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REVIEW

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Health impacts of biomass burning aerosols: Relation to oxidative stress and inflammation

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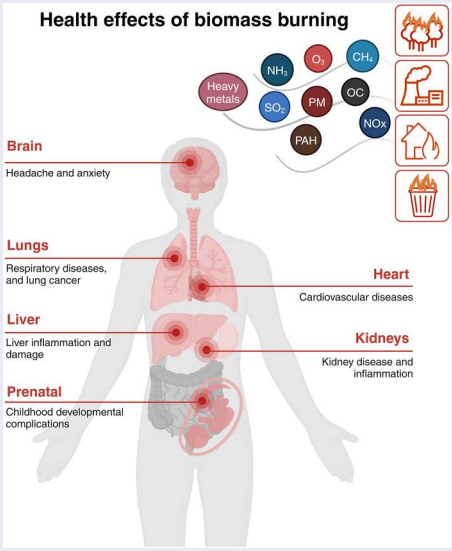
ABSTRACT

Exposure to biomass-burning particulate matter (PM) is associated with various adverse health effects, including respiratory and cardiovascular conditions, cancer, and systemic effects. Multiple mechanisms underlying PM toxicity components derived from biomass burning elicit harmful effects, such as reactive oxygen species (ROS) generation, inflammation, genotoxicity, and tissue-specific damage. Specific compounds or families of compounds present in biomass-burning PM, such as polyaromatic hydrocarbons (PAHs) and their derivatives, have been identified as key contributors to the observed health effects. Their roles in oxidative stress, DNA damage, and cell death have been elucidated in various organs, such as the lungs, liver, kidneys, and brain, providing valuable insights into the systemic biological influence of biomass-burning-related diseases. Current knowledge of the impact of biomass burning highlights the imperative need for further research to understand the health implications of this environmental challenge and the importance of mitigating the adverse effects of increased exposure to biomass-burning pollution to protect the well-being of exposed populations worldwide. This review focuses on the crucial roles of oxidative stress and inflammation in mediating health effects, triggered by exposure to biomass-burning aerosols. It examines various aspects of the health-related impacts of biomass-burning emissions, particularly those from PM components. The review highlights the health consequences on exposed populations, emphasizing specific biochemical responses, contributions to toxicity mechanisms, tissue-specific effects, and the families of compounds responsible for these effects.

GRAPHICAL ABSTRACT



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1. Introduction

The health impacts of biomass burning have recently become a subject of increasing concern because of its significant influence on the environment and human well-being (Aguilera et al. 2021). Biomass burning involves a wide range of activities, including accidental wildfires (forest fires and peatland smoldering), agricultural fires due to land-use change, residential wood and solid-fuel combustion, and power generation, which collectively affect climate and human health (Chen et al. 2017; Fuller et al. 2022; Karanasiou et al. 2021; Lelieveld et al. 2015; Olsen et al. 2020; Woo et al. 2020). Biomass burning substantially influences global air quality, particularly in regions such as South America, Africa, North America, the Amazon region, Siberia, the Indo-Gangetic Plain (IGP) of India, and Australia, where it is the primary source of air pollution (Aguilera et al. 2021; Burke et al. 2023; Lelieveld et al. 2015; Ojha et al. 2020; Patel, Satish, and Rastogi 2021). Biomass burning is widespread in Southeast Asia and Europe for various purposes, including agriculture, cooking, residential energy production, and waste disposal (Chen et al. 2017; Fuller et al. 2022; Lelieveld et al. 2015; Olsen et al. 2020). The global anthropogenic emission of primary particulate matter smaller than 2.5 µm ($PM_{2.5}$) is about 50,000 Gg per year (Klimont et al. 2017; Sigsgaard et al. 2015), with a large fraction of these emissions originated by biomass combustion (~16,000 Gg/year) i.e., either by residential combustion or agricultural fires. In 2010, the contribution of wildfires added about 33,000 Gg of primary $PM_{2.5}$ emission (Klimont et al. 2017). These wildfire emissions are enhanced by climate change, arson and forestry, which are indirectly influenced by mankind. Biomass burning exposure can be categorized into two primary groups: short-term exposure, which involves occasional and seasonal exposure often encountered in communities near forest fires or intentional agricultural burning (Aguilera et al. 2021; Burke et al. 2023; Drventić et al. 2023; Karanasiou et al. 2021), and long-term exposure, characterized by chronic exposure resulting from the continuous use of biomass materials for cooking or heating (Arslan, Aykan Tüzün, and Arslan 2020; Kamal et al. 2022; Rahman, Niemann, and Munson-McGee 2022; Wen et al. 2023).

The direct health implications of biomass burning are significant, as millions of people worldwide are exposed to mixtures of airborne particles and irritant gases from burning biomass emissions (Chen et al. 2017; Karanasiou et al. 2021; Lelieveld et al. 2015; Ojha et al. 2020; Oliveira et al. 2019; Patel, Satish, and Rastogi 2021). These residential emissions release sulfur dioxide (SO_2), nitrogen oxides (NOx), volatile organic compounds (VOCs), and ammonia (NH_3), which are critical precursors for secondary aerosols and tropospheric ozone (O_3) formation (Andreae 2019; Lim et al. 2019). Biomass combustion results in substantial greenhouse gas emissions, including methane (CH_4) and carbon dioxide (CO_2), comparable to fossil fuel combustion (Yoro and Daramola 2020). PM emissions from biomass burning, within the $PM_{2.5}$ fraction, comprise a mixture of black carbon (BC), brown carbon (BrC), organic carbon (OC), inorganic materials, and potentially heavy metals (Li, Misovich, et al. 2021; Matamba et al. 2021; Reid et al. 2005). In addition, wildfires represent a global phenomenon that releases substantial amounts of smoke pollution, affecting air quality and public health, carrying pollutants like PM, VOCs, polyaromatic hydrocarbons (PAHs), phenols, carbonyls, alcohols, and benzene across vast distances (Aguilera et al. 2021; Burke et al. 2023; Matamba et al. 2021; Reid et al. 2016), with PM being the most prevalent and concerning pollutant (Adachi et al. 2019; Li, Misovich, et al. 2021; Oliveira et al. 2019). The composition, transport, and toxicity of wildfire smoke are influenced by various factors, including fuel type, combustion phase, landscape characteristics, aging by atmospheric chemical reactions, weather conditions, and climate change (Chen et al. 2017; Karanasiou et al. 2021; Lelieveld et al. 2015; Oliveira et al. 2019; Reid et al. 2016). Recent trends, such as the increased frequency and intensity of wildfires in regions such as the western USA and Australia, underscore the increasing importance of understanding the health implications of biomass burning (Chen et al. 2017; Karanasiou et al. 2021; Lelieveld et al. 2015; Oliveira et al. 2019). Particularly, in developed areas where pollution is controlled and biomass burning events are challenging, health effects increase owing to the aging population, which is more sensitive to exposure (Andersen et al. 2007; Sigsgaard et al. 2015).

The health effects of biomass-burning emissions are strongly driven by oxidative stress and inflammation (Bayo Jimenez et al. 2022; Danielsen et al. 2010; Hahad et al. 2020; Mondal et al. 2018; Pardo, Qiu, et al. 2020) (Figure 1). Key molecular pathways include the NF-E2–related factor 2 (Nrf2) pathway, which regulates the cellular antioxidant response to counteract the oxidative damage caused by reactive oxygen species (ROS), and the aryl hydrocarbon receptor (AhR) pathway, which triggers inflammation and helps metabolize toxins when exposed to biomass

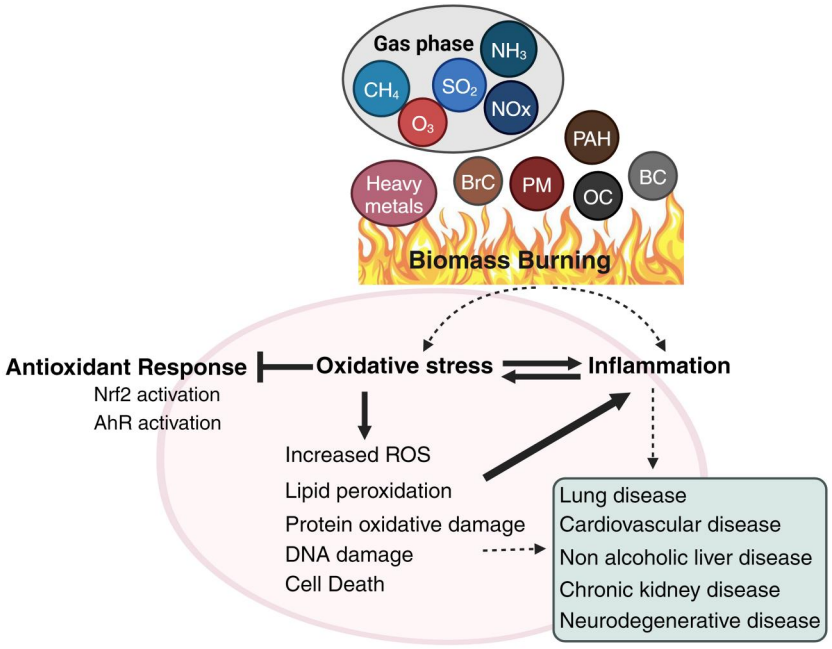

Figure 1. Biomass burning exerts its influence through inflammation and oxidative stress. Exposure to biomass-burning materials generates reactive oxygen species (ROS) and plays a central role in various aspects of inflammation. ROS can oxidize and harm essential molecules within the body, including lipids, proteins, and DNA, potentially leading to cell death and, subsequently, the development of diseases. Disruption of redox balance also influences the activation of redox-sensitive signaling pathways, such as Nrf2 and aryl hydrocarbon receptor (AhR). Furthermore, PAHs and other aromatic pollutant from biomass burning activate the AhR pathway, further contributing to the physio-pathological inflammatory effects. Gas phase: sulfur dioxide (SO_2), nitrogen oxides (NO_x), ammonia (NH_3), ozone (O_3), methane (CH_4), and carbon dioxide (CO_2). Particulate matter (PM), polycyclic aromatic hydrocarbons (PAH), black carbon (BC), brown carbon (BrC), and organic carbon (OC).

smoke components Understanding these mechanisms in specific tissues and organs is essential for developing strategies to mitigate the health consequences of air pollution caused by biomass burning (Croft et al. 2017; Danielsen et al. 2010; Efriza et al. 2023; Li et al. 2015; Milani et al. 2020; Pardo, Qiu, et al. 2020; Patel et al. 2020).

2. Oxidative potential (OP) of biomass-burning aerosols and its impact on human health

2.1. Biomass-burning fractions and OP

Biomass-burning emissions change the atmospheric composition and aerosol properties (de Miranda et al. 2016; Ponczek et al. 2022). The complex interplay between aerosol composition (Hartikainen et al. 2020), organic and inorganic components (Miersch et al. 2019), and their impact on human health and air quality reciprocally influences the oxidative potential (OP) of particles. OP refers to the ability of certain components in atmospheric particles to generate ROS and reduce the levels of antioxidants (Farahani et al. 2022; Li et al. 2023; Li, Li, et al. 2022; Tuet et al. 2019; Wong et al. 2019). Oxidizing compounds that induce OP include quinones, organoperoxides, peroxyacyl nitrates, electron-deficient alkenes, and transition metals. Such pollutants can be enriched in fresh and atmospherically processed biomass-burning emissions, consequently contributing to urban $PM_{2.5}$ OP (Chowdhury et al. 2022; Ojha et al. 2020; Patel, Satish, and Rastogi 2021). Antioxidants, such as phenols, polyphenols (Kjällstrand and Petersson 2001; Li et al. 2023), or compounds which can be further metabolized to oxidants or redox-cycling active compounds (e.g., PAH, and PAH-polyols) are also present in biomass burning aerosols. The balance of oxidative and antioxidative compounds, determines the OP burden of biomass-burning PM. For example, in Milan, Italy, during the winter season, enhanced biomass-burning activities resulted in a substantial increase in OP, which was identified as the primary contributor to the OP of $PM_{2.5}$ (Farahani et al. 2022; Hakimzadeh et al. 2020). In another study, the OP of water-soluble biomass-burning aerosols in the Brazilian Amazon induced higher levels of intracellular ROS and reactive nitrogen species (RNS) than ambient samples collected in Atlanta and laboratory-generated biomass-burning SOA aerosols (Tuet et al. 2019). A notable correlation has been established between ROS/RNS and the presence of levoglucosan, a tracer for biomass burning,

suggesting that levoglucosan could serve as a predictor of oxidative stress during biomass burning exposure (Tuet et al. 2019).

2.2. Impact of atmospheric aging on OP

The OP in $PM_{2.5}$ is influenced by atmospheric chemical transformations, including aging processes and environmental conditions, which create intricate spatiotemporal variations and affect toxicity. Artificial aging in the laboratory exposes aerosols to high oxidant concentrations, simulating rapid oxidation but potentially oversimplifying real-world conditions. In contrast, natural aging occurs over time through gradual exposure to sunlight and natural oxidants, leading to complex changes in aerosol chemistry. This distinction is crucial for assessing aerosol toxicity accurately and understanding the broader environmental and health implications. Both types of aging processes are discussed in this section: During the fire season in Greece (July to October 2016 and 2017), OP increased owing to atmospheric aging (Li, Li, et al. 2021; Wong et al. 2019). Laboratory aging processes, such as photochemical aging (induced by UVB and UVA photolysis), ozone exposure, and OH oxidation, can transform the OP of the water-soluble fraction of wood smoke within 2 days (Wong et al. 2019). Humic-like substances (HULIS) constitute a significant redox-active fraction of biomass-burning organic aerosols. Alkaline neutralization increases the HULIS OP and peroxide contents. Dark aging subsequently results in pH-dependent toxicity and chemical changes. This process implies that a lung fluid-neutral environment can modify the OP and peroxide content of inhaled biomass-burning HULIS (Li et al. 2023; Li, Li, et al. 2022). The biomass-burning-derived fractions (primary BrC fractions, HULIS, and SOA) have strong OP. They generate ROS with varying intensities among the biomass types (Fan et al. 2018), and the OP values of the biomass burning fractions are higher than those of SOA or most secondary urban aerosols (Figure 2).

2.3. OP and cells toxicity

Studies have demonstrated a consistent relationship between OP measurements of aerosols and cellular toxicity. For example, phenol and guaiacol from biomass-burning smoke, which exhibits high OP, can form SOA that damages lung cells (Fang et al. 2024). In contrast, tar materials collected from wood pyrolysis showed reduced OP and were associated with no detectable lung cell death at environmentally relevant doses (Fang et al. 2021). Atmospheric aging has also been shown to alter OP, correlating with changes in cytotoxicity. This effect was observed with HULIS, where aging modified their cytotoxic impact on lung epithelial cells in a manner that paralleled changes in their OP (Li et al. 2023). The OP from biomass burning leads to the production of ROS, which can cause oxidative stress and various cytotoxic effects (Li, Li, et al. 2021; Tuet et al. 2019). This indicates that both the nature of the pollutants and their interactions with environmental factors influence their cytotoxic effects.

Therefore, understanding the OP of biomass-burning aerosols is essential to understanding their implications for human health and air quality. OP is a reliable measure of the oxidative toxicity of $PM_{2.5}$ exposure. Future research should focus on OP and not just the mass concentration of $PM_{2.5}$ when addressing air pollution control. The effects of biomass-burning emissions, their transformation during aging, and their role in oxidative stress highlight the need for further research and development of effective air quality control and public health policies.

3. Health effects

Evaluating the adverse health effects of biomass burning requires a combination of epidemiological investigations and laboratory-based experimental studies. Epidemiological studies are crucial to connect air pollution exposure to its impact on human health. These studies utilize advanced techniques such as biomonitoring, which directly measures exposure biomarkers in human subjects, and geographic information systems (GIS) alongside statistical models to analyze spatial and temporal pollution exposure, confounding factors, and health outcomes. However, such studies can be very long, expensive, and time-consuming, and the results of mathematical statistics are sometimes biased and cannot prove causation or elucidate biological mechanisms (Adetona et al. 2017; Aguilera et al. 2021; Arslan, Aykan Tüzün, and Arslan 2020; Bessa et al. 2023; Cherry et al. 2022; Christensen et al. 2022; Kamal et al. 2022; Oliveira et al. 2019; Ranathunga et al. 2022). *In vivo* and *in vitro* studies are frequently performed to assess PM toxicity through controlled experimental conditions. *In vivo*, studies utilize live animal models to explore broader physiological effects and the mechanisms of disease development due to PM exposure. *In vitro* studies are

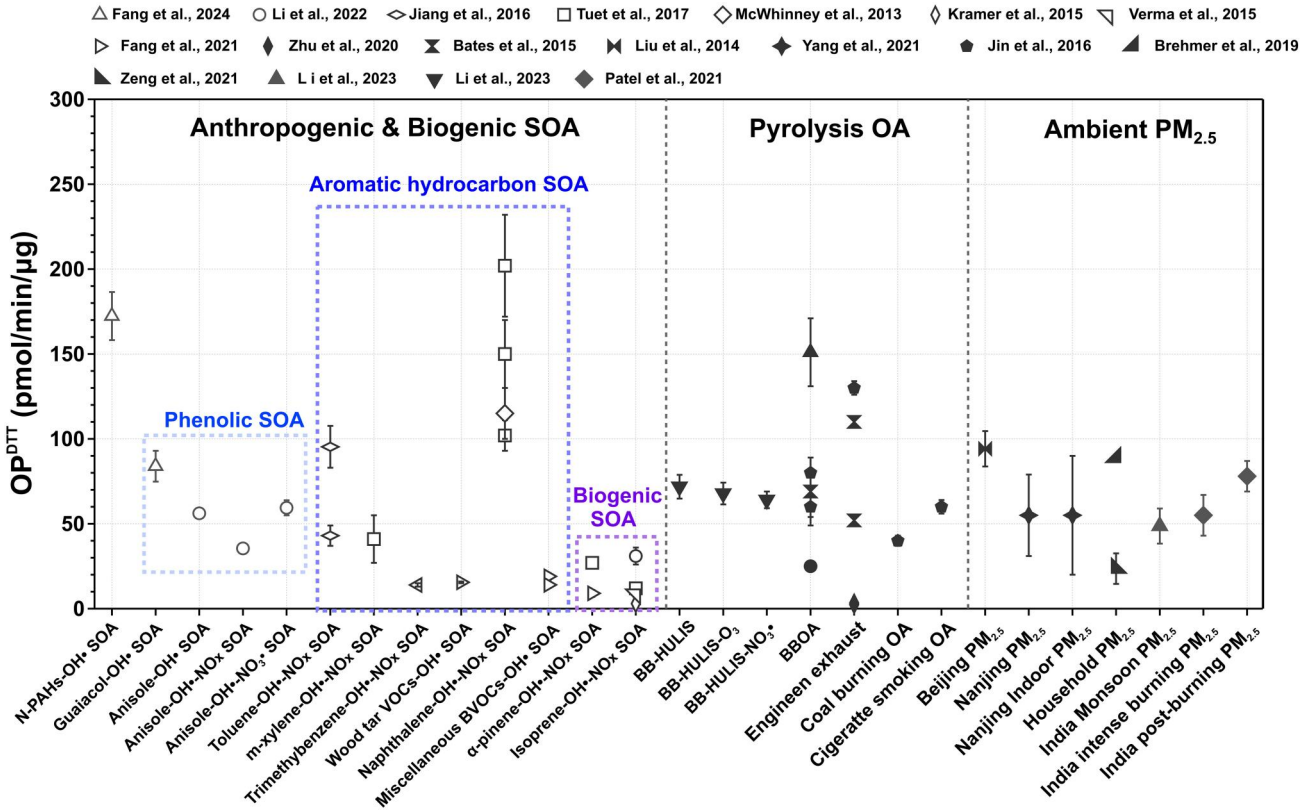

Figure 2. Oxidative potential of various organic aerosol subtypes, including secondary organic aerosols from diverse VOCs, primary aerosols from incomplete combustion of biomass and fossil fuels, and ambient $PM_{2.5}$. Phenolic secondary organic aerosol (SOA) is marked in a light blue dash line; aromatic hydrocarbon SOA is marked in a dark blue dash line; biogenic SOA is marked in a purple dash line. The box plot on the right compares the statistical average oxidative potential of biomass burning organic aerosol and urban $PM_{2.5}$ (Bates et al. 2015; Brehmer et al. 2019; Fang et al. 2022; Jiang et al. 2016; Jin et al. 2016; Kramer et al. 2016; Li et al. 2023; Li, Misovich, et al. 2022; Liu et al. 2014; McWhinney, Zhou, and Abbatt 2013; Patel, Satish, and Rastogi 2021; Tuet et al. 2017; Verma et al. 2015; Yang, Liu, and Qian 2021; Zhu et al. 2020).

pivotal as they enable researchers to observe the direct cellular effects of PM. These studies typically employ cultured cells and advanced techniques like high-throughput screening to assess the impacts of various pollutants. These diverse research methodologies—epidemiological, *in vivo*, and *in vitro*—can establish a robust framework for understanding the complex interactions between biomass burning emissions and human health. The following sections will delve into the specific findings from these research methodologies, highlighting how exposure to biomass burning emissions impacts human health.

3.1. Respiratory and cardiovascular systems

3.1.1. Epidemiology studies influencing the respiratory and cardiovascular systems

Extensive studies have been conducted on the health implications of indoor air pollution resulting from biomass burning in developing (Kamal et al. 2022; Oliveira et al. 2019; Pathak, Gupta, and Suri 2020; Ranathunga et al. 2022) and developed countries (Andersen et al. 2007; Olsen et al. 2020; Roomaney et al. 2022; Sigsgaard et al. 2015). This discussion focuses on epidemiological findings from regions where communities utilize wood for heating and/or cooking in their homes. We also discuss smaller-scale studies on occupational health risks in populations exposed to biomass and burning smoke.

Women from Ghana (Van Vliet et al. 2019) and Honduras (Walker et al. 2020) exposed to household air pollution reported frequent coughing, wheezing, phlegm, and clinic visits for respiratory infections (Van Vliet et al. 2019; Walker et al. 2020). In a large study of household air pollution across 11 low- and middle-income countries, lower levels of lung function with increased hospitalization and all-cause mortality in cross-sectional analyses were observed among individuals who used solid fuels for cooking in comparison with those who used clean fuels (Wang et al. 2023). Toxicological measurements showed that chronic exposure to household biomass fuel increased leukocyte and airway inflammation, elevated ROS levels, activated Nrf2, and reduced antioxidant enzyme

activity in the airways of women. These changes represent adaptive responses to the oxidative stress and inflammation caused by exposure to biomass fuels (Mondal et al. 2018).

The use of biomass as cooking fuel is associated with chronic bronchitis and obstructive airway disease (COPD). Women exposed to biomass and solid fuels have a higher prevalence of COPD, with clinical diagnosis indicating a more significant risk than spirometer-based diagnosis alone (Kamal et al. 2022). A systematic review and meta-analysis revealed that exposure to indoor air pollution due to biomass (fuel exposure compared to other fuels) in adults significantly causes COPD and chronic bronchitis (Pathak, Gupta, and Suri 2020), providing information on how air pollution affects lung function and respiratory disease.

A notable link has been demonstrated between increased instances of common respiratory symptoms and the level of smoke exposure experienced by firefighters (Cherry et al. 2022; Greven et al. 2011). A positive correlation was identified between the frequency of firefighting in the past year and the occurrence of respiratory symptoms (cough, throat, and nose) (Cherry et al. 2022). Notably, firefighters exhibit a higher prevalence of asthma than the general population in the Netherlands, especially among individuals with atopic conditions (Greven et al. 2011).

Specific subpopulations with preexisting health conditions (Croft et al. 2017; Greven et al. 2011; Karanasiou et al. 2021) and older adults (DeFlorio-Barker et al. 2019; Johnston et al. 2019; Karanasiou et al. 2021; Liu et al. 2015) are more susceptible to biomass-burning pollutants than the general population. In a study involving patients with cardiac disease, adverse changes in fibrinogen levels were associated with increased concentrations of wood smoke markers. Elevated concentrations of $PM_{2.5}$, BC, and ultrafine particles (UFP, within 100 nm) over the preceding 96 h correlated with unfavorable alterations in markers of systemic inflammation and coagulation but not with markers of endothelial cell dysfunction or platelet activation (Croft et al. 2017) and arterial stiffness (Unosson et al. 2013). Other factors, such as ethnicity or socioeconomic status (Karanasiou et al. 2021; Woo et al. 2020), can influence health-related issues. Therefore, the impact of biomass-burning pollution and smoke extends to household air pollution, firefighters, and specific subpopulations, with a demonstrated correlation between the level of exposure and increased incidence of respiratory and cardiovascular symptoms.

3.1.2. In vivo toxicity of the respiratory and cardiovascular systems by biomass burning

As demonstrated in various studies, the toxicology and underlying mechanisms of biomass-burning emissions severely impact the respiratory and cardiovascular systems of mice (Chen et al. 2021). For example, exposure of mice to PM from peat fires or biomass burning smoke has been linked to lung inflammation, including increases in bronchoalveolar lavage fluid proteins, cytokines (such as interleukin (IL)6, tumor necrosis factor (TNF)α, and macrophages inflammatory protein (MIP)-2), neutrophils, and ROS as key contributors. A significant decrease in cardiac function and an increase in post-ischemic cardiac death in an experimentally induced ischemia model (Kim et al. 2019; Kim et al. 2014). Furthermore, acute exposure to PM collected from biomass cooking in rural Indian homes led to pro-inflammatory cytokine production, neutrophilic inflammation, airway resistance, and hyperresponsiveness in the lungs of mice lungs. IL1R, Toll-like receptor (TLR)4, and TLR2 are the primary receptors responsible for eliciting inflammatory responses through myeloid differentiation primary response protein (MyD)88 (Sussan et al. 2014). Transcriptional responses resulting from exposure to wildfire smoke in mouse lungs elicit responses related to cellular immune responses, cytokine signaling, cellular growth, proliferation, cellular stress injury, and cancer. These groupings are aligned with pulmonary toxicity markers (Koval et al. 2022), leading to alveolar damage, such as emphysema of the lung tissue (Wardoyo 2019).

Other materials, such as aerosols from water-soluble wood tar extracts, have demonstrated increased oxidative stress responses in mice and bronchial epithelial cells (Pardo, Li, et al. 2020). Notably, oxidative stress-related changes, such as altered levels of ROS and reduced expression of antioxidant genes, were observed, as evidenced by elevated levels of the lipid peroxidation adduct malondialdehyde (MDA). Exposure to smoke elevated plasma carboxyhemoglobin (COHb) levels and damages the tracheal surface, resulting in epithelial loss. Furthermore, plasma extracellular vesicles (EVs) carry miRNAs associated with cardiovascular disease, showing exposure-induced alterations in mice. Lung and heart gene expression patterns indicated responses related to the Nrf2-mediated oxidative stress response, hypoxia signaling, and hypoxia-inducible factor (HIF)1A signaling during exposure to biomass-burning $PM_{2.5}$, suggesting intercellular and system-level communication between tissues in response to wildfire exposure (Carberry et al. 2022).

The studies discussed here revealed that exposure to biomass-burning pollution, whether from peat fires, pinewood, grain straw, or wood tar aerosols, triggers a wide range of adverse effects. These effects include elevated lung inflammation, oxidative stress, pro-inflammatory cytokine production, airway resistance, alveolar damage, and damage to the tracheal surface. Notably, the type of fuel and combustion conditions influenced the outcomes, with smoke from flaming combustion showing greater toxicity, even though it contains a lower mass of PM. However, it is essential to consider that health risks due to exposure are a function of both toxicity and the mass of PM. Therefore, exposure to high concentrations of smoke from smoldering combustion could pose a more significant hazard than exposure to more toxic but much lower mass concentration flaming smoke. This highlights that both the toxicity and concentration of PM must be considered when assessing health effects.

3.1.3. In vitro toxicity of the respiratory and cardiovascular systems by biomass burning

Cell culture experiments allow the evaluation of specific components of biomass burning and their biological effects. Studies on A549, MRC5 human lung cells, and cardiomyocytes exposed to PM_{10}, $PM_{2.5}$, or PAHs during biomass burning have revealed multiple adverse effects, including elevated ROS levels, inflammatory cytokines, autophagy, DNA damage, apoptosis, and necrosis (Bae et al. 2022; de Oliveira Alves et al. 2017; do Nascimento et al. 2023; Kanashova et al. 2018; Lima de Albuquerque et al. 2021; Marchetti et al. 2019; Marchetti et al. 2021; Pardo et al. 2021; Qi et al. 2019). Mitochondrial damage emerged as an early event in this process (Atwi et al. 2022; Bayo Jimenez et al. 2022; Bessa et al. 2023; Dubick et al. 2002; Pardo et al. 2021; Pardo, Li, et al. 2020; Pardo, Qiu, et al. 2020). *In vitro* exposure of human lung epithelial cells to PM from burning oak, pine, and hickory resulted in lower metabolic activity (Atwi et al. 2022; Lima de Albuquerque et al. 2021; Marchetti et al. 2019; Marchetti et al. 2021), whereas aged PM induced more cell death by apoptosis, probably due to changes in OP (Atwi et al. 2022; Li et al. 2023; Li, Li, et al. 2021). Nitroaromatics are important markers from primary and secondary biomass-burning emissions and are major pro-inflammatory components in urban $PM_{2.5}$ (Zhang et al. 2023). Specifically, nitrophenols from biomass and fuel combustion induce cytotoxic effects, membrane disintegration, and phospholipid rearrangements in lung cell lines (BEAS-2B and A549) (Majewska et al. 2021).

Several studies have examined the influence of biomass-burning materials on phase I and phase II antioxidant systems, including Nrf2 and AhR (Kanashova et al. 2018; Marchetti et al. 2019; Marchetti et al. 2021). Long-term exposure to $PM_{2.5}$ from biomass combustion induced an anti-oxidative response through Nrf2 activation and increased intracellular glutathione levels, which was facilitated by the c-Jun N-terminal kinase JNK1/2 in BEAS-2B cells (Merk et al. 2020). The exposure of women in rural villages in China to biomass burning-influenced $PM_{2.5}$ samples resulted in changes in AhR-related and Nrf2-related genes (*ho1, sod1/2, nad(p)h quinone dehydrogenase (nqo)1, and catalase*) in human lung epithelial cells (Lai et al. 2021). Exposure to combustion-derived particles from wood and pellets leads to alterations in cell migration and inflammation by NF-κB, inducing pro-carcinogenic effects in AhR-deficient A549 cells (Marchetti et al. 2019; Marchetti et al. 2021; Vázquez-Gómez et al. 2022). These studies highlight the complex interactions between biomass-burning materials and cellular responses, including those of the Nrf2 and AhR systems, and their potential health implications.

Activation of transient receptor potential ankyrin (TRPA)1 and epidermal growth factor receptor (EGFR), along with changes in p38, mitogen-activated protein kinase (MAPK), and glycogen synthase kinase (GSK)3β activity, played a role in the overproduction of gel-forming glycoprotein MUC5AC in bronchial epithelial cells exposed to biomass-burning PM (Memon et al. 2020). In studies examining gene and pathway responses to $PM_{2.5}$ toxicity from biomass burning, various genes (*jun, cxcl8, mx2, il1a,* and *ptgs2*) involved in inflammation, differentiation, apoptosis, cell migration, and wound healing were identified (Li et al. 2015; Yuan and Zhang 2023).

The exposure of human lung cells discussed here, shows elevated levels of ROS, inflammatory cytokines, autophagy, DNA damage, apoptosis, necrosis, and cell death. Moreover, specific signaling pathways, including EGFR, p38 MAPK, and GSK3β, have been implicated in bronchial epithelial cells exposed to biomass smoke PM. These findings collectively highlight the intricate signaling mechanisms involved in the cellular responses to biomass-burning PM and offer a deeper understanding of its impact on respiratory health.

In this section, we explore the impact of biomass burning aerosols on respiratory and cardiovascular diseases. Epidemiological studies from developing and developed regions have demonstrated that exposure to pollutants, mainly PM and PAHs, is associated with various respiratory and cardiovascular conditions.

Notably, exposure to PAH-rich smoke from household biomass use has been correlated with increased cases of chronic bronchitis and COPD. This correlation is supported by *in vivo* research showing that PM from biomass burning triggers lung inflammation, elevated ROS levels, and significant cardiovascular effects in mice models, indicating a direct physiological response to these pollutants. *In vitro* experiments further these findings by demonstrating that biomass-derived PM and PAHs induce oxidative stress and inflammatory cytokine production in human lung cells, leading to cellular damage and apoptosis. These detailed mechanistic insights highlight the crucial role of specific pollutants, such as PAHs and PM, in driving systemic health effects.

3.2. Impacts on liver function

As a central organ in metabolic processing and detoxification, the liver plays a crucial role in managing the systemic impacts of particles from biomass burning (Arslan, Aykan Tüzün, and Arslan 2020; Milani et al. 2020; Zundel et al. 2022). The extrapulmonary distribution of these particles, particularly from the lungs to other vital organs, is pivotal in understanding the systemic effects of PM on human health (U.S. Epa 2019). The translocation of these particles or components primarily involves two key pathways: direct penetration and immune response facilitation. Ultrafine particles (UFPs), those less than 100 nanometers, can bypass the defenses of the alveolar region due to their small size (U.S. Epa 2019). These particles are capable of penetrating the air-blood barrier, entering the bloodstream, and then circulating to deposit in various tissues, including critical organs such as the heart, brain, liver, and kidneys (Arslan, Aykan Tüzün, and Arslan 2020; Milani et al. 2020; Wang et al. 2022; Zundel et al. 2022). The smaller the particles, the deeper their penetration into the body, heightening their potential for causing systemic health impacts. Soluble species of particles can more easily penetrate organs than insoluble ones, yet it is often the insoluble particles that can cause damage to the DNA (Pardo et al. 2021). Additionally, PM can be phagocytized by alveolar macrophages—immune cells responsible for engulfing pathogens and debris. Once internalized, these particles may be transported out of the lungs *via* the lymphatic system, further distributing them throughout the body (Kreyling et al. 2018; Wang et al. 2022). Thus, organs like the kidneys and liver are particularly vulnerable to these circulating particulates, given their roles in filtering and detoxifying the blood, making them potential sites for particle deposition (Kreyling et al. 2018).

Moreover, the accumulation of biomass burning components, notably PAHs, can significantly impact the liver due to their lipophilic, toxic, and carcinogenic properties. PAHs and their derivatives, markers of exposure to wildfire smoke (Adetona et al. 2017), are metabolized and detoxified by the liver. Liver enzymes may transform certain PAHs into highly reactive and toxic metabolites that can damage DNA and other cellular components (Patel et al. 2020; Vondráček and Machala 2021). Hence, understanding how biomass burning might affect the liver is vital for public health.

3.2.1. Epidemiology studies following exposure to biomass burning influencing the liver

No meaningful associations were found between the use of household fuels and the risk of pancreatic or liver cancers. Similarly, lifetime exclusive use of burning fuels does not reveal significant cancer associations (Sheikh et al. 2020). Participants who developed pancreatic and hepatobiliary cancers reported burning kerosene, and very few recently reported burning gas and biomass as the predominant household fuels. However, liver damage and chronic diseases are correlated with exposure to biomass-burning emissions. The long-term self-reported use of solid cooking fuels is associated with a higher risk of chronic digestive diseases. Women, especially those with a body mass index (BMI) $\geq$28, exhibited positive correlations between the use of solid cooking fuels and conditions such as chronic digestive diseases, hepatic fibrosis/cirrhosis, nonalcoholic fatty liver disease (NAFLD), and cholecystitis (Wen et al. 2023). However, another extensive prospective study showed that the extended use of solid fuels for cooking and smoking was independently linked to an elevated risk of mortality from chronic liver disease. Higher risks were observed among those with solid fuel use durations of longer than 20 years and higher levels of exposure (Chan et al. 2020). Other studies have shown a significant association between liver function and exposure to PAH, a major component of biomass-burning emissions (Efriza et al. 2023; Xu et al. 2021). Specifically, urinary PAH metabolites correlated with serum gamma-glutamyltransferase (GGT) levels, indicating liver damage. Additionally, exposure to PAH-albumin and benzo[a]pyrene (BaP)-DNA adducts in the blood increases the risk of hepatocellular carcinoma. Furthermore, higher levels of urinary PAH metabolites were linked to an increased risk of abnormal levels of liver enzymes, such as alanine transaminase (ALT), aspartate transaminase (AST), and GGT, indicating

impaired liver function (Efriza et al. 2023; Hu et al. 2021; Xu et al. 2021). The association between PAHs and the risk of developing NAFLD in the US population indicates that specific PAHs, such as 2-OHN, 2-OHPhe, and 9-OHF, are positively correlated with NAFLD risk. This association is mediated by changes in serum lipid levels, specifically high-density lipoproteins and triglycerides (Hu et al. 2021).

3.2.2. In vivo liver toxicity by biomass burning

Several studies have explored the influence of biomass-burning components on animal liver (Jiang et al. 2021). A study on seasonal and chemical variations in urban $PM_{2.5}$ particles from Beijing, China, highlighted higher pollution levels during the heating season, characterized by the presence of significant PAH in organic extracts. Exposure to these extracts led to increased lung and liver inflammation and activated Nrf2 and related genes associated with antioxidant responses in the liver. Elevated levels of lipid peroxidation adducts in the liver indicate oxidative and toxic damage, and genes related to PAH detoxification are expressed (Pardo et al. 2018; Wardoyo 2019). These findings underscore the effects of biomass-burning components on the liver and the role of Nrf2-mediated antioxidant responses in alleviating the associated inflammation and oxidative stress responses. Liver oxidative damage was also observed in a rat model examining inhalation injuries experienced by firefighters and burn victims. Exposure to fir and pine smoke increases TBARS levels, indicating oxidative damage to the liver (Dubick et al. 2002). Furthermore, in a study involving rats exposed to wood smoke PM and ambient PM collected from areas with operating wood stoves, rats orally exposed to PM from the wood stove area exhibited increased levels of εdG (mutagenic exocyclic DNA adducts), elevated expression of pro-inflammatory cytokines and HO1, and DNA damage in the liver. These findings suggest that wood smoke PM and carbon black (CB) significantly affect the oxidative stress response, inflammation, and genotoxicity in organs other than at the point of entry (Danielsen et al. 2010; Johnston et al. 2019).

These findings collectively demonstrate that exposure to PAH aerosols may damage the liver and induce oxidative stress, inflammation, and genotoxicity, emphasizing the adverse impact of biomass burning on this organ. These findings underscore the need to consider the potential liver-related health risks associated with biomass burning and the importance of antioxidant responses in mitigating inflammation and oxidative stress (Figure 3).

3.2.3. In vitro hepatocyte toxicity by biomass burning

The effects of biomass burning, and PAHs *in vitro* are explored. Retene, an alkylated PAH primarily emitted by biomass combustion, and its potential health implications were examined in human HepG2 liver cells. Although retene had minimal impact on cell viability, it induced DNA strand breaks, micronuclei formation, and ROS production in a dose- and time-dependent manner. Genotoxic effects are associated with the activation of checkpoint kinase 1 (Chk1), a replication stress marker. Oxidative stress has emerged as a key mechanism underlying reten toxicity (Scaramboni et al. 2023). PAHs have been implicated in various molecular and toxicological mechanisms (Andrysík et al. 2007; Patel et al. 2020; Vondráček and Machala 2021), including their role in tumor migration and invasion of liver cells. PAHs induce ROS generation and activate the p38 MAPK signaling pathway, a pivotal factor in the migration of human HepG2 cells (Song et al. 2011) and rat liver epithelial WB-F344 'stem-like' cells (Andrysík et al. 2007). PAH exposure upregulates the expression of genes associated with cell migration, a response inhibited by ROS scavengers (Song et al. 2011). Chrysene, another PAH produced by the incomplete burning of organic materials, reduces cell viability, increases cellular ROS levels, and affects the expression of detoxification enzymes in phases I and II of xenobiotic metabolism. It upregulates cytochrome P450 isoforms and downregulates GSH. Increased inflammatory markers and decreased antioxidant factors, including Nrf2 and its related genes, have been observed (Zhu et al. 2023), suggesting a key role of inflammation and oxidative stress in liver toxicity. Furthermore, exposure of liver cells to wood tar aerosols in an NAFLD model resulted in increased cell death and oxidative stress, including elevated levels of ROS, lipid peroxidation adducts, and a higher degree of DNA damage in cells exposed to wood tar aerosols. Exposure to wood tar aerosols can exacerbate the effects on liver cells, particularly under hepatic steatosis conditions (Pardo et al. 2023).

Biomass burning releases many pollutants, including PM, PAHs, and other VOCs, which can cause significant liver damage. This has been shown by epidemiological, *in vivo*, and *in vitro* studies. Long-term exposure to biomass smoke, especially from household fuels, is linked to an increased risk of chronic liver diseases, such as NAFLD and other digestive diseases. *In vivo* studies on animals exposed to biomass smoke have demonstrated elevated liver inflammation, oxidative stress, and disruptions in

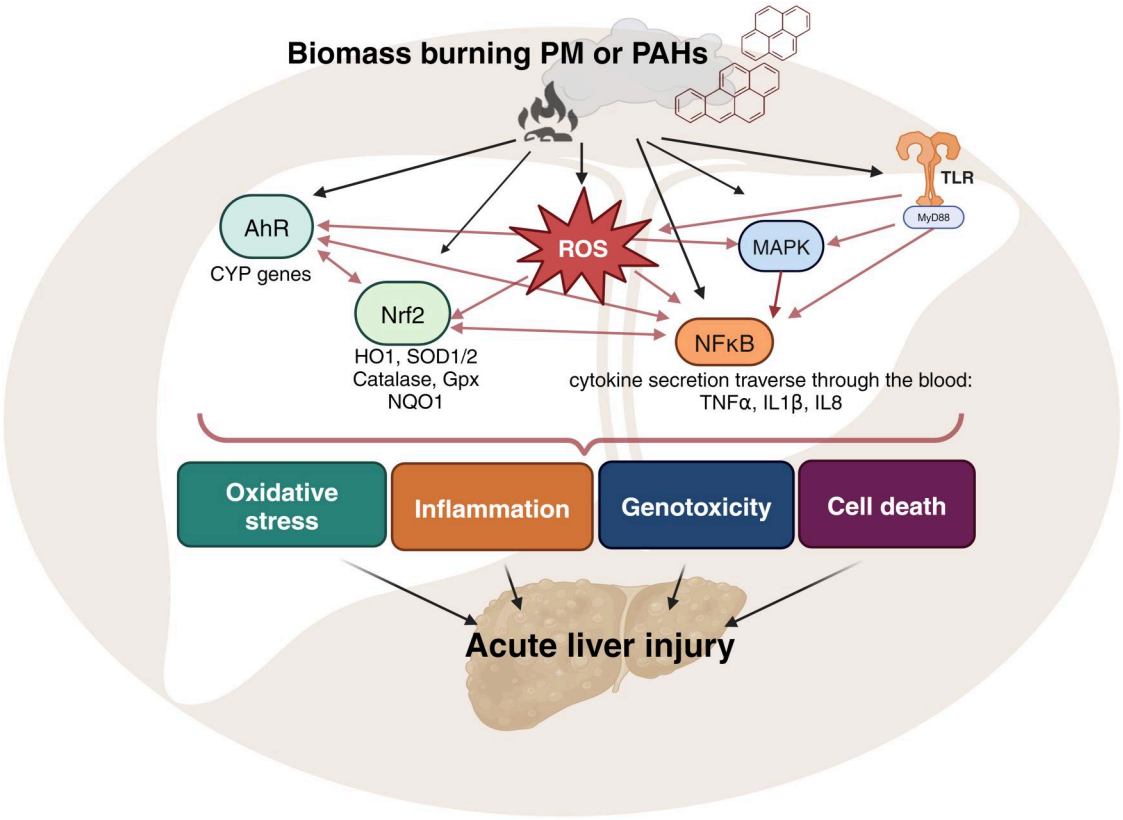

Figure 3. Exposure to biomass-burning particulate matter (PM) or polycyclic aromatic hydrocarbons (PAHs) initiates a complex molecular signaling cascade in the liver. Reactive oxygen species (ROS) are generated following exposure to biomass burning and trigger oxidative stress, inflammation, and genotoxicity through different mechanisms. PAHs can activate aryl hydrocarbon receptors (AhR) mediated by ROS or directly in the liver cells as they traverse through the bloodstream. AhR induces the expression of phase I protective genes (involved in xenobiotic metabolism). Nuclear factor erythroid 2-related factor 2 (Nrf2), a key regulator against oxidative stress, is activated by ROS and upregulates antioxidant genes (heme oxygenase, HO1; superoxide dismutase, SOD1,2; glutathione peroxidase, Gpx1; and NAD(P)H Quinone Dehydrogenase, NQO1). Prolonged or acute exposure disrupts the oxidative stress status, leading to oxidative stress. PAHs or ROS activate nuclear factor-kappa B (NFκB) and generate an inflammatory environment [cytokine secretion can traverse through the bloodstream (tumor necrosis factor, TNFα, interleukin, IL1β, and IL8)]. PAHs can also activate toll-like receptors (TLRs), which are membrane-bound receptors, and further activate the cells' mitogen-activated protein kinase (MAPK) pathway. The MAPK pathway is involved in many cellular responses, including immune responses, which enhance the inflammatory response. Biomass burning PM or PAHs induce oxidative stress inflammation directly or indirectly, contributing to genotoxicity and cumulative damage to the liver cells. Black arrows indicate mechanisms that start outside and influence inside the cells; red arrows indicate inner cell mechanisms.

metabolic detoxification pathways, with a notable activation of Nrf2 and related antioxidant responses. *In vitro* studies have further shown that PAHs from biomass burning can cause genotoxic effects, oxidative stress, and cell death in hepatocyte cultures (Figure 3). These studies reveal a complex interplay between environmental pollutants and biological responses at the molecular level, ultimately leading to significant liver dysfunction and disease.

3.3. Impacts on kidneys function

The kidneys are essential for maintaining the body's internal environment, eliminating waste, regulating fluid and electrolyte balance, and supporting various bodily functions. Impaired or compromised kidney function is a risk factor for non-communicable diseases, such as diabetes, hypertension, and cardiovascular disease (Afsar et al. 2019; Paoin et al. 2022).

3.3.1. Epidemiology studies following exposure to biomass burning influencing the kidneys

Few studies have explored the association between exposure to PAHs and kidney disease or damage in humans. In the Mexican Huasteca Potosina community, where traditional cooking practices involve burning biomass, 100% of the population was exposed to hydroxylated PAHs (OH-PAHs) and exhibited kidney damage biomarkers (Flores-Ramírez et al. 2021). Another study, utilizing data from the 2015–2016 National Health and

Nutrition Examination Survey (NHANES), examined seven different urinary PAH concentrations in relation to chronic kidney disease, defined by the estimated glomerular filtration rate (eGFR) and albumin to creatinine ratio (ACR). The results indicated that only urinary 2-hydroxynaphthalene levels were significantly associated with an increased risk of chronic kidney disease. Consequently, urinary 2-hydroxynaphthalene levels may be linked to chronic kidney disease in this population (Rahman, Niemann, and Munson-McGee 2022). Furthermore, urinary levoglucosan has been studied as a potential biomarker of wood smoke exposure in wildland firefighters for kidney damage. This study found an overall increase in urinary levoglucosan concentrations from pre- to post-shift among firefighters. However, the changes in these concentrations were inconsistent. As such, urinary levoglucosan might not be an effective biomarker of woodsmoke exposure in occupational settings (Naeher et al. 2013) but has proven effective in mice (Migliaccio et al. 2009). However, an analysis of firefighters in South Carolina who were exposed to wildland fire smoke during prescribed burns revealed elevated PAH exposure during the burn season, with significantly higher post-shift OH-PAH levels. Specifically, 4-hydroxyphenanthrene (4PHE) was associated with $PM_{2.5}$ and levoglucosan exposure, suggesting its potential as a valuable biomarker for assessing wildland fire smoke exposure (Adetona et al. 2017). Additionally, analysis of travelers from Germany to China exposed to high levels of PAHs showed higher levels of 8-hydroxy-2′-deoxyguanosine, MDA, F2a-isoprostanes and hydroxylated PAH in urine samples, indicating that exposure to PAH from PM pollution leads to oxidative stress and damage metabolites in the urine (Wu et al. 2017).

Collectively, these studies illustrate that exposure to PAHs and wood smoke can have significant health implications. They provide evidence that PAH exposure is linked to kidney damage and chronic kidney disease, emphasizing the need to consider the health impacts of PAH exposure in affected populations. Additionally, studies have suggested that while some biomarkers, such as urinary 2-hydroxynaphthalene and hydroxylated PAH metabolites, may be useful for assessing exposure to wood smoke and PAHs, their effectiveness as biomarkers can vary and may be influenced by factors other than exposure.

3.3.2. In vivo kidney toxicity by biomass burning

A novel label-free mass spectrometry imaging method was developed to monitor the behavior of carbonaceous aerosol (CA) particles within the human body. A previous study found evidence of CA translocation to extrapulmonary organs, including the kidneys, liver, spleen, and brain (Jiang et al. 2021). The exposure of mice to biomass burning or PAHs significantly affects the kidneys, inducing potential health-related biological mechanisms (Migliaccio et al. 2009; Ruan et al. 2021). Chronic exposure of male mice to phenanthrene, a PAH formed during the incomplete combustion of organic matter, results in kidney hypertrophy, injury, and fibrosis. This exposure also triggers an adaptive immune response, particularly the release of cytokines associated with T helper cells (Ruan et al. 2021). Additionally, $PM_{2.5}$, which contains potentially carcinogenic compounds, induces kidney inflammation, oxidative stress, and alterations in the renin-angiotensin system in mice (Yuan et al. 2022). The exposed mice exhibit downregulation of Nrf2 and HO1 and upregulation of inflammatory cytokines in the kidneys. These findings suggest that oxidative stress and inflammatory reactions influence the regulation of the renin-angiotensin system (Yuan et al. 2022). These examples reveal that various biomass-burning components can lead to kidney hypertrophy, injury, inflammation, oxidative stress, alterations in the renin-angiotensin system, and fibrosis in mice. This further highlights the intricate mechanisms underlying kidney damage caused by environmental pollutants.

3.3.3. In vitro kidney toxicity by biomass burning

In cellular experiments, exposure to $PM_{2.5}$ containing highly toxic PAHs induced inflammation and apoptosis in mouse renal tubular epithelial cells (mRTECs) through the NLRP3/IL-1β pathway. Vitamin D and Vitamin D receptors play crucial roles in detoxifying $PM_{2.5}$ by degrading harmful PAHs and exhibiting anti-inflammatory properties (Huang et al. 2022). Nitrated PAHs (NPAHs), such as 1-nitropyrene, 9-nitroanthracene, and 6-nitrochrysene, derived from incomplete combustion in Ljubljana, Slovenia, were toxic to human kidney cells (HEK293T) (Drventić et al. 2023). In addition, a study that analyzed three common hardwoods (mesquite, cherry, and oak) under three combustion scenarios (flaming, smoldering, and incomplete) showed that during flaming combustion, all hardwoods notably enhanced the promoter activity of the sterile alpha motif pointed domain-containing E-twenty-six transcription factor in HEK293T cells. This transcription factor is a biomarker linked to mucin gene expression and is associated with mucus production in pulmonary diseases. (Singh et al. 2023). Notably, exposure to PAHs from $PM_{2.5}$ exposure significantly decreased cell viability,

increased LDH release, and increased expression of kidney injury marker 1 in human kidney cells (HK-2 cells). This exposure activates both the antioxidant pathway, with increased Nrf2, HO1, and NQO1, and the apoptotic pathway, characterized by elevated levels of pro-apoptotic proteins (caspase-3, caspase-8, and Bax) (Huang et al. 2020).

Research spanning epidemiological, *in vivo*, and *in vitro* studies consistently shows that exposure to biomass burning and its constituents, particularly PAHs, has significant adverse effects on kidney health. Epidemiological data reveal strong associations between biomarkers like hydroxylated PAHs and urinary 2-hydroxy naphthalene with kidney damage and chronic kidney disease. Supporting these findings, *in vivo* studies in mice demonstrate that exposure to phenanthrene and $PM_{2.5}$ from biomass burning induces kidney hypertrophy, inflammation, oxidative stress, and disruptions in the renin-angiotensin system, suggesting severe physiological impacts. Further, *in vitro* experiments illuminate the cellular mechanisms behind these effects, showing that PAHs trigger inflammation and apoptosis in renal cells *via* pathways such as NLRP3/IL-1β and adversely affect cell viability while promoting mucus production—markers indicative of cellular stress and damage. Collectively, these studies highlight the susceptibility of kidney cells to the detrimental effects of environmental pollutants and require further research to support these biological responses.

3.4. Brain and neuropsychological functions

Several studies have shown associations between air pollutants and a wide range of outcomes throughout an individual's lifetime, including their impact on mental health, neurodevelopment, and cognitive aging. Several experimental studies have elucidated the biological effects of air pollutants on the brain, as particles pass through the blood-brain barrier (BBB), which acts as a protective shield against toxins and pathogens. A deeper understanding of how these effects affect brain biology can provide valuable insights into the complex relationship between air pollution and neurotoxicity, ultimately enhancing the design of epidemiological studies that investigate these connections.

3.4.1. Epidemiology studies influencing neuropsychological function

A study investigating the link between exposure to biomass-burning emissions and their effects on headaches and brain damage in young women used blood samples for biochemical analysis and brain magnetic resonance imaging (MRI) scans to assess brain damage. Individuals with a history of biomass smoke exposure had an approximately eight-fold higher prevalence of gliotic foci than those without such exposure. This suggests that indoor air pollution resulting from the use of solid biomass fuel increases the risk of headaches and brain damage (Arslan, Aykan Tüzün, and Arslan 2020). Another study explored the impact of indoor air pollution from biomass combustion on the neurodevelopment of children under five years of age in Sri Lanka. A correlation was found between CO and $PM_{2.5}$ from households using biomass burning as their primary cooking fuel and children exhibiting 'suspected' scores in developmental assessments related to language, social behavior, play, and gross motor skills (Ranathunga et al. 2022). Studies exploring neurodevelopment during pregnancy have shown a link between prenatal exposure to household air pollutants and developmental issues in children aged two years, impacting cognitive, language, and adaptive abilities (Christensen et al. 2022; Morgan et al. 2023).

Additionally, during an extensive wildfire season, research identified a correlation between smoke exposure and an increase in emergency department visits for cardiovascular and cerebrovascular issues among adults, particularly those over the age of 65 (Wettstein et al. 2018).

3.4.2. Neuropsychological function toxicity in vivo and in vitro

Toxicological studies examining the impact of air pollution on neuropsychological functions in animals and neuronal cells have revealed several concerning effects of air pollution on the brain. These effects include impaired neurotransmitter signaling, elevated levels of cerebral cytokines, activation of neuronal immune cells, disruption of the BBB, and elevated oxidative stress levels, as indicated by oxidized low-density lipoprotein (Hahad et al. 2020). In a previous study, male mice were exposed to UFPs from biomass burning via intratracheal instillation. Various brain regions have been examined as markers of oxidative stress, inflammation, and Alzheimer's disease. This exposure leads to inflammatory responses in the mouse brain, causing significant oxidative stress and changes in the amyloid precursor protein (APP) and beta-secretase (BACE)1 protein levels, indicative of neurodegeneration (Milani et al. 2020). Another study involving mice exposed to wood smoke observed notable changes in the brain endothelial cells. This analysis

revealed two distinct populations of endothelial cells with varying levels of CD31 expression. Exposure to wood smoke increased the proportion of CD31Hi cells linked to an anti-inflammatory response, whereas CD31Med cells were associated with a pro-inflammatory response. Metabolomic analysis has shown disruptions in neurotransmitters and signaling molecules in the hippocampus, along with decreased hippocampal NAD + levels, suggesting a dynamic and prolonged neuroinflammatory response with potential long-term behavioral and health implications (Scieszka et al. 2023). Moreover, the influence of paternal exposure to biomass smoke on offspring behavior and cognitive abilities has been studied in male rats (Sosedova et al. 2020; Vokina et al. 2022). The offspring of these exposed males showed reduced exploratory behavior, locomotor activity, and spatial navigation and increased anxiety levels when born immediately after exposure. However, those born after long-term exposure did not significantly differ from the control group in terms of locomotor and exploratory activities. However, females showed elevated anxiety levels and impaired cognitive function (Sosedova et al. 2020).

Studies on PAH neurotoxicity revealed that exposure to BaP impaired neurodifferentiation by increasing cell numbers and decreasing cell enlargement and neurite outgrowth during differentiation. BaP also reduced the expression of neurotransmitter markers, particularly those related to the acetylcholine phenotype, suggesting that even at low, nontoxic levels, BaP directly impairs neurodifferentiation (Olasehinde and Olaniran 2022; Slotkin and Seidler 2009). Additionally, exposure to BaP reduced the density of axons in the hippocampal CA1 and CA3 areas and upregulated pro-inflammatory genes *(tnfα, Il-1β*, *Il-18*, and *Nlrp3)*, indicating a role in neurodegeneration (Abd El Naby et al. 2023). PAH from PM mediated oxidative stress responses by Nrf2 activation, especially in the cerebellum, suggesting that Nrf2-mediated oxidative stress responses may contribute to the damage caused by PAHs (Bayo Jimenez et al. 2022; Haghani et al. 2020).

Research conducted across epidemiological, *in vivo*, and *in vitro* studies consistently demonstrates that biomass-burning emissions have profound effects on brain and neuropsychological functions. Epidemiological evidence links exposure to biomass smoke with significant neurological damage, evident from the increased prevalence of gliotic foci in young women and developmental delays in children who are exposed to household air pollution from biomass fuels. *In vivo* studies reveal that UFPs from biomass smoke not only induce oxidative stress and inflammation in the brain but also disrupt the BBB and alter key proteins associated with neurodegenerative diseases like Alzheimer's. These biological impacts are mirrored in *in vitro* experiments which show that exposure to PM containing PAHs impairs neurotransmitter signaling, enhances pro-inflammatory responses, and disrupts neurodifferentiation processes in neuronal cells. Together, these studies highlight the severe neurotoxic potential of biomass-burning pollutants and emphasize the urgent need for a comprehensive understanding and targeted interventions to mitigate these neurological risks.

4. Conclusions

Biomass burning has become a critical issue with wide-ranging implications for the environment and human health. It encompasses various activities, including wildfires, agricultural fires, residential wood and solid fuel combustion, and power generation, all of which collectively affect air quality and contribute to climate change. Furthermore, wildfires are projected to increase under most global warming scenarios, posing the risk of exposing large populations. Biomass-burning emissions contain various pollutants, including VOC, PM, all of which are linked to oxidative stress and inflammation. Changes in atmospheric composition and aerosol properties resulting from biomass burning emphasize the complex interplay between these factors. While biomass burning releases a diverse array of pollutants, PM and PAHs are a significant focus of this paper. Other significant emissions like VOCs, CO, NOx, SO_2, and CH_4 also contribute to the complex health impacts associated with biomass burning. The shared emission profile of these compounds across different burning contexts highlights the challenges in evaluating the overall health effects of such emissions.

The inhalation of respirable particles emitted from biomass-burning sources poses a significant burden to human health, with the lungs acting as the primary entry point. These particles can induce immediate pulmonary damage, and their impact extends beyond the respiratory system, traversing the bloodstream, reaching vital organs throughout the body, and even passing through the protective BBB. The intricate interplay between respiratory and circulatory systems highlights the far-reaching threat of respirable particle toxicity. Biomass-burning components, such as PAHs, can significantly influence the liver owing to their

toxic and carcinogenic properties. Toxicological studies have demonstrated that exposure to PAHs can cause liver damage *via* oxidative stress, inflammation, and genotoxicity. PAHs may also cause kidney damage, including hypertrophy, injury, fibrosis, inflammation, oxidative stress, and alterations in the renin-angiotensin system. Studies emphasizing the diverse neurological consequences of exposure to air pollutants have underscored the importance of understanding how these effects interact with brain biology and contribute to neuropsychological dysfunction. Less discussed but equally critical are the effects of co-emitted CO, NOx, SO_2, and NH_3. These compounds interfere with blood oxygenation and exacerbate respiratory conditions, while heavy metals such as lead, mercury, and cadmium can pose long-term neurological and renal risks. Furthermore, secondary pollutants such as ozone and SOA, formed from primary emissions, can exacerbate the impact on respiratory and cardiovascular health.

These findings emphasize the need to consider the potential health risks associated with exposure to environmental pollutants. Further research is essential to gain a deeper understanding of the influence of biomass burning on susceptible populations, including the identification of vulnerable subgroups, synergistic effects between several health risk factors, and the development of targeted interventions to mitigate health risks. Factors such as high or poor dietary nutrition, levels of physical activity, and habits such as smoking and alcohol consumption, combined with exposure to biomass-burning emissions, may enhance their detrimental health effects. Exploring the interactions among these risk factors will lead to a more comprehensive understanding of their collective influence on health outcomes. Additionally, investigating the impact of biomass-burning components on remote tissues in the body is crucial to uncover potential systemic impacts and provide a comprehensive assessment of the health consequences of exposure to biomass-burning emissions. Another aspect would be to investigate the chemical complexity of biomass-burning particles, including atmospheric transformation. In summary, a comprehensive evaluation of the health effects of biomass burning through both epidemiological and laboratory-based studies underscores the importance of addressing this global issue to protect the well-being of affected populations and develop strategies for reducing its impact on public health.

Although biomass burning is a significant contributor to global warming and has various implications for human health, the effects of exposure to biomass-burning emissions are not uniform because they are shaped by multiple factors. These factors include emission variability, fuel type, combustion conditions, aerosol properties, exposure duration, complex aging processes that occur in the atmosphere, and uncertainties associated with measurements. To comprehensively understand the influence of biomass burning on air quality, atmospheric composition, and health, it is essential to account for variables such as particle size, distribution, and trajectories of air masses. Such considerations are crucial for distinguishing the contributions of local and remote sources to the complex effects of biomass burning on the environment and global population. Implementing measures to reduce emissions, promoting sustainable practices, and enhancing global cooperation are essential components toward mitigating the impact of biomass burning on air quality and public health.

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References

Abd El Naby, W., S. C. Zong, A. Fergany, F. A. Ekuban, S. Ahmed, Y. Reda, H. Sato, S. Ichihara, N. Kubota, S. Yanagita, et al. 2023. Exposure to benzo[a]pyrene decreases noradrenergic and serotonergic axons in hippocampus of mouse brain. *Int. J. Mol. Sci.* 24 (12):9895. doi: 10.3390/ijms24129895.

Adachi, K., A. J. Sedlacek, L. Kleinman, S. R. Springston, J. Wang, D. Chand, J. M. Hubbe, J. E. Shilling, T. B. Onasch, T. Kinase, et al. 2019. Spherical tarball particles form through rapid chemical and physical changes of organic matter in biomass-burning smoke. *Proc. Natl.*

Acad. Sci. USA 116 (39):19336–19341. doi. doi: 10.1073/pnas.1900129116.

Adetona, O., C. D. Simpson, Z. Li, A. Sjodin, A. M. Calafat, and L. P. Naeher. 2017. Hydroxylated polycyclic aromatic hydrocarbons as biomarkers of exposure to wood smoke in wildland firefighters. *J. Expo. Sci. Environ. Epidemiol.* 27 (1):78–83. doi: 10.1038/jes.2015.75.

Afsar, B., R. Elsurer Afsar, A. Kanbay, A. Covic, A. Ortiz, and M. Kanbay. 2019. Air pollution and kidney disease: Review of current evidence. *Clin. Kidney J.* 12 (1):19–32. doi: 10.1093/ckj/sfy111.

Aguilera, R., T. Corringham, A. Gershunov, and T. Benmarhnia. 2021. Wildfire smoke impacts respiratory health more than fine particles from other sources: Observational evidence from Southern California. *Nat. Commun.* 12 (1):1493. doi: 10.1038/s41467-021-21708-0.

Andersen, Z. J., P. Wahlin, O. Raaschou-Nielsen, T. Scheike, and S. Loft. 2007. Ambient particle source apportionment and daily hospital admissions among children and elderly in Copenhagen. *J. Expo. Sci. Environ. Epidemiol.* 17 (7):625–636. doi: 10.1038/sj.jes.7500546.

Andreae, M. O. 2019. Emission of trace gases and aerosols from biomass burning – an updated assessment. *Atmos. Chem. Phys.* 19 (13):8523–8546. doi: 10.5194/acp-19-8523-2019.

Andrysík, Z., J. Vondrácek, M. Machala, P. Krcmár, L. Svihálková-Sindlerová, A. Kranz, C. Weiss, D. Faust, A. Kozubík, and C. Dietrich. 2007. The aryl hydrocarbon receptor-dependent deregulation of cell cycle control induced by polycyclic aromatic hydrocarbons in rat liver epithelial cells. *Mutat. Res.* 615 (1–2):87–97. doi: 10.1016/j.mrfmmm.2006.10.004.

Arslan, A., F. Aykan Tüzün, and H. Arslan. 2020. Relationship between biomass exposure, chronic headache and brain damage in young women. *Eastern J. Med.* 25 (1):126–131. doi: 10.5505/ejm.2020.25428.

Atwi, K., S. N. Wilson, A. Mondal, R. C. Edenfield, K. M. Symosko Crow, O. El Hajj, C. Perrie, C. K. Glenn, C. A. Easley, H. Handa, et al. 2022. Differential response of human lung epithelial cells to particulate matter in fresh and photochemically aged biomass-burning smoke. *Atmos. Environ.* 271:118929. doi: 10.1016/j.atmosenv.2021.118929.

Bae, H. R., M. Chandy, J. Aguilera, E. M. Smith, K. C. Nadeau, J. C. Wu, and D. T. Paik. 2022. Adverse effects of air pollution-derived fine particulate matter on cardiovascular homeostasis and disease. *Trends Cardiovasc. Med.* 32 (8):487–498. doi: 10.1016/j.tcm.2021.09.010.

Bates, J. T., R. J. Weber, J. Abrams, V. Verma, T. Fang, M. Klein, M. J. Strickland, S. E. Sarnat, H. H. Chang, J. A. Mulholland, et al. 2015. Reactive oxygen species generation linked to sources of atmospheric particulate matter and cardiorespiratory effects. *Environ. Sci. Technol.* 49 (22):13605–13612. doi: 10.1021/acs.est.5b02967.

Bayo Jimenez, M. T., K. Frenis, O. Hahad, S. Steven, G. Cohen, A. Cuadrado, T. Münzel, and A. Daiber. 2022. Protective actions of nuclear factor erythroid 2-related factor 2 (nrf2) and downstream pathways against environmental stressors. *Free Radic. Biol. Med.* 187:72–91. doi: 10.1016/j.freeradbiomed.2022.05.016.

Bessa, M. J., B. Sarmento, M. Oliveira, and F. Rodrigues. 2023. In vitro data for fire pollutants: Contribution of studies using human cell models towards firefighters' occupational. *J. Toxicol. Environ. Health. B Crit. Rev.* 26 (4):238–255. doi: 10.1080/10937404.2023.2187909.

Brehmer, C., A. Lai, S. Clark, M. Shan, K. Ni, M. Ezzati, X. Yang, J. Baumgartner, J. J. Schauer, and E. Carter. 2019. The oxidative potential of personal and household pm2.5 in a rural setting in southwestern china. *Environ. Sci. Technol.* 53 (5):2788–2798. doi: 10.1021/acs.est.8b05120.

Burke, M., M. L. Childs, B. de la Cuesta, M. Qiu, J. Li, C. F. Gould, S. Heft-Neal, and M. Wara. 2023. The contribution of wildfire to pm2.5 trends in the USA. *Nature.* 622 (7984):761–766. doi: 10.1038/s41586-023-06522-6.

Carberry, C. K., L. E. Koval, A. Payton, H. Hartwell, Y. Ho Kim, G. J. Smith, D. M. Reif, I. Jaspers, M. Ian Gilmour, and J. E. Rager. 2022. Wildfires and extracellular vesicles: Exosomal micrornas as mediators of cross-tissue cardiopulmonary responses to biomass smoke. *Environ. Int.* 167:107419. doi: 10.1016/j.envint.2022.107419.

Chan, K. H., D. A. Bennett, O. P. Kurmi, L. Yang, Y. Chen, J. Lv, Y. Guo, Z. Bian, C. Yu, X. Chen, et al. 2020. Solid fuels for cooking and tobacco use and risk of major chronic liver disease mortality: A prospective cohort study of 0.5 million Chinese adults. *Int. J. Epidemiol.* 49 (1):45–55. doi: 10.1093/ije/dyz216.

Chen, H., J. M. Samet, P. A. Bromberg, and H. Tong. 2021. Cardiovascular health impacts of wildfire smoke exposure. *Part. Fibre Toxicol.* 18 (1):2. doi: 10.1186/s12989-020-00394-8.

Chen, J., C. Li, Z. Ristovski, A. Milic, Y. Gu, M. S. Islam, S. Wang, J. Hao, H. Zhang, C. He, et al. 2017. A review of biomass burning: Emissions and impacts on air quality, health and climate in china. *Sci. Total Environ.* 579:1000–1034. doi: 10.1016/j.scitotenv.2016.11.025.

Cherry, N., N. Broznitsky, M. Fedun, and T. Zadunayski. 2022. Respiratory tract and eye symptoms in wildland firefighters in two Canadian provinces: Impact of discretionary use of an n95 mask during successive rotations. *Int. J. Environ. Res. Public Health.* 19 (20):13658. doi: 10.3390/ijerph192013658.

Chowdhury, S., A. Pozzer, A. Haines, K. Klingmüller, T. Münzel, P. Paasonen, A. Sharma, C. Venkataraman, and J. Lelieveld. 2022. Global health burden of ambient pm2.5 and the contribution of anthropogenic black carbon and organic aerosols. *Environ. Int.* 159:107020. doi: 10.1016/j.envint.2021.107020.

Christensen, G. M., C. Rowcliffe, J. Chen, A. Vanker, N. Koen, M. J. Jones, N. Gladish, N. Hoffman, K. A. Donald, C. J. Wedderburn, et al. 2022. *In-utero* exposure to indoor air pollution or tobacco smoke and cognitive development in a South African birth cohort study. *Sci. Total Environ.* 834:155394. doi: 10.1016/j.scitotenv.2022.155394.

Croft, D. P., S. J. Cameron, C. N. Morrell, C. J. Lowenstein, F. Ling, W. Zareba, P. K. Hopke, M. J. Utell, S. W. Thurston, K. Thevenet-Morrison, et al. 2017. Associations between ambient wood smoke and other particulate pollutants and biomarkers of systemic inflammation, coagulation and thrombosis in cardiac patients. *Environ. Res.* 154:352–361. doi: 10.1016/j.envres.2017.01.027.

Danielsen, P. H., S. Loft, N. R. Jacobsen, K. A. Jensen, H. Autrup, J. L. Ravanat, H. Wallin, and P. Møller. 2010. Oxidative stress, inflammation, and DNA damage in rats after intratracheal instillation or oral exposure to ambient

air and wood smoke particulate matter. *Toxicol. Sci.* 118 (2):574–585. doi: 10.1093/toxsci/kfq290.

de Miranda, R. M., F. Lopes, N. É. do Rosário, M. A. Yamasoe, E. Landulfo, and M. de Fatima Andrade. 2016. The relationship between aerosol particles chemical composition and optical properties to identify the biomass burning contribution to fine particles concentration: A case study for São Paulo city, Brazil. *Environ. Monit. Assess.* 189 (1):6. doi: 10.1007/s10661-016-5659-7.

de Oliveira Alves, N., A. T. Vessoni, A. Quinet, R. S. Fortunato, G. S. Kajitani, M. S. Peixoto, S. d S. Hacon, P. Artaxo, P. Saldiva, C. F. M. Menck, et al. 2017. Biomass burning in the Amazon region causes DNA damage and cell death in human lung cells. *Sci. Rep.* 7 (1):10937. doi: 10.1038/s41598-017-11024-3.

DeFlorio-Barker, S., J. Crooks, J. Reyes, and A. G. Rappold. 2019. Cardiopulmonary effects of fine particulate matter exposure among older adults, during wildfire and non-wildfire periods, in the United States 2008–2010. *Environ. Health Perspect.* 127 (3):37006. doi: 10.1289/ehp3860.

do Nascimento, R. d K. S., J. S. Carvalho, R. R. Miranda, M. A. Lima, F. V. Rocha, V. Zucolotto, I. Lynch, and R. C. Urban. 2023. *In vitro* toxicity and lung cancer risk: Atmospheric particulate matter from a city in southeastern Brazil impacted by biomass burning. *Chemosphere.* 338:139484. doi: 10.1016/j.chemosphere.2023.139484.

Drventić, I., M. Glumac, I. Carev, and A. Kroflič. 2023. Seasonality of polyaromatic hydrocarbons (pahs) and their derivatives in pm2.5 from Ljubljana, combustion aerosol source apportionment, and cytotoxicity of selected nitrated polyaromatic hydrocarbons (npahs). *Toxics* 11 (6):518. doi: 10.3390/toxics11060518.

Dubick, M. A., S. C. Carden, B. S. Jordan, P. C. Langlinais, and D. W. Mozingo. 2002. Indices of antioxidant status in rats subjected to wood smoke inhalation and/or thermal injury. *Toxicology* 176 (1–2):145–157. doi: 10.1016/s0300-483x(02)00132-4.

Efriza, E., S. H. Alshahrani, A. O. Zekiy, G. R. L. Al-Awsi, S. K. Sharma, A. A. Ramírez-Coronel, N. Shakeel, Y. Riadi, Z. Aminov, R. S. Zabibah, et al. 2023. Exposure to polycyclic aromatic hydrocarbons and liver function: A systematic review of observational studies. *Air Qual. Atmos. Health* 16 (5):1079–1088. doi: 10.1007/s11869-023-01324-1.

Fan, X., M. Li, T. Cao, C. Cheng, F. Li, Y. Xie, S. Wei, J. Song, and P. A. Peng. 2018. Optical properties and oxidative potential of water- and alkaline-soluble brown carbon in smoke particles emitted from laboratory simulated biomass burning. *Atmos. Environ.* 194:48–57. doi: 10.1016/j.atmosenv.2018.09.025.

Fang, T., Y.-K. Huang, J. Wei, J. E. Monterrosa Mena, P. S. J. Lakey, M. T. Kleinman, M. A. Digman, and M. Shiraiwa. 2022. Superoxide release by macrophages through NADPH oxidase activation dominating chemistry by isoprene secondary organic aerosols and quinones to cause oxidative damage on membranes. *Environ. Sci. Technol.* 56 (23): 17029–17038. doi: 10.1021/acs.est.2c03987.

Fang, Z., A. Lai, C. Dongmei, L. Chunlin, R. Carmieli, J. Chen, X. Wang, Y. Rudich. 2024. Secondary organic aerosol generated from biomass burning emitted phenolic compounds: Oxidative potential, reactive oxygen species, and cytotoxicity. *Environ. Sci. Technol.* 58(19): 8194–8206. doi: 10.1021/acs.est.3c09903.

Fang, Z., C. Li, Q. He, H. Czech, T. Gröger, J. Zeng, H. Fang, S. Xiao, M. Pardo, E. Hartner, et al. 2021. Secondary organic aerosols produced from photochemical oxidation of secondarily evaporated biomass burning organic gases: Chemical composition, toxicity, optical properties, and climate effect. *Environ. Int.* 157:106801. doi: 10.1016/j.envint.2021.106801.

Farahani, V. J., A. Altuwayjiri, M. Pirhadi, V. Verma, A. A. Ruprecht, E. Diapouli, K. Eleftheriadis, and C. Sioutas. 2022. The oxidative potential of particulate matter (pm) in different regions around the world and its relation to air pollution sources. *Environ. Sci. Atmos.* 2 (5):1076–1086. doi: 10.1039/D2EA00043A.

Flores-Ramírez, R., M. Ortega-Romero, O. Christophe-Barbier, J. G. Meléndez-Marmolejo, M. Rodriguez-Aguilar, H. A. Lee-Rangel, and L. Díaz de León-Martínez. 2021. Exposure to polycyclic aromatic hydrocarbon mixtures and early kidney damage in Mexican indigenous population. *Environ. Sci. Pollut. Res. Int.* 28 (18):23060–23072. doi: 10.1007/s11356-021-12388-w.

Fuller, R., P. J. Landrigan, K. Balakrishnan, G. Bathan, S. Bose-O'Reilly, M. Brauer, J. Caravanos, T. Chiles, A. Cohen, L. Corra, et al. 2022. Pollution and health: A progress update. *Lancet. Planet. Health.* 6 (6):e535–e547. doi: 10.1016/S2542-5196(22)00090-0.

Greven, F. E., J. M. Rooyackers, H. A. M. Kerstjens, and D. J. Heederik. 2011. Respiratory symptoms in firefighters. *Am. J. Ind. Med.* 54 (5):350–355. doi: 10.1002/ajim.20929.

Haghani, A., T. E. Morgan, H. J. Forman, and C. E. Finch. 2020. Air pollution neurotoxicity in the adult brain: Emerging concepts from experimental findings. *J. Alzheimers. Dis.* 76 (3):773–797. doi. doi: 10.3233/JAD-200377.

Hahad, O., J. Lelieveld, F. Birklein, K. Lieb, A. Daiber, and T. Münzel. 2020. Ambient air pollution increases the risk of cerebrovascular and neuropsychiatric disorders through induction of inflammation and oxidative stress. *Int. J. Mol. Sci.* 21 (12):4306. doi: 10.3390/ijms21124306.

Hakimzadeh, M., E. Soleimanian, A. Mousavi, A. Borgini, C. De Marco, A. A. Ruprecht, and C. Sioutas. 2020. The impact of biomass burning on the oxidative potential of pm2.5 in the metropolitan area of Milan. *Atmos. Environ.* 224:117328. doi: 10.1016/j.atmosenv.2020.117328.

Hartikainen, A., P. Tiitta, M. Ihalainen, P. Yli-Pirilä, J. Orasche, H. Czech, M. Kortelainen, H. Lamberg, H. Suhonen, H. Koponen, et al. 2020. Photochemical transformation of residential wood combustion emissions: Dependence of organic aerosol composition on oh exposure. *Atmos. Chem. Phys.* 20 (11):6357–6378. doi: 10.5194/acp-20-6357-2020.

Hu, Z., Y. Li, Y. Yang, W. Yu, W. Xie, G. Song, Y. Qian, and Z. Song. 2021. Serum lipids mediate the relationship of multiple polyaromatic hydrocarbons on non-alcoholic fatty liver disease: A population-based study. *Sci. Total Environ.* 780:146563. doi: 10.1016/j.scitotenv.2021.146563.

Huang, J., P. Zhang, Q. An, L. He, and L. Wang. 2022. New insights into the treatment mechanisms of vitamin d on pm2.5-induced toxicity and inflammation in mouse renal

tubular epithelial cells. *Int. Immunopharmacol.* 108: 108747. doi: 10.1016/j.intimp.2022.108747.

Huang, X., X. Shi, J. Zhou, S. Li, L. Zhang, H. Zhao, X. Kuang, and J. Li. 2020. The activation of antioxidant and apoptosis pathways involved in damage of human proximal tubule epithelial cells by pm2.5 exposure. *Environ. Sci. Eur.* 32 (1):2–13. doi: 10.1186/s12302-019-0284-z.

Jiang, H., M. Jang, T. Sabo-Attwood, and S. E. Robinson. 2016. Oxidative potential of secondary organic aerosols produced from photooxidation of different hydrocarbons using outdoor chamber under ambient sunlight. *Atmos. Environ.* 131:382–389. doi: 10.1016/j.atmosenv.2016.02.016.

Jiang, Y., J. Sun, C. Xiong, H. Liu, Y. Li, X. Wang, and Z. Nie. 2021. Mass spectrometry imaging reveals in situ behaviors of multiple components in aerosol particles. *Angew. Chem. Int. Ed. Engl.* 60 (43):23225–23231. doi: 10.1002/anie.202103874.

Jin, W., S. Su, B. Wang, X. Zhu, Y. Chen, G. Shen, J. Liu, H. Cheng, X. Wang, S. Wu, et al. 2016. Properties and cellular effects of particulate matter from direct emissions and ambient sources. *J. Environ. Sci. Health.* 51 (12): 1075–1083. doi: 10.1080/10934529.2016.1198632.

Johnston, H. J., W. Mueller, S. Steinle, S. Vardoulakis, K. Tantrakarnapa, M. Loh, and J. W. Cherrie. 2019. How harmful is particulate matter emitted from biomass burning? A Thailand perspective. *Curr. Pollution Rep.* 5 (4): 353–377. doi: 10.1007/s40726-019-00125-4.

Kamal, R., A. K. Srivastava, C. N. Kesavachandran, V. Bihari, and A. Singh. 2022. Chronic obstructive pulmonary disease (COPD) in women due to indoor biomass burning: A meta analysis. *Int. J. Environ. Health Res.* 32 (6):1403–1417. doi: 10.1080/09603123.2021.1887460.

Kanashova, T., O. Sippula, S. Oeder, T. Streibel, J. Passig, H. Czech, T. Kaoma, S. C. Sapcariu, M. Dilger, H.-R. Paur, et al. 2018. Emissions from a modern log wood masonry heater and wood pellet boiler: Composition and biological impact on air-liquid interface exposed human lung cancer cells. *JMCM* 1:23–35. doi: 10.31083/j.jmcm.2018.01.004.

Karanasiou, A., A. Alastuey, F. Amato, M. Renzi, M. Stafoggia, A. Tobias, C. Reche, F. Forastiere, S. Gumy, P. Mudu, et al. 2021. Short-term health effects from outdoor exposure to biomass burning emissions: A review. *Sci. Total Environ.* 781:146739. doi: 10.1016/j.scitotenv.2021.146739.

Kim, Y. H., C. King, T. Krantz, M. M. Hargrove, I. J. George, J. McGee, L. Copeland, M. D. Hays, M. S. Landis, M. Higuchi, et al. 2019. The role of fuel type and combustion phase on the toxicity of biomass smoke following inhalation exposure in mice. *Arch. Toxicol.* 93 (6): 1501–1513. doi: 10.1007/s00204-019-02450-5.

Kim, Y. H., H. Tong, M. Daniels, E. Boykin, Q. T. Krantz, J. McGee, M. Hays, K. Kovalcik, J. A. Dye, and M. I. Gilmour. 2014. Cardiopulmonary toxicity of peat wildfire particulate matter and the predictive utility of precision cut lung slices. *Part. Fibre Toxicol.* 11 (1):29. doi: 10.1186/1743-8977-11-29.

Kjällstrand, J., and G. Petersson. 2001. Phenolic antioxidants in wood smoke. *Sci. Total Environ.* 277 (1–3):69–75. doi: 10.1016/S0048-9697(00)00863-9.

Klimont, Z., K. Kupiainen, C. Heyes, P. Purohit, J. Cofala, P. Rafaj, J. Borken-Kleefeld, and W. Schöpp. 2017. Global anthropogenic emissions of particulate matter including black carbon. *Atmos. Chem. Phys.* 17 (14):8681–8723. doi: 10.5194/acp-17-8681-2017.

Koval, L. E., C. K. Carberry, Y. H. Kim, E. McDermott, H. Hartwell, I. Jaspers, M. I. Gilmour, and J. E. Rager. 2022. Wildfire variable toxicity: Identifying biomass smoke exposure groupings through transcriptomic similarity scoring. *Environ. Sci. Technol.* 56 (23):17131–17142. doi: 10.1021/acs.est.2c06043.

Kramer, A. J., W. Rattanavaraha, Z. Zhang, A. Gold, J. D. Surratt, and Y.-H. Lin. 2016. Assessing the oxidative potential of isoprene-derived epoxides and secondary organic aerosol. *Atmos. Environ.* 130:211–218. doi: 10.1016/j.atmosenv.2015.10.018.

Kreyling, W. G., W. Möller, U. Holzwarth, S. Hirn, A. Wenk, C. Schleh, M. Schäffler, N. Haberl, N. Gibson, and J. C. Schittny. 2018. Age-dependent rat lung deposition patterns of inhaled 20 nanometer gold nanoparticles and their quantitative biokinetics in adult rats. *ACS Nano*. 12 (8):7771–7790. doi: 10.1021/acsnano.8b01826.

Lai, A., J. Baumgartner, J. J. Schauer, Y. Rudich, and M. Pardo. 2021. Cytotoxicity and chemical composition of women's personal pm2.5 exposures from rural china. *Environ. Sci. Atmos.* 1 (6):359–371. doi: 10.1039/D1EA00022E.

Lelieveld, J., J. S. Evans, M. Fnais, D. Giannadaki, and A. Pozzer. 2015. The contribution of outdoor air pollution sources to premature mortality on a global scale. *Nature.* 525 (7569):367–371. doi: 10.1038/nature15371.

Li, C., D. Calderon-Arrieta, M. Pardo, D. Cai, A. Laskin, J. Chen, and Y. Rudich. 2023. Atmospheric aging modifies the redox potential and toxicity of humic-like substances (hulis) from biomass burning. *Environ. Sci. Atmos.* 3 (12):1791–1804. doi: 10.1039/D3EA00104K.

Li, C., Z. Fang, H. Czech, E. Schneider, C. P. Rüger, M. Pardo, R. Zimmermann, J. Chen, A. Laskin, and Y. Rudich. 2022a. pH modifies the oxidative potential and peroxide content of biomass burning hulis under dark aging. *Sci. Total Environ.* 834:155365. doi: 10.1016/j.scitotenv.2022.155365.

Li, C., M. V. Misovich, M. Pardo, Z. Fang, A. Laskin, J. Chen, and Y. Rudich. 2022. Secondary organic aerosol formation from atmospheric reactions of anisole and associated health effects. *Chemosphere* 308 (Pt 2):136421. doi: 10.1016/j.chemosphere.2022.136421.

Li, J., J. Li, G. Wang, K. F. Ho, W. Dai, T. Zhang, Q. Wang, C. Wu, L. Li, L. Li, et al. 2021. Effects of atmospheric aging processes on in vitro induced oxidative stress and chemical composition of biomass burning aerosols. *J. Hazard. Mater.* 401:123750. doi: 10.1016/j.jhazmat.2020.123750.

Li, R., X. Kou, L. Xie, F. Cheng, and H. Geng. 2015. Effects of ambient pm2.5 on pathological injury, inflammation, oxidative stress, metabolic enzyme activity, and expression of c-fos and c-jun in lungs of rats. *Environ. Sci. Pollut. Res. Int.* 22 (24):20167–20176. doi: 10.1007/s11356-015-5222-z.

Li, W., P. Ge, M. Chen, J. Tang, M. Cao, Y. Cui, K. Hu, and D. Nie. 2021b. Tracers from biomass burning emissions and identification of biomass burning. *Atmosphere* 12 (11):1401. doi: 10.3390/atmos12111401.

Lim, C. Y., D. H. Hagan, M. M. Coggon, A. R. Koss, K. Sekimoto, J. de Gouw, C. Warneke, C. D. Cappa, and

J. H. Kroll. 2019. Secondary organic aerosol formation from the laboratory oxidation of biomass burning emissions. *Atmos. Chem. Phys.* 19 (19):12797–12809. doi: 10.5194/acp-19-12797-2019.

Lima de Albuquerque, Y., E. Berger, C. Li, M. Pardo, C. George, Y. Rudich, and A. Géloën. 2021. The toxic effect of water-soluble particulate pollutants from biomass burning on alveolar lung cells. *Atmosphere* 12 (8):1023. doi: 10.3390/atmos12081023.

Liu, J. C., G. Pereira, S. A. Uhl, M. A. Bravo, and M. L. Bell. 2015. A systematic review of the physical health impacts from non-occupational exposure to wildfire smoke. *Environ. Res.* 136:120–32. doi: 10.1016/j.envres.2014.10.015.

Liu, Q., J. Baumgartner, Y. Zhang, Y. Liu, Y. Sun, and M. Zhang. 2014. Oxidative potential and inflammatory impacts of source apportioned ambient air pollution in Beijing. *Environ. Sci. Technol.* 48 (21):12920–9. doi: 10.1021/es5029876.

Majewska, M., F. Khan, I. S. Pieta, A. Wróblewska, R. Szmigielski, and P. Pieta. 2021. Toxicity of selected airborne nitrophenols on eukaryotic cell membrane models. *Chemosphere* 266:128996. doi: 10.1016/j.chemosphere.2020.128996.

Marchetti, S., E. Longhin, R. Bengalli, P. Avino, L. Stabile, G. Buonanno, A. Colombo, M. Camatini, and P. Mantecca. 2019. *In vitro* lung toxicity of indoor pm10 from a stove fueled with different biomasses. *Sci. Total Environ.* 649: 1422–33. doi: 10.1016/j.scitotenv.2018.08.249.

Marchetti, S., S. Mollerup, K. B. Gutzkow, C. Rizzi, T. Skuland, M. Refsnes, A. Colombo, J. Øvrevik, P. Mantecca, and J. A. Holme. 2021. Biological effects of combustion-derived particles from different biomass sources on human bronchial epithelial cells. *Toxicol. In Vitro.* 75:105190. doi: 10.1016/j.tiv.2021.105190.

Matamba, T., A. Tahmasebi, S. K. Rish, and J. Yu. 2021. Understanding the enhanced production of poly-aromatic hydrocarbons during the pyrolysis of lignocellulosic biomass components under pressurized entrained-flow conditions. *Fuel Process. Technol.* 213:106645. doi: 10.1016/j.fuproc.2020.106645.

McWhinney, R. D., S. Zhou, and J. P. D. Abbatt. 2013. Naphthalene Soa: Redox activity and naphthoquinone gas–particle partitioning. *Atmos. Chem. Phys.* 13 (19): 9731–44. doi: 10.5194/acp-13-9731-2013.

Memon, T. A., N. D. Nguyen, K. L. Burrell, A. F. Scott, M. Almestica-Roberts, E. Rapp, C. E. Deering-Rice, and C. A. Reilly. 2020. Wood smoke particles stimulate MUC5AC overproduction by human bronchial epithelial cells through TRPA1 and EGFR signaling. *Toxicol. Sci.* 174 (2):278–90. doi: 10.1093/toxsci/kfaa006.

Merk, R., K. Heßelbach, A. Osipova, D. Popadić, W. Schmidt-Heck, G.-J. Kim, S. Günther, A. G. Piñeres, I. Merfort, and M. Humar. 2020. Particulate matter (pm2.5) from biomass combustion induces an anti-oxidative response and cancer drug resistance in human bronchial epithelial beas-2b cells. *Int. J. Environ. Res. Public Health.* 17 (21):8193. doi: 10.3390/ijerph17218193.

Miersch, T., H. Czech, A. Hartikainen, M. Ihalainen, J. Orasche, G. Abbaszade, J. Tissari, T. Streibel, J. Jokiniemi, O. Sippula, et al. 2019. Impact of photochemical ageing on polycyclic aromatic hydrocarbons (PAH) and oxygenated PAH (oxy-PAH/OH-PAH) in logwood stove emissions. *Sci. Total Environ.* 686:382–392. doi: 10.1016/j.scitotenv.2019.05.412.

Migliaccio, C. T., M. A. Bergauff, C. P. Palmer, F. Jessop, C. W. Noonan, and T. J. Ward. 2009. Urinary levoglucosan as a biomarker of wood smoke exposure: Observations in a mouse model and in children. *Environ. Health Perspect.* 117 (1):74–79. doi: 10.1289/ehp.11378.

Milani, C., F. Farina, L. Botto, L. Massimino, E. Lonati, E. Donzelli, E. Ballarini, L. Crippa, P. Marmiroli, A. Bulbarelli, et al. 2020. Systemic exposure to air pollution induces oxidative stress and inflammation in mouse brain, contributing to neurodegeneration onset. *Int. J. Mol. Sci.* 21 (10):3699. doi: 10.3390/ijms21103699.

Mondal, N. K., H. Saha, B. Mukherjee, N. Tyagi, and M. R. Ray. 2018. Inflammation, oxidative stress, and higher expression levels of NRF2 and NQO1 proteins in the airways of women chronically exposed to biomass fuel smoke. *Mol. Cell. Biochem.* 447 (1–2):63–76. doi: 10.1007/s11010-018-3293-0.

Morgan, Z. E. M., M. J. Bailey, D. I. Trifonova, N. C. Naik, W. B. Patterson, F. W. Lurmann, H. H. Chang, B. S. Peterson, M. I. Goran, and T. L. Alderete. 2023. Prenatal exposure to ambient air pollution is associated with neurodevelopmental outcomes at 2 years of age. *Environ. Health.* 22 (1):11. doi: 10.1186/s12940-022-00951-y.

Naeher, L. P., D. B. Barr, O. Adetona, and C. D. Simpson. 2013. Urinary levoglucosan as a biomarker for woodsmoke exposure in wildland firefighters. *Int. J. Occup. Environ. Health.* 19 (4):304–10. doi: 10.1179/2049396713y.0000000037.

Ojha, N., A. Sharma, M. Kumar, I. Girach, T. U. Ansari, S. K. Sharma, N. Singh, A. Pozzer, and S. S. Gunthe. 2020. On the widespread enhancement in fine particulate matter across the Indo-Gangetic plain towards winter. *Sci. Rep.* 10 (1):5862. doi: 10.1038/s41598-020-62710-8.

Olasehinde, T. A., and A. O. Olaniran. 2022. Neurotoxicity of polycyclic aromatic hydrocarbons: A systematic mapping and review of neuropathological mechanisms. *Toxics* 10 (8):417. doi: 10.3390/toxics10080417.

Oliveira, M., K. Slezakova, C. Delerue-Matos, M. C. Pereira, and S. Morais. 2019. Children environmental exposure to particulate matter and polycyclic aromatic hydrocarbons and biomonitoring in school environments: A review on indoor and outdoor exposure levels, major sources and health impacts. *Environ. Int.* 124:180–204. doi: 10.1016/j.envint.2018.12.052.

Olsen, Y., J. K. Nøjgaard, H. R. Olesen, J. Brandt, T. Sigsgaard, S. C. Pryor, T. Ancelet, M. d M. Viana, X. Querol, and O. Hertel. 2020. Emissions and source allocation of carbonaceous air pollutants from wood stoves in developed countries: A review. *Atmos. Pollut. Res.* 11 (2):234–251. doi: 10.1016/j.apr.2019.10.007.

Paoin, K., K. Ueda, P. Vathesatogkit, T. Ingviya, S. Buya, R. Dejchanchaiwong, A. Phosri, X. T. Seposo, C. Kitiyakara, N. Thongmung, et al. 2022. Long-term air pollution exposure and decreased kidney function: A longitudinal cohort study in Bangkok metropolitan region, Thailand from 2002 to 2012. *Chemosphere* 287 (Pt 1):132117. doi: 10.1016/j.chemosphere.2021.132117.

Pardo, M., C. Li, Z. Fang, S. Levin-Zaidman, N. Dezorella, H. Czech, P. Martens, U. Käfer, T. Gröger, C. P. Rüger, et al. 2021. Toxicity of water- and organic-soluble wood

tar fractions from biomass burning in lung epithelial cells. *Chem. Res. Toxicol.* 34 (6):1588–603. doi: 10.1021/acs.chemrestox.1c00020.

Pardo, M., C. Li, Q. He, S. Levin-Zaidman, M. Tsoory, Q. Yu, X. Wang, and Y. Rudich. 2020. Mechanisms of lung toxicity induced by biomass burning aerosols. *Part. Fibre Toxicol.* 17 (1):4. doi: 10.1186/s12989-020-0337-x.

Pardo, M., C. Li, A. Jabali, L. M. Petrick, Z. Ben-Ari, and Y. Rudich. 2023. Toxicity mechanisms of biomass burning aerosols in *in vitro* hepatic steatosis models. *Sci. Total Environ.* 905:166988. doi: 10.1016/j.scitotenv.2023.166988.

Pardo, M., X. Qiu, R. Zimmermann, and Y. Rudich. 2020. Particulate matter toxicity is Nrf2 and mitochondria dependent: The roles of metals and polycyclic aromatic hydrocarbons. *Chem. Res. Toxicol.* 33 (5):1110–1120. doi: 10.1021/acs.chemrestox.0c00007.

Pardo, M., F. Xu, X. Qiu, T. Zhu, and Y. Rudich. 2018. Seasonal variations in fine particle composition from Beijing prompt oxidative stress response in mouse lung and liver. *Sci. Total Environ.* 626:147–155. doi: 10.1016/j.scitotenv.2018.01.017.

Patel, A., R. Satish, and N. Rastogi. 2021. Remarkably high oxidative potential of atmospheric PM2.5 coming from a large-scale paddy-residue burning over the northwestern Indo-Gangetic plain. *ACS Earth Space Chem.* 5 (9):2442–2452. doi: 10.1021/acsearthspacechem.1c00125.

Patel, A. B., S. Shaikh, K. R. Jain, C. Desai, and D. Madamwar. 2020. Polycyclic aromatic hydrocarbons: Sources, toxicity, and remediation approaches. *Front. Microbiol.* 11:562813. doi: 10.3389/fmicb.2020.562813.

Pathak, U., N. C. Gupta, and J. C. Suri. 2020. Risk of COPD due to indoor air pollution from biomass cooking fuel: A systematic review and meta-analysis. *Int. J. Environ. Health Res.* 30 (1):75–88. doi: 10.1080/09603123.2019.1575951.

Ponczek, M., M. A. Franco, S. Carbone, L. V. Rizzo, D. Monteiro dos Santos, F. G. Morais, A. Duarte, H. M. J. Barbosa, and P. Artaxo. 2022. Linking the chemical composition and optical properties of biomass burning aerosols in Amazonia. *Environ. Sci: Atmos.* 2 (2):252–269. doi: 10.1039/D1EA00055A.

Qi, Z., Y. Song, Q. Ding, X. Liao, R. Li, G. Liu, S. Tsang, and Z. Cai. 2019. Water soluble and insoluble components of pm2.5 and their functional cardiotoxicities on neonatal rat cardiomyocytes *in vitro*. *Ecotoxicol. Environ. Saf.* 168:378–87. doi: 10.1016/j.ecoenv.2018.10.107.

Rahman, H. H., D. Niemann, and S. H. Munson-McGee. 2022. Association of chronic kidney disease with exposure to polycyclic aromatic hydrocarbons in the US population. *Environ. Sci. Pollut. Res. Int.* 29 (16):24024–34. doi: 10.1007/s11356-021-17479-2.

Ranathunga, N., P. Perera, S. Nandasena, N. Sathiakumar, A. Kasturiratne, and R. Wickremasinghe. 2022. Effects of indoor air pollution on the development of children under five years of age in Sri Lanka. *Atmosphere.* 13 (4):509. doi: 10.3390/atmos13040509.

Reid, C. E., M. Brauer, F. H. Johnston, M. Jerrett, J. R. Balmes, and C. T. Elliott. 2016. Critical review of health impacts of wildfire smoke exposure. *Environ. Health Perspect.* 124 (9):1334–43. doi: 10.1289/ehp.1409277.

Reid, J. S., T. F. Eck, S. A. Christopher, R. Koppmann, O. Dubovik, D. P. Eleuterio, B. N. Holben, E. A. Reid, and J. Zhang. 2005. A review of biomass burning emissions part III: Intensive optical properties of biomass burning particles. *Atmos. Chem. Phys.* 5 (3):827–49. doi: 10.5194/acp-5-827-2005.

Roomaney, R. A., C. Y. Wright, E. Cairncross, N. Abdelatif, A. Cois, E. B. Turawa, O. F. Owotiwon, I. Neethling, B. Nojilana, R. Pacella, et al. 2022. Estimating the burden of disease attributable to household air pollution from cooking with solid fuels in South Africa for 2000, 2006 and 2012. *S. Afr. Med. J.* 112 (8b):718–728. doi: 10.7196/SAMJ.2022.v112i8b.16474.

Ruan, F., L. Wu, H. Yin, L. Fang, C. Tang, S. Huang, L. Fang, Z. Zuo, C. He, and J. Huang. 2021. Long-term exposure to environmental level of phenanthrene causes adaptive immune response and fibrosis in mouse kidneys. *Environ. Pollut.* 283:117028. doi: 10.1016/j.envpol.2021.117028.

Scaramboni, C., M. L. Arruda Moura Campos, D. Junqueira Dorta, D. Palma de Oliveira, S. R. Batistuzzo de Medeiros, M. F. de Oliveira Galvão, and K. Dreij. 2023. Reactive oxygen species-dependent transient induction of genotoxicity by Retene in human liver HEPG2 cells. *Toxicol. In Vitro.* 91:105628. doi: 10.1016/j.tiv.2023.105628.

Scieszka, D., Y. Jin, S. Noor, E. Barr, M. Garcia, J. Begay, G. Herbert, R. P. Hunter, K. Bhaskar, R. Kumar, et al. 2023. Biomass smoke inhalation promotes neuroinflammatory and metabolomic temporal changes in the hippocampus of female mice. *J. Neuroinflammation.* 20 (1):192. doi: 10.1186/s12974-023-02874-y.

Sheikh, M., H. Poustchi, A. Pourshams, M. Khoshnia, A. Gharavi, M. Zahedi, G. Roshandel, S. G. Sepanlou, A. Fazel, M. Hashemian, et al. 2020. Household fuel use and the risk of gastrointestinal cancers: The golestan cohort study. *Environ. Health Perspect.* 128 (6):67002. doi: 10.1289/ehp5907.

Sigsgaard, T., B. Forsberg, I. Annesi-Maesano, A. Blomberg, A. Bølling, C. Boman, J. Bønløkke, M. Brauer, N. Bruce, M.-E. Héroux, et al. 2015. Health impacts of anthropogenic biomass burning in the developed world. *Eur. Respir. J.* 46 (6):1577–1588. doi: 10.1183/13993003.01865-2014.

Singh, D., D. D. Tassew, J. Nelson, M.-C. G. Chalbot, I. G. Kavouras, Y. Tesfaigzi, and P. Demokritou. 2023. Physicochemical and toxicological properties of wood smoke particulate matter as a function of wood species and combustion condition. *J. Hazard. Mater.* 441:129874. doi: 10.1016/j.jhazmat.2022.129874.

Slotkin, T. A., and F. J. Seidler. 2009. Benzo[a]pyrene impairs neurodifferentiation in PC12 cells. *Brain Res. Bull.* 80 (1–2):17–21. doi: 10.1016/j.brainresbull.2009.06.003.

Song, M. K., Y. J. Kim, M. Song, H. S. Choi, Y. K. Park, and J. C. Ryu. 2011. Polycyclic aromatic hydrocarbons induce migration in human hepatocellular carcinoma cells (HEPG2) through reactive oxygen species-mediated p38 MAPK signal transduction. *Cancer Sci.* 102 (9):1636–1644. doi: 10.1111/j.1349-7006.2011.02000.x.

Sosedova, L. M., V. A. Vokina, M. A. Novikov, V. S. Rukavishnikov, E. S. Andreeva, O. M. Zhurba, and A. N. Alekseenko. 2020. Paternal biomass smoke exposure in

rats produces behavioral and cognitive alterations in the offspring. *Toxics.* 9 (1):3. doi: 10.3390/toxics9010003.

Sussan, T. E., V. Ingole, J. H. Kim, S. McCormick, J. Negherbon, J. Fallica, J. Akulian, L. Yarmus, D. Feller-Kopman, M. Wills-Karp, et al. 2014. Source of biomass cooking fuel determines pulmonary response to household air pollution. *Am. J. Respir. Cell Mol. Biol.* 50 (3): 538–548. doi: 10.1165/rcmb.2013-0201OC.

Tuet, W. Y., Y. Chen, L. Xu, S. Fok, D. Gao, R. J. Weber, and N. L. Ng. 2017. Chemical oxidative potential of secondary organic aerosol (SOA) generated from the photooxidation of biogenic and anthropogenic volatile organic compounds. *Atmos. Chem. Phys.* 17 (2):839–853. doi: 10.5194/acp-17-839-2017.

Tuet, W. Y., F. Liu, N. de Oliveira Alves, S. Fok, P. Artaxo, P. Vasconcellos, J. A. Champion, and N. L. Ng. 2019. Chemical oxidative potential and cellular oxidative stress from open biomass burning aerosol. *Environ. Sci. Technol. Lett.* 6 (3):126–132. doi: 10.1021/acs.estlett.9b00060.

Unosson, J., A. Blomberg, T. Sandström, A. Muala, C. Boman, R. Nyström, R. Westerholm, N. L. Mills, D. E. Newby, J. P. Langrish, et al. 2013. Exposure to wood smoke increases arterial stiffness and decreases heart rate variability in humans. *Part. Fibre Toxicol.* 10 (1):20. doi: 10.1186/1743-8977-10-20.

U.S. EPA. 2019. Integrated Science Assessment (ISA) for Particulate Matter (Final Report, Dec 2019). EPA/600/R-19/188, U.S. Environmental Protection Agency, Washington, DC.

Van Vliet, E. D. S., P. L. Kinney, S. Owusu-Agyei, N. W. Schluger, K. A. Ae-Ngibise, R. M. Whyatt, D. W. Jack, O. Agyei, S. N. Chillrud, E. A. Boamah, et al. 2019. Current respiratory symptoms and risk factors in pregnant women cooking with biomass fuels in rural Ghana. *Environ. Int.* 124:533–40. doi: 10.1016/j.envint.2019.01.046.

Vázquez-Gómez, G., M. Karasová, Z. Tylichová, M. Kabátková, A. Hampl, J. Matthews, J. Neča, M. Ciganek, M. Machala, and J. Vondráček. 2022. Aryl hydrocarbon receptor (AHR) limits the inflammatory responses in human lung adenocarcinoma a549 cells via interference with nf-& kappa B signaling. *Cells* 11 (4):707. doi: 10.3390/cells11040707.

Verma, V., Y. Wang, R. El-Afifi, T. Fang, J. Rowland, A. G. Russell, and R. J. Weber. 2015. Fractionating ambient humic-like substances (hulis) for their reactive oxygen species activity – assessing the importance of quinones and atmospheric aging. *Atmos. Environ.* 120:351–359. doi: 10.1016/j.atmosenv.2015.09.010.

Vokina, V. A., L. M. Sosedova, M. A. Novikov, E. A. Titov, E. S. Andreeva, and V. S. Rukavishnikov. 2022. Effects of daily peat smoke exposure on present and next generations. *Toxics* 10 (12):750. doi: 10.3390/toxics10120750.

Vondráček, J., and M. Machala. 2021. The role of metabolism in toxicity of polycyclic aromatic hydrocarbons and their non-genotoxic modes of action. *Curr. Drug Metab.* 22 (8):584–595. doi: 10.2174/1389200221999201125205725.

Walker, E. S., M. L. Clark, B. N. Young, S. Rajkumar, M. L. Benka-Coker, A. M. Bachand, R. D. Brook, T. L. Nelson, J. Volckens, S. J. Reynolds, et al. 2020. Exposure to household air pollution from biomass cookstoves and self-reported symptoms among women in rural Honduras. *Int. J. Environ. Health Res.* 30 (2):160–173. doi: 10.1080/09603123.2019.1579304.

Wang, W., Y. Lin, H. Yang, W. Ling, L. Liu, W. Zhang, D. Lu, Q. Liu, and G. Jiang. 2022. Internal exposure and distribution of airborne fine particles in the human body: Methodology, current understandings, and research needs. *Environ. Sci. Technol.* 56 (11):6857–6869. doi: 10.1021/acs.est.1c07051.

Wang, Y., M. Duong, M. Brauer, S. Rangarajan, A. Dans, F. Lanas, P. Lopez-Jaramillo, T. Puoane, K. Yeates, J. Chifamba, et al. 2023. Household air pollution and adult lung function change, respiratory disease, and mortality across eleven low- and middle-income countries from the pure study. *Environ. Health Perspect.* 131 (4):47015. doi: 10.1289/ehp11179.

Wardoyo, A. Y. P. 2019. Biomass burning; ultrafine particles, concentration, and organ effect. *J. Phys: Conf. Ser.* 1321 (3):032134. doi: 10.1088/1742-6596/1321/3/032134.

Wen, Q., T. Liu, Y. Yu, Y. Zhang, Y. Yang, R. Zheng, L. Li, R. Chen, and S. Wang. 2023. Self-reported primary cooking fuels use and risk of chronic digestive diseases: A prospective cohort study of 0.5 million Chinese adults. *Environ. Health Perspect.* 131 (4):47002. doi: 10.1289/ehp10486.

Wettstein, Z. S., S. Hoshiko, J. Fahimi, R. J. Harrison, W. E. Cascio, and A. G. Rappold. 2018. Cardiovascular and cerebrovascular emergency department visits associated with wildfire smoke exposure in California in 2015. *J. Am. Heart Assoc.* 7 (8):e007492. doi: 10.1161/jaha.117.007492.

Wong, J. P. S., M. Tsagkaraki, I. Tsiodra, N. Mihalopoulos, K. Violaki, M. Kanakidou, J. Sciare, A. Nenes, and R. J. Weber. 2019. Effects of atmospheric processing on the oxidative potential of biomass burning organic aerosols. *Environ. Sci. Technol.* 53 (12):6747–6756. doi: 10.1021/acs.est.9b01034.

Woo, S. H. L., J. C. Liu, X. Yue, L. J. Mickley, and M. L. Bell. 2020. Air pollution from wildfires and human health vulnerability in Alaskan communities under climate change. *Environ. Res. Lett.* 15 (9):094019. doi: 10.1088/1748-9326/ab9270.

Wu, X., J. Lintelmann, S. Klingbeil, J. Li, H. Wang, E. Kuhn, S. Ritter, and R. Zimmermann. 2017. Determination of air pollution-related biomarkers of exposure in urine of travellers between Germany and China using liquid chromatographic and liquid chromatographic-mass spectrometric methods: A pilot study. *Biomarkers* 22 (6):525–536. doi: 10.1080/1354750x.2017.1306753.

Xu, C., Q. Liu, J. Liang, Z. Weng, J. Xu, Z. Jiang, and A. Gu. 2021. Urinary biomarkers of polycyclic aromatic hydrocarbons and their associations with liver function in adolescents. *Environ. Pollut.* 278:116842. doi: 10.1016/j.envpol.2021.116842.

Yang, F., C. Liu, and H. Qian. 2021. Comparison of indoor and outdoor oxidative potential of pm2.5: Pollution levels, temporal patterns, and key constituents. *Environ. Int.* 155:106684. doi: 10.1016/j.envint.2021.106684.

Yoro, K. O., and M. O. Daramola. 2020. Chapter 1 – CO_2 emission sources, greenhouse gases, and the global

warming effect. In *Advances in carbon capture*, eds. M. R. Rahimpour, M. Farsi, and M. A. Makarem, 3–28. Sawston, Cambridge: Woodhead Publishing.

Yuan, C.-S., C.-S. Lai, G.-P. Chang-Chien, Y.-L. Tseng, and F.-J. Cheng. 2022. Kidney damage induced by repeated fine particulate matter exposure: Effects of different components. *Sci. Total Environ.* 847:157528. doi: 10.1016/j.scitotenv.2022.157528.

Yuan, Q., and H. Zhang. 2023. Identification of differentially expressed genes and pathways in beas-2b cells upon long-term exposure to particulate matter (pm(2.5)) from biomass combustion using bioinformatics analysis. *Environ. Health Prev. Med.* 28 (0):51. doi: 10.1265/ehpm.22-00272.

Zhang, Q., H. Ma, J. Li, H. Jiang, W. Chen, C. Wan, B. Jiang, G. Dong, X. Zeng, D. Chen, et al. 2023. Nitroaromatic compounds from secondary nitrate formation and biomass burning are major proinflammatory components in organic aerosols in Guangzhou: A bioassay combining high-resolution mass spectrometry analysis. *Environ. Sci. Technol.* 57 (51):21570–21580. doi: 10.1021/acs.est.3c04983.

Zhu, J., S. Wang, H. Wang, S. Jing, S. Lou, A. Saiz-Lopez, and B. Zhou. 2020. Observationally constrained modeling of atmospheric oxidation capacity and photochemical reactivity in Shanghai, china. *Atmos. Chem. Phys.* 20 (3): 1217–1232. doi: 10.5194/acp-20-1217-2020.

Zhu, Z., Z. Chen, T. Sakurai, H. Chiba, and S.-P. Hui. 2023. Adverse effects of chrysene on human hepatocytes via inducement of oxidative stress and dysregulation of xenobiotic metabolism. *Polycyclic Aromat. Compd.* 43 (2): 1030–1041. doi: 10.1080/10406638.2021.2023200.

Zundel, C. G., P. Ryan, C. Brokamp, A. Heeter, Y. Huang, J. R. Strawn, and H. A. Marusak. 2022. Air pollution, depressive and anxiety disorders, and brain effects: A systematic review. *Neurotoxicology* 93:272–300. doi: 10.1016/j.neuro.2022.10.011.