

Perspective

Open Access



Biomass burning enhanced human exposure risks in the context of global warming

ZeZhi Peng, Jian Sun

Department of Environmental Science and Engineering, Xi'an Jiaotong University, Xi'an 710049, Shaanxi, China.

Correspondence to: Prof. Jian Sun, Department of Environmental Science and Engineering, Xi'an Jiaotong University, 28 Xianning West Road, Xi'an 710049, Shaanxi, China. E-mail: sunjian0306@mail.xjtu.edu.cn

How to cite this article: Peng, Z.; Sun, J. Biomass burning enhanced human exposure risks in the context of global warming. *J. Environ. Expo. Assess.* 2025, 4, 3. <https://dx.doi.org/10.20517/jeea.2024.43>

Received: 23 Oct 2024 **First Decision:** 10 Jan 2025 **Revised:** 15 Jan 2025 **Accepted:** 23 Jan 2025 **Published:** 13 Feb 2025

Academic Editor: Stuart Harrad **Copy Editor:** Pei-Yun Wang **Production Editor:** Pei-Yun Wang

Abstract

In the context of global warming, biomass energy is gaining popularity due to its perceived carbon neutrality. However, the pollutants emitted during biomass combustion and the associated human health concerns are often overlooked. The increased frequency of heatwaves and wildfires contributes significantly to carbon dioxide and particulate matter (PM) emissions, posing substantial risks to public health. Furthermore, the greening of high-latitude regions as a result of global warming has increased the availability of biomass for local use. However, incomplete combustion of this biomass can lead to indoor air pollution and heightened health risks. Additionally, more frequent cold waves increase the demand for space heating, leading to increased household biomass burning, which raises exposure risks, especially in low- and middle-income countries. It is, therefore, essential to focus on human exposure risks associated with biomass burning, particularly from pathways not typically emphasized in the context of global warming.

Keywords: Biomass burning, human health, exposure risk, air pollution

Biomass energy, often considered a carbon-neutral alternative to fossil fuels, is becoming increasingly widespread. It is used extensively for thermal energy, electricity generation, and transportation fuels, accounting for approximately 10% of the total primary energy supply worldwide^[1]. However, during both intentional and accidental biomass combustion, numerous by-products [e.g., carbon monoxide (CO),



© The Author(s) 2025. **Open Access** This article is licensed under a Creative Commons Attribution 4.0 International License (<https://creativecommons.org/licenses/by/4.0/>), which permits unrestricted use, sharing, adaptation, distribution and reproduction in any medium or format, for any purpose, even commercially, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license, and indicate if changes were made.



nitrogen oxides (NO_x), volatile organic compounds (VOCs), particulate matter (PM)] are released alongside carbon dioxide (CO₂), with significant environmental and health impact [Table 1]^[2-15]. Certain combustion by-products, such as water vapor, methane, and black carbon, exhibit strong light-absorbing properties and contribute to the greenhouse effect^[16-18]. Conversely, components like sulfates reflect solar radiation and can potentially have a cooling effect^[19]. Despite the known toxic effects of certain combustion products [e.g., polycyclic aromatic hydrocarbons (PAHs)], their impact on human health has not been sufficiently acknowledged. More than 2.3 billion people worldwide still rely on biomass for cooking or heating, leading to over 3.7 million premature deaths annually^[20]. In addition to these direct impacts, pollutants from biomass burning contribute to exposure risks in less obvious ways.

The increased intensity and frequency of summer heatwaves, exacerbated by global warming, have made wildfires a major source of human health threats^[21]. Over the past two decades, more frequent and intense wildfires have generated far more air pollutants than controlled biomass combustion, posing severe threats to human health and the environment. In North America, particularly the western U.S., the frequency of wildfires has increased dramatically, impacting air quality and resulting in significant health consequences, including respiratory issues and hospital admissions^[22]. Similarly, Australia, which faced a devastating fire season during the “Black Summer” of 2019-2020, has also seen an increase in wildfire intensity, leading to severe health impacts due to smoke exposure^[23]. The characteristics of local vegetation, including the presence of grasslands and forests, significantly heighten the risk of wildfires. Furthermore, social factors, particularly in sparsely populated areas, affect disaster management and response capabilities. Inadequate infrastructure development in many regions, especially regarding investments in fire prevention facilities and emergency response systems, further weakens their ability to effectively respond to wildfires. Economic pressures may compel local governments to reduce budgets for disaster prevention and mitigation, exacerbating the frequency and impact of wildfires. In conclusion, economic factors intertwine with natural and social factors, collectively driving the intensification of the global wildfire phenomenon. These regions also suffer from severe air pollution caused by wildfires, which not only degrades outdoor air quality but may also result in elevated concentrations of pollutants infiltrating indoor environments. This phenomenon poses long-term health issues for both urban and rural populations. Between 2001 and 2022, global forest fires resulted in approximately 33.9 billion tons of CO₂ emissions^[24]. The smoke from wildfires offset around 25% of the improvement in fine particulate matter (PM_{2.5}) air quality achieved in the United States from 2000 to 2016^[25]. Studies have shown that PM_{2.5} generated by wildfires exacerbates respiratory and cardiovascular diseases, with potentially greater toxicity than equivalent doses of ambient PM_{2.5}^[26]. For every 10 µg/m³ increase in the daily average PM_{2.5} concentration from wildfires, all-cause mortality increased by 3.1%, cardiovascular mortality by 2.6%, and respiratory mortality by 7.7%^[27].

Biomass burning in the residential sector has also increased. On the one hand, global warming has accelerated vegetation growth in high-latitude regions, resulting in increased biomass availability for local use^[28]. In these regions, biomass is often used in open fires or rudimentary stoves, leading to incomplete combustion and the release of substantial pollutants^[29,30]. CO, one of the pollutants emitted, poses significant health risks. Exposure to CO impairs the oxygen-carrying capacity of red blood cells, leading to symptoms such as dizziness and, in higher concentrations, severe cardiovascular and respiratory consequences^[31,32].

On the other hand, global warming’s extreme weather phenomena also manifest as winter cold waves. During such events, low-income populations often rely on biomass burning for heating^[33]. Crude heating methods, such as three-stone stoves in Africa or Kang beds in rural China, result in low combustion efficiency and high emissions of pollutants, including CO, CO₂, PM, VOCs, and PAHs^[34,35]. These pollutants not only degrade ambient air quality but also significantly impact indoor air quality, increasing health risks

Table 1. Health hazards of gaseous pollutants generated from biomass combustion

Pollutants	Exposure dose	Exposure duration	Toxic effects	Country	Ref.
PM _{2.5}	PM _{2.5} > 20.4 µg/m ³	On the day of death, the day prior to death, and the 4 days prior to death, to wildfire smoke exposure on referent (non-event, or control) days for the same decedent	All ages and age-stratified sub-groups. All-cause mortality increased by 1.0% greater on wildfire smoke days compared to non-wildfire smoke days. Respiratory mortality increased by 9.0%; COPD mortality increased by 14.0%	the United States	[3,4]
	Biomass burning PM _{2.5} from monitoring sites and atmospheric chemistry model. BB affected day when BB-PM _{2.5} > 5 µg/m ³	The percentage difference in cardiopulmonary hospitalizations for a 10 µg/m ³ increase in PM _{2.5} was examined at single-day lags ranging from the day of exposure [lag day 0 (lag 0)] up through 6 days postexposure	Asthma risk was 6.9% on smoke days, and the increased risk was similar on smoke and non-smoke days. During smoke days, the older population is more likely to experience strong, acute respiratory responses such as asthma, bronchitis, and wheezing, compared with non-smoke days	the United States	[5]
	The overall mean PM _{2.5} = 8.8 µg/m ³ , cold season (November-April) PM _{2.5} = 9.8 µg/m ³ , warm season (May-October) PM _{2.5} = 7.2 µg/m ³	The exposure periods of interest in this study included the same day as hospital admission for myocardial infarction (lag 0) and the 3-day mean exposure preceding hospital admissions (including the day of admission)	Each 5 µg/m ³ increase in 3-day mean PM _{2.5} was associated with an increased risk of MI among elderly subjects (≥ 65 years)	British, Columbia and Canada	[6]
CO	70-120 ppm, 200 ppm	Low concentration for 4 h	Exposure to between 70 and 120 ppm CO results in COHb levels between 10% and 20% and is usually asymptomatic. Such an exposure is generally regarded as subclinical. On the other hand, inhaling more than 200 ppm CO results in COHb levels of approximately 30% and causes headaches, dizziness, and impaired judgment	the United States	[7,8]
	35-12,800 ppm	Low concentration for 8 h, high concentration for less than 3 min	35 ppm, headache and dizziness within 6 to 8 h of constant exposure 100 ppm, slight headache in 2 to 3 h 200 ppm, slight headache within 2 to 3 h; loss of judgment 400 ppm, frontal headache within 1 to 2 h 800 ppm, dizziness, nausea, and convulsions within 45 min; insensible within 2 h 1,600 ppm, headache, tachycardia, dizziness, and nausea within 20 min; death in less than 2 h 3,200 ppm, 50% headache, dizziness, and nausea in 5 to 10 min; death within 30 min 6,400 ppm, 60% headache and dizziness in 1 to 2 min; convulsions, respiratory arrest, and death in less than 20 min 12,800 ppm, death in less than 3 min	North America	[9]
NO _x	0.62 ppm to Parkinson's disease, 2.61 ppm to preterm birth, 1.54 mg/m ³ to cardiovascular disease	-	CO was significantly associated with Parkinson's disease, with a pooled RR of 1.574, and also with CVD, with an RR of 1.024	China	[10]
	NO ₂ , the national average concentration was 31 µg/m ³ in 272 cities	The present-day average lags from lag 0 to lag 6, and average lags from lag 0 to lag 0-13	NO ₂ was associated with mortality from all considered cardiorespiratory diseases, including hypertension, coronary heart disease, stroke, and COPD. The magnitude of the association of NO ₂ with total mortality was similar to that with cardiovascular mortality and lower than that with respiratory mortality	China	[11,12]
	NO ₂ maximum average concentration is 13.71-68.44 µg/m ³	-	The reported significant health effects included CVD, childhood asthma, COPD, diabetes mellitus, lung cancer, Parkinson's disease, and preterm birth. In detail, each 10 µg/m ³ increase in NO ₂ was associated with the highest increase in risk for childhood asthma RR (1.134, 95%CI: 1.084-1.186), followed by preterm birth	China	[10]

O ₃	O ₃ , 40.6 ppb to Parkinson's disease; from 51.7 to 94 µg/m ³	-	(RR 1.079, 95%CI: 1.007-1.157), lung cancer (RR 1.055, 95%CI:1.010-1.101), Parkinson's disease (RR 1.030, 95%CI:1.007-1.053), diabetes mellitus (RR 1.019, 95%CI:1.009-1.029), and COPD (RR 1.016, 95%CI: 1.012-1.120)	China	[10]
VOCs	Urinary metabolites of acrolein (CEMA and 3HPMA), 1,3-butadiene (DHBMA and MHBMA3), and crotonaldehyde (HPMMA), VOC metabolite: 88.3 ng/mg of creatinine for CEMA, 148.9 ng/mg for 3HPMA, 195.0 ng/mg for DHBMA, 4.7 ng/mg for MHBMA3, and 107.2 ng/mg for HPMMA diastolic BPs and RHI had a log-normal distribution. The levels of catecholamines and metabolites were gamma distributed	-	When the concentration increased by 10 µg/m ³ , O ₃ was significantly associated with Parkinson's disease, with an RR of 1.067 (95%CI: 1.018-1.118). O ₃ was further significantly associated with preterm birth, with an RR of 1.039 (95%CI: 1.004-1.075)	the United States	[13]
PAHs	Above the average of 597 ng/g	-	Exposure to VOCs acrolein, 1,3-butadiene, and crotonaldehyde is associated with vascular dysfunction. Stratified analysis revealed an increased association with systolic BP in Black participants despite lower levels of urinary 3HPMA. Bayesian kernel machine regression (BKMR) analysis confirmed that 3HPMA was the major metabolite associated with higher BP in the presence of other metabolites. We also found that 3HPMA and DHBMA were associated with decreased endothelial function	the United States	[13]
	Pyrene and BaP, the average BaP concentration was about 30 mg/m ³	Long-term exposure	Investigated placental PAH levels in 80 fetuses or newborns with neural tube defects in China. The results of their study showed that the risk of a defect was 4 to 5 times greater	Republic of Korea	[14]
	Benzo(a) pyrene, the median values of B[a]P daily exposure doses for children, adolescents, adults, and seniors of male were estimated to be 392.42, 511.01, 571.56, and 532.56 ng/d, respectively	(B[a]P) daily exposure	Short-term health effects: eye irritation, nausea, vomiting, diarrhea; long-term health effects: an increased risk of skin, lung, bladder, and gastrointestinal cancers	China	[15]
			Implied significant cancer risk, the median values of ILCR were estimated to be 1.19×10^{-5} , 7.91×10^{-6} , 4.04×10^{-5} , and 8.03×10^{-6} for children, adolescents, adults, and seniors of male, respectively; the median values of ILCRs for all population groups fell within the range of 10^{-6} - 10^{-5} , being higher than the acceptable risk level (10^{-6})		

COPD: Chronic obstructive pulmonary disease; BB: biomass burning; COHb: carboxyhemoglobin; RR: relative risk; CVD: cardiovascular diseases; VOCs: volatile organic compounds; BPs: blood pressures; RHI: reactive hyperemia index; PAHs: polycyclic aromatic hydrocarbons; B[a]P: B[a]P equivalent; ILCR: incremental lifetime cancer risk.

for residents^[36]. Approximately 56% of soot emissions in Europe can be attributed to residential heating, primarily from wood burning^[37]. Studies have shown that cooking with biomass fuel three times a day can raise the 24-hour average indoor PM_{2.5} concentration by as much as 342 µg/m³^[38]. In India, household biomass burning was one of the most significant individual contributors to the disease burden in 2016^[39]. Indoor air pollution has varying health impacts among age and gender groups. For children, the most severe health risk is biomass smoke-induced pneumonia, while for adults, cardiovascular diseases represent the main health burden. Women are disproportionately affected by chronic obstructive pulmonary disease and lung cancer due to prolonged exposure during cooking^[40,41].

In summary, amid the current context of global warming, biomass energy is receiving unprecedented attention. However, the negative health effects of exposure to pollutants from biomass combustion are increasing through pathways that are generally overlooked. For instance, the formation of secondary pollutants, like ozone, occurs due to reactions between NO_x and VOCs, spreading long-range health effects. Harmful gaseous pollutants, such as PAHs, can

transform into solid particulates that, when inhaled, may trigger respiratory and cardiovascular diseases. Additionally, long-term exposure to low doses of various pollutants can lead to cumulative health effects, impacting the immune, respiratory, and cardiovascular systems. Moreover, climate change can indirectly affect ecosystems through altered vegetation patterns, which change pollutant concentrations and distributions, ultimately influencing human and ecological health. Given the disparities in global economic development and changes in vegetation patterns due to global warming, it is crucial to focus on reducing the adverse health impacts of biomass burning. Changes in forests and grasslands may lead to an increase in combustible biomass, subsequently affecting the frequency and scale of biomass burning. Furthermore, climate change can elevate the incidence of forest fires, exacerbating pollutant emissions. While biomass can reduce energy costs, neglecting its associated health and environmental costs may result in hidden economic costs. Additionally, biomass burning in agricultural and forestry regions may influence soil quality and agricultural productivity, ultimately affecting economic sustainability. Regarding the control of the hazards posed by biomass burning to human health, we advocate that in regions with slightly higher economic levels, this can be achieved by promoting cleaner energy sources, such as electricity and solar power, which would reduce reliance on biomass combustion. In contrast, in lower-income regions where communities must depend on biomass fuels for their daily needs, it is essential to enhance public awareness and education concerning the health risks associated with biomass burning. Furthermore, promoting safer practices within these communities can substantially mitigate the use of harmful burning methods. To address gender exposure inequality, it is crucial to specifically target women who are disproportionately affected by the health risks of biomass burning due to their role in cooking. Public health education campaigns should focus on increasing women's awareness of these risks. Additionally, providing women with access to cleaner cooking technologies and alternative energy sources, such as improved cookstoves or solar cookers, can reduce their exposure. Efforts should also be made to ensure that women are included in decision-making processes regarding energy choices and health interventions, empowering them to adopt safer practices in their households. This is particularly important as biomass utilization continues to expand in response to global warming. Efforts should be directed toward mitigating both direct and indirect health effects from pollutants released during combustion and those transformed in the atmosphere.

DECLARATIONS

Authors' contributions

Drafted the initial version and contributed to the revisions of the perspective: Peng, Z.

Conceptualized and revised the perspective: Sun, J.

Availability of data and materials

Not applicable.

Financial support and sponsorship

This work was supported by the Natural Science Foundation of Shaanxi Province, China (No. 2024JC-ZDXM-20; No. 2021ZDLSF05-07).

Conflicts of interest

Sun, J. is an Editorial Board member of *Journal of Environmental Exposure Assessment*. Sun, J. was not involved in any steps of editorial processing, notably including reviewer selection, manuscript handling, and decision making. The other author declared that there are no conflicts of interest.

Ethical approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Copyright

© The Author(s) 2025.

REFERENCES

1. Antar, M.; Lyu, D.; Nazari, M.; Shah, A.; Zhou, X.; Smith, D. L. Biomass for a sustainable bioeconomy: an overview of world biomass production and utilization. *Renew. Sustain. Energy Rev.* **2021**, *139*, 110691. DOI
2. Jiang, K.; Xing, R.; Luo, Z.; et al. Pollutant emissions from biomass burning: a review on emission characteristics, environmental impacts, and research perspectives. *Particuology* **2024**, *85*, 296-309. DOI
3. Karanasiou, A.; Alastuey, A.; Amato, F.; et al. Short-term health effects from outdoor exposure to biomass burning emissions: a review. *Sci. Total. Environ.* **2021**, *781*, 146739. DOI
4. Doubleday, A.; Schulte, J.; Sheppard, L.; et al. Mortality associated with wildfire smoke exposure in Washington state, 2006-2017: a case-crossover study. *Environ. Health.* **2020**, *19*, 4. DOI PubMed PMC
5. DeFlorio-Barker, S.; Crooks, J.; Reyes, J.; Rappold, A. G. 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.* **2019**, *127*, 37006. DOI PubMed PMC
6. Weichenthal, S.; Kulka, R.; Lavigne, E.; et al. Biomass burning as a source of ambient fine particulate air pollution and acute myocardial infarction. *Epidemiology* **2017**, *28*, 329-37. DOI PubMed PMC
7. Winter, P. M. Carbon monoxide poisoning. *JAMA.* **1976**, *236*, 1502. DOI PubMed
8. Levy, R. J. Carbon monoxide pollution and neurodevelopment: a public health concern. *Neurotoxicol. Teratol.* **2015**, *49*, 31-40. DOI PubMed PMC
9. Goldstein, M. Carbon monoxide poisoning. *J. Emerg. Nurs.* **2008**, *34*, 538-42. DOI PubMed
10. Chen, Z.; Liu, N.; Tang, H.; et al. Health effects of exposure to sulfur dioxide, nitrogen dioxide, ozone, and carbon monoxide between 1980 and 2019: a systematic review and meta-analysis. *Indoor. Air.* **2022**, *32*, e13170. DOI PubMed
11. Chen, R.; Yin, P.; Meng, X.; et al. Associations between ambient nitrogen dioxide and daily cause-specific mortality: evidence from 272 Chinese cities. *Epidemiology* **2018**, *29*, 482-9. DOI
12. Wang, L.; Liu, C.; Meng, X.; et al. Associations between short-term exposure to ambient sulfur dioxide and increased cause-specific mortality in 272 Chinese cities. *Environ. Int.* **2018**, *117*, 33-9. DOI
13. McGraw, K. E.; Riggs, D. W.; Rai, S.; et al. Exposure to volatile organic compounds - acrolein, 1,3-butadiene, and crotonaldehyde - is associated with vascular dysfunction. *Environ. Res.* **2021**, *196*, 110903. DOI PubMed PMC
14. Kim, K. H.; Jahan, S. A.; Kabir, E.; Brown, R. J. A review of airborne polycyclic aromatic hydrocarbons (PAHs) and their human health effects. *Environ. Int.* **2013**, *60*, 71-80. DOI PubMed
15. Xia, Z.; Duan, X.; Qiu, W.; et al. Health risk assessment on dietary exposure to polycyclic aromatic hydrocarbons (PAHs) in Taiyuan, China. *Sci. Total. Environ.* **2010**, *408*, 5331-7. DOI
16. Tollefson, J. Soot a major contributor to climate change. *Nature.* **2013**. DOI
17. Hay, W. W. Water vapor - the major greenhouse gas. In *Experimenting on a small planet*. Springer International Publishing: Cham; 2021. pp 477-90. DOI
18. Etminan, M.; Myhre, G.; Highwood, E. J.; Shine, K. P. Radiative forcing of carbon dioxide, methane, and nitrous oxide: a significant revision of the methane radiative forcing. *Geophys. Res. Lett.* **2016**, *43*, 12614-23. DOI
19. Zheng, M.; Song, D.; Zhang, D.; Cao, Y.; Fan, H. Using sulfur isotopes to constrain the sources of sulfate in PM_{2.5} during the winter in Jiaozuo City. *Atmos. Environ.* **2024**, *332*, 120618. DOI
20. IEA. SDG7: data and projections. <https://www.iea.org/reports/sdg7-data-and-projections>. (accessed 2025-02-13).
21. Gibb, K.; Beckman, S.; Vergara, X. P.; Heinzerling, A.; Harrison, R. Extreme heat and occupational health risks. *Annu. Rev. Public. Health.* **2024**, *45*, 315-35. DOI PubMed
22. Sacks, J. D.; Migliaccio, C. T.; Reid, C. E.; Montrose, L. Shifting the conversation on wildland fire smoke exposures: more smoke within and across years requires a new approach to inform public health action. *ACS. EST. Air.* **2025**. DOI
23. Bui, H. X.; Li, Y.; Dommenget, D. Controlling factors of wildfires in Australia and their changes under global warming. *Environ. Res. Lett.* **2024**, *19*, 094030. DOI
24. You, X. Surge in extreme forest fires fuels global emissions. *Nature.* **2023**. DOI PubMed
25. Wildfires are worsening air quality in the United States. *Nature.* **2023**. DOI PubMed
26. Aguilera, R.; Corringham, T.; Gershunov, A.; Benmarhnia, T. Wildfire smoke impacts respiratory health more than fine particles from other sources: observational evidence from Southern California. *Nat. Commun.* **2021**, *12*, 1493. DOI PubMed PMC
27. Ye, T.; Xu, R.; Yue, X.; et al. Short-term exposure to wildfire-related PM_{2.5} increases mortality risks and burdens in Brazil. *Nat. Commun.* **2022**, *13*, 7651. DOI PubMed PMC
28. Park, H.; Jeong, S.; Peñuelas, J. Accelerated rate of vegetation green-up related to warming at northern high latitudes. *Glob. Chang.*

- Biol.* **2020**, *26*, 6190-202. DOI
29. Mishra, S.; Tripathi, S. N.; Kanawade, V. P.; et al. Rapid night-time nanoparticle growth in Delhi driven by biomass-burning emissions. *Nat. Geosci.* **2023**, *16*, 224-30. DOI
 30. Khodmanee, S.; Amnuaylojaroen, T. Impact of biomass burning on ozone, carbon monoxide, and nitrogen dioxide in Northern Thailand. *Front. Environ. Sci.* **2021**, *9*, 641877. DOI
 31. Huzar, T. F.; George, T.; Cross, J. M. Carbon monoxide and cyanide toxicity: etiology, pathophysiology and treatment in inhalation injury. *Expert. Rev. Respir. Med.* **2013**, *7*, 159-70. DOI PubMed
 32. Yadav, I. C.; Devi, N. L. Biomass burning, regional air quality, and climate change. encyclopedia of environmental health. Elsevier; 2019. pp. 386-91. DOI
 33. Raju, S.; Siddharthan, T.; McCormack, M. C. Indoor air pollution and respiratory health. *Clin. Chest. Med.* **2020**, *41*, 825-43. DOI PubMed PMC
 34. Zhai, Z.; Yates, A. P.; Duanmu, L.; Wang, Z. An evaluation and model of the Chinese Kang system to improve indoor thermal comfort in northeast rural China - Part-1: model development. *Renew. Energy.* **2015**, *84*, 3-11. DOI
 35. Bantu, A. A.; Nuwagaba, G.; Kizza, S.; Turinayo, Y. K. Design of an improved cooking stove using high density heated rocks and heat retaining techniques. *J. Renew. Energy.* **2018**, *2018*, 1-9. DOI
 36. Men, Y.; Li, Y.; Luo, Z.; et al. Interpreting highly variable indoor PM_{2.5} in rural North China using machine learning. *Environ. Sci. Technol.* **2023**, *57*, 18183-92. DOI
 37. Heating with wood: a problem for health and climate. 2016. <https://www.clean-heat.eu/en/actions/info-material/download/background-paper-residential-wood-burning-3.html>. (accessed 2025-02-13).
 38. Luo, Z.; Zhang, L.; Li, G.; et al. Evaluating co-emissions into indoor and outdoor air of EC, OC, and BC from in-home biomass burning. *Atmos. Res.* **2021**, *248*, 105247. DOI
 39. Rooney, B.; Zhao, R.; Wang, Y.; et al. Impacts of household sources on air pollution at village and regional scales in India. *Atmos. Chem. Phys.* **2019**, *19*, 7719-42. DOI
 40. Balmes, J. R. Household air pollution from domestic combustion of solid fuels and health. *J. Allergy. Clin. Immunol.* **2019**, *143*, 1979-87. DOI
 41. Ali, M. U.; Yu, Y.; Yousaf, B.; et al. Health impacts of indoor air pollution from household solid fuel on children and women. *J. Hazard. Mater.* **2021**, *416*, 126127. DOI