmation requires further analytical methods beyond SEM.[11] Given Türkiye's well-documented pre-existing environmental and

occupational asbestos burden, these findings warrant serious attention to the long-term risk of mesothelioma and lung cancer among both survivors and rescue workers.[13]

Beyond particulate exposure, deteriorating shelter conditions, compromised sanitation, and stress-related immunosuppression substantially increase the post-earthquake burden of respiratory infections. A retrospective study of 1,122 patients after the Kahramanmaraş earthquake found significant increases in hospitalization for pneumonia and COPD exacerbations,[14] while a systematic review following the 2011 Great East Japan earthquake identified pneumonia as the most frequent post-disaster respiratory disease.[15] A multiplex reverse transcription quantitative polymerase chain reaction study from Gaziantep tent cities (n = 830) identified rhinovirus (20%), *Streptococcus pneumoniae* (12.9%), respiratory syncytial virus A/B (12.1%), and *Haemophilus influenzae* type b (10.9%) as the dominant respiratory pathogens, providing pathogen-level data from the Kahramanmaraş aftermath.[16] A multi-center observational study of adults admitted to intensive care after the same earthquake further documented the burden of severe respiratory infection requiring critical care support in the affected region.[17]

Güleç Balbay et al.[12] comprehensively reviewed the respiratory effects of earthquakes, identifying inhalation injury, respiratory infections, exacerbation of pre-existing disease, thoracic trauma, and pulmonary and venous thromboembolism as the principal complications, with the quality of building infrastructure emerging as a key determinant of both injury severity and infection risk.

Thoracic trauma is a frequently overlooked complication that is associated with mortality. Imaging in survivors trapped under rubble after Kahramanmaraş most commonly identified thoracic injuries—rib fractures, pulmonary contusion, pneumothorax, hemothorax, and pulmonary thromboembolism.[18] The earthquake-specific injury pattern differs from non-earthquake trauma. Prolonged entrapment precipitates crush syndrome, characterized by hypovolemia, hyperkalemia, and myoglobinuria; reperfusion injury after rescue can, in turn, progress to ARDS. Long-bone fractures increase the risk of fat embolism. Crush-related immobility and hypercoagulability further elevate the risk of pulmonary thromboembolism, which may evade detection during the acute phase.[12,18] Taken together, these findings underline the need for systematic evaluation of thoracic trauma, pulmonary contusion, and thromboembolic complications alongside inhalation injury in earthquake medicine.

Wildfires

Wildfires have become more frequent, severe, and prolonged over the past decade, under the influence of climate change. Wildfire smoke is a complex mixture of chemicals, including PM2.5, carbon monoxide (CO), ozone, nitrogen oxides, benzene, and various volatile organic compounds.[19] Wildfire-specific PM2.5 elicits stronger oxidative and inflammatory responses than urban PM2.5 at equivalent concentrations, reflecting a distinct toxic profile.[20]

Mechanistically, wildfire PM2.5 inhalation causes lung injury by inducing oxidative stress, systemic inflammation, disruption of airway epithelial integrity, and increased susceptibility to infection. Sustained exposure increases the risk of developing COPD, accelerates disease progression, and is associated with lung cancer and increased mortality.[19,20]

A study of more than six million emergency department visits demonstrated that each 1 $\mu g/m^3$ increase in wildfire smoke PM2.5 was associated with a 1.6% rise in asthma-related visits, 0.4% in COPD, and 0.4% in bronchitis, with effects markedly stronger than those of urban PM2.5.[21] A systematic meta-analysis of wildfire smoke exposure reported a 7% increase in overall health risk, a 9% increase in emergency department visits, and a 4% increase in hospitalizations, with respiratory disease and COPD risks rising by 7% and asthma by 11%.[22] In pediatric populations, wildfire smoke exposure has been associated with substantially increased asthma-related emergency department visits, and fine particles in California wildfire smoke have been linked specifically to pediatric respiratory hospitalizations.[23] In older adults with COPD, increased hospitalization and mortality have been consistently reported.[20]

The January 2025 Los Angeles (LA) wildfires offered a recent and stark illustration of the urban-interface respiratory threat. Within the most affected census tracts, daily mean PM2.5 reached 101.7 $\mu g/m^3$ at the downtown LA regulatory monitor, with concomitant elevations in nitrogen dioxide (NO_2) concentrations. Preliminary analyses of healthcare utilization documented increases of more than 35% in cardiovascular and respiratory telehealth visits among exposed populations.[24]

Wildfires also affect first-response personnel. A systematic review of 26 studies in firefighters and other responders found adverse respiratory effects in 24 studies, including decreased lung function, airway dysfunction, and increased respiratory symptoms.[25] Occupational wildfire smoke exposure has been shown to adversely affect lung function, reinforcing the need for respiratory protection as a standard component of wildfire response.

Floods and Hurricanes

Floods and hurricanes are among the most frequent and widely impactful natural disasters globally. Their respiratory health consequences arise through three principal mechanisms: aspiration injury, deterioration of indoor air quality due to mold and biological contamination, and an elevated risk of respiratory infection in crowded evacuation conditions.[7]

Drowning and near-drowning events produce the most acute respiratory complications. A cohort of 144 near-drowning patients reported acute respiratory failure on presentation in 54%, although early bacterial aspiration pneumonia did not independently determine mortality and was generally manageable with standard supportive care.[26] The pathophysiology of aspiration differs by water type: freshwater dilutes and inactivates pulmonary surfactant, whereas hyperosmolar seawater induces fluid shift into the alveolar space and disrupts the alveolar-capillary barrier, with both ultimately producing surfactant dysfunction and impaired gas exchange.[27,28] Aspiration of contaminated water further predisposes individuals to polymicrobial infection. A systematic meta-analysis identified Gram-negative bacteria as

the principal pathogens, with *Aeromonas* species, *Haemophilus influenzae*, and *Pseudomonas aeruginosa* among the most frequently isolated organisms; severe cases may progress to ARDS, requiring intensive care support.[29]

Rapid mold growth in flood-damaged interiors poses an additional acute and long-term respiratory hazard. The Houston-3H study, conducted after Hurricane Harvey in 2017, documented a significant increase in upper-airway allergic symptoms among exposed individuals and found strong associations between exposure to contaminated water and mold and the occurrence of multiple allergic manifestations.[30] Measurements in New Orleans after Hurricanes Katrina and Rita found markedly higher airborne mold concentrations in severely water-damaged homes than in lightly damaged dwellings, with endotoxin and fungal glucan levels comparable to those in agricultural settings.[31] A large cohort study of more than 11 million adults aged ≥65 years documented post-flood increases of 4.6% in all-cause emergency department visits, 6.9% in hospitalizations, and 8.3% in respiratory hospitalizations specifically.[32]

In addition, power outages following hurricanes pose an underrecognized threat to patients receiving home oxygen or domiciliary ventilation. Sandy (2012) and Maria (2017) provided clinical exemplars, and population-level data confirm significantly increased hospitalization risk in oxygen-dependent COPD patients during outages.[33]

Crowded evacuation centers and temporary shelters create conditions favorable for outbreaks of respiratory infection in the aftermath of floods and hurricanes. Influenza, tuberculosis (TB), and other droplet-transmitted pathogens spread more readily in densely populated indoor settings, with concurrent malnutrition, sleep deprivation, and chronic stress further weakening immune responses.[7,8] The combination of deteriorating shelter conditions, sanitation breakdown, and reduced access to health services amplifies the burden. These findings highlight the need to place respiratory health at the center of flood and hurricane response plans and to pre-configure infection control protocols in evacuation centers.

Volcanic Eruptions

Volcanic eruptions simultaneously release multiple respiratory irritants-ash particles, sulfur dioxide (SO_2), hydrogen sulfide (H_2S), hydrogen chloride (HCl), and CO-with respiratory effects varying by particle size, crystalline silica content, and gas concentration.[34] Volcanic ash induces airway injury through oxidative stress, inflammatory cytokine release, and impaired mucociliary clearance; the presence of cristobalite increases the risk of silicosis.[35]

Epidemiological evidence consistently associates acute exposure to volcanic emissions with increased respiratory morbidity. A recent scoping review of the global burden of volcanic exposure identified cough, sputum production, dyspnea, chest tightness, and wheeze as the principal symptoms, with significant increases in emergency department visits for asthma and COPD exacerbations. Chronic exposure has been linked to silicosis, pneumoconiosis, and lung cancer; respirable volcanic particles may cross the alveolocapillary barrier and contribute to systemic cardiovascular and cerebrovascular disease.[34]

The 2014–2015 Holuhraun eruption in Iceland provided a detailed picture of mass exposure to SO_2. In Reykjavik, approximately 250 km from the eruption site, a registry study showed significant increases in daily asthma medication use after the eruption began and significant rises in primary care and emergency utilization on high-SO_2 days.[36] Volcanic smog from Kilauea adversely affected respiratory symptoms and lung function in nearby Hawaiian populations, with high-exposure areas showing marked increases in cough prevalence.[37] The 2021 Cumbre Vieja (Tajogaite) eruption on La Palma, the first major eruption in a densely populated European setting in decades, has provided the most contemporary dataset: the Icelandic Volcanoes and Respiratory Health cohort study of 1,002 participants documented respiratory symptom patterns and pulmonary function changes during and after eruptive activity, establishing a framework for short-, medium-, and long-term respiratory follow-up.[38] These data demonstrate that the respiratory effects of volcanic emissions extend far beyond the immediate eruption zone.

Taken together, the evidence supports the use of appropriate respiratory protective equipment by responders, the systematic pre-planning of early evacuation for at-risk populations, close clinical follow-up of patients with chronic respiratory disease, and the establishment of long-term surveillance programs that capture both acute and chronic exposure scenarios.

Chemical Disasters

Chemical disasters involve the uncontrolled release of toxic substances through industrial or transport accidents, or by deliberate dissemination. Among toxic industrial chemicals (TICs), chlorine, ammonia, phosgene, and H_2S are the principal respiratory threats. These substances affect different anatomic regions depending on their water solubility: highly soluble agents—ammonia and HCl—rapidly damage the upper airways; chlorine, with intermediate solubility, affects both upper and lower airways; phosgene and nitrogen oxides, with low solubility, reach the lower airways and alveoli and produce severe pulmonary edema hours after exposure.[39]

Chlorine gas is among the most commonly encountered TICs in industrial accidents. Its intermediate solubility causes conjunctival irritation, laryngospasm, bronchospasm, and pulmonary edema.[39] Phosgene accounted for more than 80% of all chemical casualties in World War I and remains widely produced today (approximately 12 million metric tons annually) for use in the manufacture of plastics, pesticides, and pharmaceuticals. Its hallmark feature—a prolonged, asymptomatic latency followed by refractory pulmonary edema and ARDS—poses substantial diagnostic challenges in mass-casualty scenarios.[40]

The August 4, 2020 Beirut Port explosion dramatically illustrated the respiratory impact of industrial chemical disasters. The detonation of approximately 2,750 tons of ammonium nitrate produced one of the largest non-nuclear explosions of the modern era, injuring more than 6,000 people and killing 220 people.[41] Inhalation of nitric oxide and NO_2 gases caused

ARDS, diffuse distal airway inflammation, pulmonary edema, and methemoglobinemia.[41,42] Severe exposure produced fibrous bronchiolar obstruction lasting for weeks and reactive airway dysfunction syndrome.[41]

Medical response to chemical disasters rests on early airway management, lung-protective ventilation strategies, active airway suctioning, and respiratory physiotherapy. Identification of the agent is critical for both treatment and decontamination, but is often dependent on clinical presentation and exposure circumstances. Critical gaps in chemical disaster preparedness include rapid overflow of triage capacity, secondary exposure risk to healthcare workers, and the potential for systemic biochemical injury to develop before clinical findings emerge. Chemical disaster response, therefore, requires agent-specific antidote protocols, respiratory protection standards for first responders, and realistic mass-exposure exercises performed in concert.[43]

Blast Injuries and Blast Lung

Blast injuries arise in military, terrorist, and industrial settings and result from a distinct injury mechanism. Explosions injure the respiratory system through four mechanisms: (1) primary blast injury from direct effects of the supersonic pressure wave; (2) secondary injury from penetrating debris; (3) tertiary injury from victim displacement; and (4) quaternary injury from burns and toxic smoke inhalation.[44]

Primary blast lung injury (PBLI) occurs because the gas-filled lung is the organ most susceptible to damage from pressure waves. The supersonic pressure wave disrupts the alveolar–capillary membrane, producing interstitial and alveolar hemorrhage, pulmonary contusion, pneumothorax, pneumomediastinum, and arterial gas embolism. The incidence of blast lung ranges from 6–11% in open-air military explosions to over 90% in enclosed-space terrorist attacks, reflecting amplification of the pressure wave.[44,45] A key feature of PBLI is the relative paucity of external findings in the early hours despite the potential for rapid progression to ARDS over subsequent hours; most cases require mechanical ventilation and intensive care.

A multi-center study of mass casualty blast events identified blast lung as the strongest independent predictor of severe injury, with severe injury 18.8 times more likely in cases with documented blast lung.[46] No specific treatment protocol exists for PBLI; management is largely adapted from ARDS protocols, with lung-protective ventilation as the cornerstone.[45] A comprehensive review of combat-related ARDS literature emphasized that most evidence derives from retrospective data, that standardized blast-specific management protocols are still under development, and that systematic data on long-term pulmonary function in survivors remain insufficient.[47]

Biological Disasters and Pandemics

Biological disasters include epidemics and pandemics caused by intentional or accidental mass dissemination of pathogenic microorganisms. The severe acute respiratory syndrome (SARS) (2003), H1N1 (2009), Middle East respiratory syndrome (MERS) (2012), and coronavirus disease-2019 (COVID-19) (2019) outbreaks in the twenty-first century have demonstrated the devastating impact of biological threats on modern health systems, with the respiratory system as the primary target and acute respiratory failure and ARDS as the leading causes of death.[48,49]

The COVID-19 pandemic exposed the respiratory burden of biological disasters at an unprecedented scale. With more than 770 million confirmed cases globally, a substantial proportion of patients admitted to intensive care have developed acute hypoxemic respiratory failure requiring invasive mechanical ventilation. COVID-19 ARDS exhibited distinct pulmonary vascular dysregulation, with gas exchange impairment not consistently correlating with respiratory system compliance—a finding that revealed a pathophysiological profile separate from classical ARDS.[50]

Health system strain during the pandemic peaked with respect to intensive care capacity and ventilator supply. In a study of 625 U.S. hospitals, 63% issued at least one capacity alert during the pandemic; among those, 63% reported emergency department crowding, 61% reported intensive care unit (ICU) capacity overflow, and 12% reported ventilator shortages.[51] These data underscore the need to position ventilators as strategic preparedness resources and to address capacity inequities across countries.[51,52]

The long-term respiratory effects of COVID-19 extend the burden of the disaster well beyond the acute phase. A systematic review and meta-analysis of 3,066 discharged patients found residual abnormalities on chest computed tomography (CT) in approximately half of patients, with ground-glass opacity (44.1%) as the most common finding, and impaired diffusing capacity of the lungs for carbon monoxide observed in 34.8%. Post-COVID pulmonary fibrosis has been recognized as a distinct long-term phenotype requiring structured follow-up.[53]

The cumulative lessons of SARS, MERS, and COVID-19 define the core components of biological disaster preparedness: early warning and transparent reporting; rapid expansion of ICU capacity; stockpiling of respiratory protective equipment; and infection-control protocols for healthcare workers. Countries with prior outbreak experience were reported to have implemented faster containment measures during the COVID-19 pandemic.[48,54,55]

Displaced Populations

Displaced populations include those forced into temporary shelters and refugee camps following earthquakes, floods, conflict, or climate-related disasters. In addition to disaster-related respiratory risks, these groups face distinct environmental hazards: crowded and poorly ventilated indoor spaces, biomass fuel smoke exposure, malnutrition, and limited access to health services.[56,57] The burden of respiratory infectious diseases is markedly elevated in refugee populations, with upper respiratory tract infections among the most frequently reported complaints.[58]

Türkiye, which hosts the world's largest refugee population, provides one of the most informative settings for understanding

the respiratory burden among displaced populations. A national review of infectious disease patterns among more than 3.5 million Syrian refugees in Türkiye reported that 1,299,209 cases of respiratory tract infection and 108 active TB cases were identified and treated in temporary shelters between 2012 and 2016.[59] More recently, a multi-center retrospective analysis of TB patterns across Türkiye demonstrated higher rates of cavitary TB (50.9% vs. 28.9%; $P = 0.002$) and greater immunosuppression among migrant populations compared with non-migrants, although overall drug resistance rates remained similar.[60] A recent study from northwestern Ethiopia documented a TB prevalence of 7.6% in internally displaced populations, with prolonged camp residence and limited healthcare access as principal risk factors.[61] Evidence from armed conflict settings further supports this pattern, with conflict-associated rises in TB notifications, reductions in treatment success, and increased risk of TB spread to non-conflict regions through mass displacement.[62]

Biomass fuel smoke exposure is a major environmental contributor to chronic respiratory disease in displaced populations. Combustion of wood, dung, and crop residues for heating and cooking generates indoor air concentrations of PM2.5, CO, and polycyclic aromatic hydrocarbons. Chronic exposure to biomass smoke has been associated with more than a 2.5-fold increase in COPD risk.[63,64] Biomass exposure has been linked to acute lower respiratory tract infections and asthma-like symptoms in children and to chronic bronchitis and COPD in adults.[65] A systematic review of interventions to reduce respiratory infection burden in refugees and migrants found that arrival vaccination is effective in reducing secondary infections and outbreaks, although controlled evidence remains limited.[57]

Comprehensive care for displaced populations, therefore, requires reframing shelter design and ventilation standards through a respiratory health lens, integrating routine TB screening into disaster health services, reducing biomass smoke exposure, and implementing early post-arrival vaccination programs. The respiratory burden of displaced populations remains insufficiently addressed in disaster medicine.

The respiratory emergencies associated with each disaster type, principal exposures, and clinical syndromes are summarized in Table 1.

DIAGNOSIS AND MANAGEMENT

Diagnosis and management of respiratory emergencies in disaster settings face distinct challenges: resource constraints, high patient volumes during mass-casualty incidents, and inadequate medical infrastructure. Meeting these challenges requires pre-positioned diagnostic tools and clinical decision protocols tailored to the field.

Point-of-care ultrasound (POCUS) has become an increasingly valuable bedside tool. POCUS detects pneumothorax, pleural effusion, pulmonary consolidation, and pulmonary edema with high diagnostic accuracy and significantly shortens the time to diagnosis for patients with dyspnea compared with conventional radiography.[66] Pre-hospital lung ultrasonography performed by paramedics has demonstrated technical feasibility and moderate-to-high agreement with expert interpretation.[67]

Peripheral oxygen saturation (SpO_2) remains the primary monitoring tool in disaster settings owing to its low cost, non-invasive nature, and ease of use. A pre-hospital SpO_2 ≤90% supports triage to hospital admission and independently predicts in-hospital mortality and length of stay.[68] In CO poisoning, however, standard pulse oximetry yields misleading values; non-invasive pulse CO-oximetry that measures carboxyhemoglobin can serve as a first-line screen, but its sensitivity is approximately 65%, and arterial blood gas analysis

Table 1. Respiratory emergencies by disaster type: principal exposures, clinical syndromes, and pathophysiological mechanisms

Disaster type	Principal respiratory exposure	Principal clinical syndrome	Pathophysiological mechanism
Earthquake	Silica, asbestos, structural dust, debris smoke	Acute inhalation injury, pneumonia, COPD/asthma exacerbation, pulmonary thromboembolism	Particulate inhalation, mucosal injury, inflammation, hypercoagulability
Wildfires	PM2.5, CO, ozone, volatile organic compounds	Asthma/COPD exacerbation, ARDS, CO poisoning	Oxidative stress, bronchospasm, alveolar injury
Floods and hurricanes	Mold spores, aspiration, contaminated water	Respiratory tract infection, asthma exacerbation, aspiration pneumonia	Biological contamination, allergen amplification, surfactant disruption
Volcanic eruptions	Ash particles, SO_2, H_2S, silica	Acute bronchitis, reactive airways disease, pneumonia	Irritant gas exposure, reactive airway response
Chemical disasters	Chlorine, ammonia, phosgene, hydrogen sulfide	Toxic inhalation injury, pulmonary edema, ARDS	Direct mucosal injury, alveolar injury
Blast/terrorism	Pressure wave, combustion gases, secondary particulates	Blast lung, pulmonary contusion, pneumothorax	Barotrauma, alveolar rupture, gas embolism
Biological disasters and pandemics	Pathogenic microorganisms, aerosols	Viral pneumonia, ARDS, respiratory failure	Viral infection, cytokine storm, immune dysregulation
Displaced populations	Inadequate ventilation, crowding, biomass fuel smoke	ARI, influenza, tuberculosis, pneumonia outbreaks	Droplet/aerosol transmission, malnutrition

Biological disasters are categorized as natural disasters in the EM-DAT classification

EM-DAT: Emergency Events Database, ARI: acute respiratory infection, ARDS: acute respiratory distress syndrome, CO: carbon monoxide, COPD: chronic obstructive pulmonary disease, H_2S: hydrogen sulfide, PM2.5: fine particulate matter, SO_2: sulfur dioxide.

remains necessary for confirmation.[69] When intubation is required, end-tidal CO_2 monitoring is the gold standard for tube placement verification and ventilation management; pre-hospital capnography has been shown to predict mortality and transfusion requirements, guiding triage.[70]

Portable chest radiography enables on-site imaging where fixed radiology infrastructure is unavailable and was shown during the pandemic to be a reliable alternative for the detection of pneumothorax, consolidation, and pleural effusion in critically ill patients.[71] Arterial blood gas analysis supports the diagnosis of ARDS through calculation of PaO_2/FiO_2 ratio and assessment of acid–base status; the Berlin 2012 definition remains the standard, with the 2024 American Thoracic Society/ESICM update broadening the framework to include high-flow oxygen therapy.[72] Laboratory infrastructure requirements limit applicability in resource-constrained settings. Clinical scoring systems partially address this gap: NEWS2 has shown high prognostic value (area under the curve 0.89) in prehospital mass-casualty triage, while CRB-65—which does not require laboratory parameters—remains a pragmatic option for pneumonia severity assessment in resource-limited settings.[73,74] CT offers superior diagnostic value for blast lung and complex thoracic trauma but is limited by its requirement for fixed installations.[75]

The principal diagnostic tools for respiratory emergencies in disaster settings are summarized in Table 2. Protocols adapted to available infrastructure and personnel can substitute for standard hospital management; oxygen concentrators, simple continuous positive airway pressure devices, and oral antibiotics may be lifesaving.[76] The speed and nature of intervention directly determine outcomes. In chemical inhalation injury, early bronchodilator therapy and humidified oxygen form the foundation of management; in CO poisoning, high-flow oxygen should be initiated immediately. In cyanide toxicity and other chemical exposures, pre-positioned agent-specific antidote protocols are critical.[39,43,69] For aspiration pneumonia and respiratory tract infections, region-specific resistance patterns should guide early empirical antibiotic therapy.[29]

Triage and ventilator allocation are among the most critical challenges during mass respiratory failure events. When ventilator demand exceeds capacity, probability of survival, expected duration of ventilation, and underlying disease status are recommended as triage criteria, and allocation should be coordinated at the regional rather than institutional level.[77] Respiratory support should follow a stepped approach from nasal cannula and high-flow oxygen therapy to non-invasive and invasive mechanical ventilation, with pre-positioned contingency protocols for oxygen-dependent patients. In low- and middle-income countries, mechanical ventilation has been associated with crude mortality of 36–72% and a high risk of iatrogenic complications.[78]

ARDS represents the most severe respiratory presentation in disaster settings and requires a specialized approach. Lung-protective ventilation is the cornerstone, with tidal volumes of 4–8 mL/kg predicted body weight, plateau pressures below 30 cmH_2O, and driving pressures below 14 cmH_2O.[79] Prone positioning for at least 12 hours per day in moderate-to-severe ARDS has been shown, with high-quality evidence, to reduce mortality; high-flow nasal oxygen and neuromuscular blockade may have additional roles in selected patients.[80]

Respiratory protective equipment is a critical component of disaster response, both to protect healthcare workers from occupational exposure and to prevent respiratory injury in affected communities. Equipment selection varies by disaster type, particulate concentration, toxic gas profile, and duration of exposure. N95/FFP2 filtering facepiece respirators are recommended for earthquake and structural-collapse scenarios with high particulate loads and provide protection against dust, silica, and asbestos.[81] In wildfires, N95/FFP2 respirators protect against PM2.5 in smoke, but are inadequate for CO and volatile organic compounds, necessitating separate monitoring

Table 2. Diagnostic tools for respiratory emergencies in disaster settings

Diagnostic tool	Condition assessed	Strengths	Limitations
Lung POCUS	Pneumothorax, pleural effusion, consolidation, pulmonary edema	Rapid, radiation-free, repeatable	Requires training
Pulse oximetry (SpO_2)	Hypoxemia, oxygen requirement screening	Inexpensive, simple, continuous monitoring	Misleading in CO poisoning
Pulse CO-oximetry	Carbon monoxide poisoning	Critical in fire/blast scenarios	Expensive, limited stock
End-tidal CO_2 ($EtCO_2$)	Airway confirmation, ventilation monitoring	Gold standard for tube placement	Requires intubation
Portable chest radiography	Pneumonia, pneumothorax, contusion	Widely used	Radiation; requires portable equipment
Arterial blood gas analysis	Type of respiratory failure, pH, PaO_2/FiO_2	Acid–base and oxygenation assessment	Requires laboratory infrastructure
Clinical scores (CRB-65, NEWS2)	Pneumonia severity, mass casualty triage	Rapid triage, minimal training	CRB-65 preferable in resource-limited settings
Chest CT	Blast lung, ARDS, pulmonary embolism, complex trauma	Very high sensitivity	Requires fixed installation

ARDS: acute respiratory distress syndrome, CO: carbon monoxide, CRB-65: confusion, respiratory rate, blood pressure, age ≥65 years, CT: computed tomography, $EtCO_2$: end-tidal carbon dioxide, NEWS2: National Early Warning Score 2, PaO_2/FiO_2: arterial oxygen partial pressure/fraction of inspired oxygen, POCUS: point-of-care ultrasound; SpO_2: peripheral oxygen saturation

for CO.[19] During volcanic eruptions, N95-equivalent masks have demonstrated filtration efficiency above 89% against volcanic ash.[82] In chemical disasters involving chlorine, ammonia, phosgene, or H_2S, full-face self-contained breathing apparatus or chemical, biological, radiological, and nuclear protection is required.[43] In biological and pandemic scenarios, FFP2/N95 respirators, combined with eye protection, are a standard component of infection control.[83] Disaster planning must determine equipment needs by disaster type and stockpile equipment strategically.

Effective disaster respiratory management requires field-deployable diagnostic capacity, ventilation protocols adapted to resource-constrained settings, pre-configured triage systems, and strategic stockpiling of respiratory protective equipment, all integrated within a comprehensive preparedness framework.

VULNERABLE POPULATIONS

Disaster-related respiratory risks affect entire populations, but specific biological, physiological, and socioeconomic factors place certain groups at disproportionate risk. Children, older adults, pregnant women, patients with chronic respiratory disease, and socioeconomically disadvantaged communities are the most vulnerable groups. Preparedness planning must therefore incorporate strategies tailored to these populations.

Children are biologically more susceptible to air pollutants and disaster-related respiratory threats than adults. Lung development continues from birth to adolescence, and PM2.5 exposure during this period not only triggers acute respiratory infections and asthma exacerbations but also may permanently constrain lung function development.[84] Elevated weight-adjusted minute ventilation and immature immune responses further increase pediatric vulnerability. Wildfire-related PM2.5 has been shown to increase pediatric respiratory hospitalizations more than urban PM2.5 of equivalent concentration.[23] Prenatal exposure to air pollutants is also linked to adverse respiratory outcomes, with PM2.5 exposure during pregnancy adversely affecting lung development and elevating respiratory disease risk in adulthood.[84]

Older adults are particularly vulnerable in disaster settings due to reduced respiratory reserve, decreased respiratory muscle strength, multimorbidity, and social isolation. Increased post-disaster hospitalization and mortality have been consistently documented in older patients with COPD and asthma.[14,33] Power outages pose a critical and distinct threat to advanced-stage COPD patients on home oxygen or domiciliary mechanical ventilation; large epidemiological studies have demonstrated a significantly increased risk of hospitalization among oxygen-dependent COPD patients during outages.[33]

Pregnant women face heightened respiratory risk in disaster settings, both for themselves and for the developing fetus. Exposure to air pollutants and extreme climate events increases the risk of preterm birth and low birth weight. PM2.5 exposure during pregnancy, particularly in the second trimester, has been shown to significantly increase the risk of preterm birth, with first-trimester exposure associated with reduced birth weight.[85] A systematic review conducted in the United States found that hurricanes, floods, and tropical cyclones have been consistently linked to adverse perinatal outcomes.[86]

Low socioeconomic status has emerged as an independent determinant of disaster-related respiratory risk. Disadvantaged communities are geographically closer to climate-related disasters, face barriers in accessing health services and protective equipment, and contend with inequitable post-disaster reconstruction. World Bank data indicate that 80% of the 7.3 billion people globally exposed to unsafe PM2.5 concentrations live in low- and middle-income countries, where disaster-related respiratory injuries are reported to be more severe.[87]

CLIMATE CHANGE AND THE INCREASING DISASTER BURDEN

Climate change is fundamentally transforming the frequency, severity, and geographic distribution of disasters, with corresponding increases in the societal burden of respiratory disease. In 2023, the global mean surface temperature reached 1.45 °C above the pre-industrial baseline, breaking all previous records. This warming has been directly linked to Canada's record-breaking wildfire season, devastating floods in the Horn of Africa, and lethal heatwaves across the Northern Hemisphere. The 2024 Lancet Countdown report documented that 10 of the 15 tracked health indicators reached alarming new records in the most recent year of data.[88] The European Lancet Countdown report showed that, in a survey of 185 European cities, 48% identified exacerbations of respiratory disease as the second most common climate-related health concern.[89]

The most direct effect of climate change on respiratory health occurs through wildfires. Shifts in temperature and precipitation patterns are increasing both the frequency and severity of wildfires, with implications for global respiratory health.[20] The January 2025 LA wildfires demonstrated the potential for such events to expand into densely populated urban areas, with documented increases in cardiovascular and respiratory healthcare utilization.[24]

The respiratory impact of climate change extends beyond wildfire smoke. Rising CO_2 concentrations increase both the quantity and the allergenicity of plant pollen. In North America, the pollen season has advanced by approximately 20 days, and pollen concentrations have risen by 21%.[90] Thunderstorms with elevated humidity can fragment pollen grains into thousands of smaller particles that reach the lower airways, triggering severe asthma exacerbations (thunderstorm asthma) through osmotic shock and microaerosol formation; the frequency of such extreme weather events is rising with climate change.[91] Higher temperatures elevate ground-level ozone, accelerated post-flood mold growth exacerbates atopic respiratory disease, and heatwaves produce high concentrations of ozone and particulate matter that increase mortality in patients with chronic respiratory disease.[88,89]

Low- and middle-income countries bear a disproportionate share of the climate-related disaster burden. Limited climate adaptation capacity results in more severe respiratory injury, earlier collapse of health infrastructure, and greater difficulty in protecting vulnerable populations. Strategies for disaster risk reduction and health system strengthening must therefore be integrated into climate adaptation policy. While the Sendai Framework provides direction, current implementation lags

substantially behind the rising burden of climate-related respiratory emergencies.

CONCLUSION

Disasters—from earthquakes and pandemics to wildfires and chemical incidents—target the respiratory system through a broad range of mechanisms, and each disaster type produces acute and chronic respiratory injury via distinct pathophysiological pathways. This review demonstrates that respiratory emergencies represent a universal threat across disaster types, that vulnerable populations bear a disproportionate share of the risk, and that climate change is steadily increasing the disaster-related respiratory burden.

Three priorities emerge for the field. First, research must address persistent gaps in long-term respiratory follow-up of disaster survivors, the development of disaster-specific clinical protocols, and the inclusion of underrepresented geographic regions in the evidence base.

Second, preparedness planning must position respiratory health more centrally, with pre-stockpiled respiratory protective equipment, pre-configured triage and ventilator allocation frameworks, and strengthened diagnostic capacity at the field level. Third, in the Turkish context, the lessons of the Kahramanmaraş earthquake—from dust exposure to refugee respiratory health—should inform the integration of respiratory specialists into national disaster response architecture.

Meeting the rising burden requires a multidisciplinary agenda uniting respiratory physicians, disaster medicine specialists, public health policymakers, and climate scientists.

Footnotes

Authorship Contributions

Concept: A.E.K., B.Ö.K., Design: A.E.K., B.Ö.K., Data Collection or Processing: A.E.K., B.Ö.K., Analysis or Interpretation: A.E.K., B.Ö.K., Literature Search: A.E.K., B.Ö.K., Writing: A.E.K., B.Ö.K.

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