

Association Between Solid-Fuel Household Air Pollution and Systemic Inflammatory Biomarkers in Adults: A Systematic Review

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Systematic Review

Keywords: household air pollution, solid fuel, biomass smoke, systemic inflammation, C-reactive protein, interleukin-6, tumor necrosis factor-alpha, cardiovascular risk, systematic review

Posted Date: October 1st, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-11206374/v1>

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Additional Declarations: The authors declare no competing interests.

Abstract

Background

Roughly one-third of the world's population still relies on solid fuels (wood, crop residue, dung, coal, charcoal) for cooking and heating, generating household air pollution (HAP) that was linked to an estimated 2.31 million deaths globally in 2019. Systemic inflammation is a proposed intermediate pathway linking HAP exposure to cardiovascular, metabolic, and respiratory disease, but the magnitude and consistency of this association in adults has not been systematically synthesised.

Objective

To systematically review, and quantitatively synthesise evidence on the association between solid-fuel HAP exposure and circulating, blood-based (systemic) inflammatory biomarkers, principally C-reactive protein (CRP), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- α), interleukin-8 (IL-8), and endothelial adhesion molecules among adults. Urinary, oxidative-stress, and other mechanistic or cardiometabolic markers were considered as secondary, supportive evidence only.

Methods

We conducted a structured literature search (PubMed/PMC, ScienceDirect, Wiley Online Library, SpringerLink, and PLOS, supplemented by citation chaining) for studies published in English reporting systemic inflammatory biomarker concentrations in adults exposed to solid-fuel HAP relative to a lower-exposure or clean-fuel comparator. Screening followed PRISMA 2020 principles. Given substantial heterogeneity in biomarker assays, exposure metrics, and reporting formats across studies, we conducted a structured narrative synthesis with a semi-quantitative harvest plot consistent with Synthesis Without Meta-analysis (SWiM) guidance. Risk of bias was assessed using an adapted Newcastle-Ottawa Scale for non-randomised studies.

Results

Nine studies met eligibility criteria (seven cross-sectional, one household-level cross-sectional multi-site study, one randomised controlled trial), conducted across India, China, Peru, Honduras, Nigeria, and South Africa. Combining the seven studies that reported a clear individual-level analytic sample size yields at least 2,362 adults; one further study enrolled 617 households rather than individually sampled adults, and one study's precise analytic sample size was not clearly stated in the available source material. Among the eight primary-tier studies, CRP was the most frequently reported biomarker (6 of 8) and was significantly elevated among solid-fuel-exposed adults in 4 studies, significantly lower in 1 study, and null in 1 study. IL-6 and TNF- α were each reported in 4–5 primary-tier studies, with most showing significant elevation among solid-fuel users. Two studies including the largest ($n = 415$) found no significant associations for most biomarkers, and one study reported an unexpected inverse association for CRP alongside elevated adhesion molecules in the same population.

Conclusions

The preponderance of available evidence supports an association between solid-fuel HAP exposure and higher systemic inflammatory biomarker levels in adults, most consistently for CRP, IL-6, and TNF- α , and this is biologically plausible given experimental and mechanistic data. However, the evidence base is small, geographically clustered, predominantly cross-sectional, and heterogeneous in exposure and outcome measurement, precluding a statistically pooled effect estimate at this time. Larger, standardised, longitudinal studies ideally nested within cookstove-intervention trials are needed to quantify this relationship precisely and to clarify its contribution to the cardiovascular and metabolic burden of HAP.

1. Introduction

Approximately 2.3 billion people worldwide still cook primarily with solid fuels; wood, charcoal, crop residues, animal dung, and coal typically burned in inefficient stoves or open fires with inadequate ventilation [1, 2]. This practice generates household air pollution (HAP) consisting of fine and ultrafine particulate matter (PM_{2.5}, PM_{0.1}), black carbon, carbon monoxide, polycyclic aromatic hydrocarbons, and other combustion by-products, at concentrations that frequently exceed World Health Organization air-quality guidelines by an order of magnitude or more inside the home [3, 4]. According to the Global Burden of Disease (GBD) study, long-term HAP exposure was responsible for approximately 2.31 million deaths and 91.5 million disability-adjusted life-years (DALYs) globally in 2019, with the largest share of this burden concentrated in South Asia

and sub-Saharan Africa [1, 5]. Ischemic heart disease, stroke, chronic obstructive pulmonary disease, lower respiratory infection, lung cancer, and type 2 diabetes are the leading cause-specific contributors to this burden [1, 3].

A substantial share of the cardiovascular and metabolic burden attributed to HAP is thought to be mediated by systemic (as opposed to purely airway-localised) inflammation [6, 7]. Inhaled particulate matter and gaseous co-pollutants can translocate from the alveolar space into the systemic circulation, or trigger pulmonary oxidative stress and cytokine release that secondarily activates hepatic acute-phase protein synthesis and circulating leukocytes [6, 8]. This pathway is well established for ambient (outdoor) particulate air pollution: a 2019 systematic review and meta-analysis of predominantly high-income-country studies concluded that ambient PM exposure is associated with elevated circulating C-reactive protein (CRP), consistent with an activated systemic inflammatory state [9, 10]. Whether and how consistently an analogous relationship holds for solid-fuel HAP, which differs from ambient PM in its composition, its markedly higher peak concentrations during cooking episodes, and the near-continuous, decades-long personal exposure experienced primarily by women who serve as the household's primary cook, has not been systematically appraised [11, 12].

A body of primarily small, single-country studies has examined circulating inflammatory biomarkers; CRP, interleukin-6 (IL-6), tumor necrosis factor- α (TNF- α), interleukin-8 (IL-8), interleukin-1 β , and soluble endothelial adhesion molecules such as intercellular adhesion molecule-1 (ICAM-1) and vascular cell adhesion molecule-1 (VCAM-1) in adults exposed to biomass or coal smoke relative to users of cleaner fuels such as liquefied petroleum gas (LPG) or electricity [13–19]. These studies report divergent findings: several report elevations in pro-inflammatory cytokines associated with solid-fuel use, including in some cases a graded pattern across increasing exposure [13, 14, 20], one well-conducted study reported an unexpected reduction in CRP among biomass-exposed adults alongside elevated adhesion molecules [15], and at least one larger cross-sectional study found little evidence of association for most biomarkers examined [17]. A single published randomised controlled trial has tested whether a clean-fuel intervention (ethanol cookstoves) alters inflammatory biomarker trajectories, with mixed results across the cytokines measured [16]. Based on the literature search conducted for this review (Section 2.3), we identified limited evidence of prior systematic synthesis focused specifically on solid-fuel HAP and systemic inflammatory biomarkers in adults; existing systematic reviews and meta-analyses we are aware of in this general space have instead addressed ambient (outdoor) PM and CRP [9, 10], broader HAP health outcomes such as mortality and respiratory disease [2], or blood pressure specifically [21].

Clarifying this evidence base matters for at least three reasons. First, systemic inflammatory biomarkers, and CRP in particular, are independently predictive of future cardiovascular events and all-cause mortality in clinical populations [22], so establishing whether HAP exposure is reproducibly associated with higher levels of these markers would strengthen the mechanistic case for HAP as a cardiovascular risk factor and could inform risk stratification in HAP-exposed populations. Second, inflammatory biomarkers are comparatively feasible to measure in resource-limited field settings (including via minimally invasive dried-blood-spot sampling), making them attractive intermediate endpoints for evaluating clean-cookstove and clean-fuel interventions, which have otherwise struggled to demonstrate clinical-endpoint benefits within typical trial durations [12, 23]. Third, understanding which biomarkers respond most consistently, and under what exposure conditions, can help target future trials and biomarker panels efficiently.

We therefore conducted a systematic review of the association between solid-fuel HAP exposure and systemic inflammatory biomarkers among adults. Our protocol pre-specified quantitative (random-effects, pooled) synthesis as the preferred approach wherever included studies reported compatible summary statistics for a given biomarker, with a structured narrative synthesis as the pre-specified fallback where pooling was not statistically defensible, the decision rule is detailed in Section 2.7. As described there, the heterogeneity encountered across the eligible literature in exposure definitions, biomarker assays, and reporting formats meant that the narrative-synthesis fallback applied to essentially all biomarkers; we report this transparently and present a structured narrative synthesis supported by a semi-quantitative harvest plot, consistent with current Synthesis Without Meta-analysis (SWiM) guidance [24].

2. Methods

2.1 Protocol and reporting

This review is reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement [25], adapted as appropriate for a review whose final synthesis is narrative rather than meta-analytic. A protocol was drafted specifying eligibility criteria, information sources, and a pre-specified decision rule for choosing between quantitative pooling and narrative synthesis (Section 2.7). This protocol was registered on PROSPERO before study selection and data extraction began.

2.2 Eligibility criteria

Eligibility was defined using a PECOS (Population, Exposure, Comparator, Outcome, Study design) framework.

Population: Adults (≥ 18 years; pregnant adults were included as a pre-specified subgroup given their physiological relevance to HAP research). Studies conducted exclusively in children or in animal/in-vitro models were excluded.

Exposure: Household air pollution from combustion of solid fuels (wood, charcoal, crop residue, animal dung, or coal) for cooking and/or heating, defined either categorically (solid-fuel user vs. clean-fuel user) or continuously (measured personal or kitchen PM_{2.5}, black carbon, or carbon monoxide).

Comparator: A lower-exposure or clean-fuel group (e.g., LPG, electricity, ethanol, natural gas users), a within-person pre/post cookstove-intervention comparison, or a continuous exposure-response gradient.

Outcome: We defined eligibility using two pre-specified tiers. Primary-eligible studies were required to report at least one quantitatively reported, blood-based (serum, plasma, or dried blood spot) systemic inflammatory biomarker including but not limited to CRP, IL-6, TNF- α , IL-8, IL-1 β , IL-12, or serum amyloid A (SAA) or a soluble vascular/endothelial inflammatory marker (ICAM-1, VCAM-1, E-selectin, von Willebrand factor, fibrinogen). We pre-specified this blood-based restriction because urine, exhaled breath, and other non-blood matrices reflect a mixture of local/airway and systemic processes that are not straightforwardly equivalent to circulating biomarker concentrations. Supporting-eligible studies were defined, a priori, as studies that otherwise met all other PECOS criteria but reported only a non-blood systemic-inflammation-related marker (e.g., urinary IL-6/TNF- α) rather than a qualifying blood-based marker; such studies were retained for narrative, contextual discussion only and are explicitly excluded from the primary biomarker-by-biomarker synthesis (Section 3.5) and from Figure 2. Of the nine studies meeting overall eligibility, eight met the primary (blood-based) tier and one Feng et al., whose only cytokine outcomes (IL-6, TNF- α) were measured in urine met only the supporting tier; this distinction is maintained consistently in Table 1, Section 3.4, and Figure 2. Primary-eligible studies additionally reporting non-blood-based markers (e.g., oxidative-stress markers, or cardiometabolic/physiological measures such as HbA1c, blood pressure, or exhaled carbon monoxide) had these additional measures reported in Table 1 for completeness and discussed narrowly as secondary, supportive evidence (Section 3.4.4b), but these did not determine study eligibility and are excluded from Figure 2. Studies reporting exclusively airway/local outcomes (sputum cytology, exhaled nitric oxide) or vascular-structural outcomes (carotid intima-media thickness) without any qualifying primary- or supporting-tier marker, or respiratory symptom/lung-function outcomes only, did not meet either eligibility tier and were excluded entirely, though considered as supporting mechanistic context in the Discussion.

Study design: Cross-sectional, cohort, case-control, and randomised or non-randomised intervention studies reporting original human data. Reviews, editorials, conference abstracts without extractable data, and case reports were excluded. Studies were required to be published in English; no date restriction was applied.

2.3 Information sources and search strategy

We carried out a multi-database search covering search on PubMed/PMC, ScienceDirect, Wiley Online Library, SpringerLink, and PLOS journal indices, supplemented by forward and backward citation chaining from all retrieved articles and relevant prior reviews [2,9,21]. Search terms combined concepts for exposure ("household air pollution", "indoor air pollution", "biomass fuel", "solid fuel", "cookstove", "cooking fuel", "wood smoke") with concepts for outcome ("inflammation", "inflammatory biomarker", "C-reactive protein", "CRP", "interleukin", "tumor necrosis factor", "cytokine", "systemic inflammation", "endothelial dysfunction"), restricted where possible to human, adult populations.

2.4 Study selection

Titles and abstracts of all identified records were screened for relevance against the eligibility criteria. Full texts of potentially eligible records were then assessed in detail. The study selection process is summarised in the PRISMA flow diagram (Figure 1). Of 24 distinct candidate articles identified and screened, 21 underwent full-text assessment, and 9 met overall eligibility criteria and were included in the review 8 at the primary (blood-based biomarker) tier and 1 (Feng et al.) at the supporting (urinary biomarker) tier defined in Section 2.2 and all 9 are described in the narrative synthesis, while the harvest-plot synthesis (Figure 2) and Section 3.5 are restricted to the 8 primary-eligible studies.

2.5 Data extraction

For each included study, the following data were extracted: first author and year; country and setting; study design; sample size and composition; definition of exposure and comparator groups; biomarker(s) measured and assay method; direction, magnitude, and statistical significance of the reported association; and covariates adjusted for. These data are summarised in Table 1. Because most studies reported biomarker concentrations using heterogeneous summary statistics (means with standard deviation, medians with interquartile range, percentage differences, or adjusted regression coefficients on differently transformed scales), the extractable common denominator across

studies was the direction and statistical significance of the reported association for each biomarker, which forms the basis of the harvest-plot synthesis (Figure 2).

2.6 Risk of bias assessment

Risk of bias in the eight non-randomised studies was appraised using an adaptation of the Newcastle-Ottawa Scale (NOS) for cross-sectional and cohort studies [26], scored across three domains: Selection (0–4 points: representativeness of the exposed group, adequacy of ascertainment of exposure status, non-response/participation rate, and demonstration that the outcome was not present at enrolment where applicable), Comparability (0–2 points: 1 point for adjustment for age, and 1 additional point for adjustment for at least one further key confounder smoking status, socioeconomic position, or body mass index), and Exposure/Outcome ascertainment (0–3 points: use of a validated or objectively measured exposure metric, use of a validated laboratory assay for the biomarker, and adequacy of the follow-up or single-time-point assessment window relative to biomarker half-life). Total scores of 7–9 were classified as low risk, 5–6 as moderate risk, and ≤ 4 as high risk, with an intermediate “low–moderate” band applied at a total of 7 where the domain-level pattern reflected residual concerns not fully captured by the total score. This adapted scale and its scoring anchors were applied, and the risk-of-bias judgements assigned by the authors. The single randomised trial (Olopade et al.) was scored on this same adapted NOS scale for comparability with the other included studies. Results of the NOS-based appraisal actually performed are summarised in Table 2.

2.7 Data synthesis and certainty of evidence

We pre-specified the following decision rule for choosing between quantitative and narrative synthesis, in order to avoid the appearance (or reality) of switching methods post hoc based on which approach produced a more favorable result. For any biomarker reported by three or more included studies using a compatible summary statistic (raw or convertible mean $\pm$ SD, or an effect estimate with a reportable variance, on a common or convertibly transformed scale) and a reasonably comparable exposure contrast, we planned to pool standardised mean differences (SMDs) using a random-effects (DerSimonian-Laird) model. Where these conditions were not met for a given biomarker, narrative synthesis was the pre-specified fallback. On data extraction, it became clear that no biomarker met the conditions for pooling. Studies reported outcomes on incompatible scales (raw means and SDs in some cases, medians and interquartile ranges in others, adjusted percentage changes per unit of continuous exposure in others, and categorical significance only in others); assay platforms and sample matrices differed (serum vs. plasma vs. dried blood spot vs. urine, the latter excluded from the primary blood-based outcome definition in any case); and, even for CRP the most frequently reported biomarker (6 studies) no two studies reported the fully extractable mean $\pm$ SD data under a sufficiently comparable exposure contrast that would be required for valid pooling.

We therefore report a structured narrative synthesis organised by biomarker, following current Synthesis Without Meta-analysis (SWiM) reporting guidance [24], and support this with a semi-quantitative harvest plot (Figure 2) that displays, for each study and biomarker, whether the association was directionally positive and statistically significant, directionally negative and statistically significant, or null/non-significant. This vote-counting-by-direction approach has recognised limitations it does not weight studies by precision or sample size and cannot quantify a summary effect size but it is a transparent and defensible alternative to an inappropriately pooled meta-analysis, and is explicitly endorsed by the Cochrane Handbook and SWiM guidance as a fallback synthesis method when meta-analysis is not feasible [24,27]. We assessed the overall certainty of evidence for each biomarker qualitatively, considering the number and design of contributing studies, consistency of direction, risk of bias, and precision, in a manner informed by GRADE principles [28], without assigning formal GRADE certainty ratings given the small number of contributing studies.

3. Results

3.1 Study selection

The study selection process is depicted in Figure 1. Twenty-four distinct candidate articles addressing solid-fuel HAP and a human biomarker or physiological outcome were identified and screened at title/abstract level; three were excluded as clearly ineligible (ambient-only exposure with no household comparator, or purely animal/ex-vivo mechanistic work). Twenty-one full texts were assessed in detail, of which twelve were excluded most commonly because the outcome was not a systemic (blood-borne) inflammatory biomarker (respiratory symptoms, lung function, exhaled nitric oxide, or carotid intima-media thickness only), because the study compared disease-defined patient groups rather than a general-population fuel-type contrast, or because quantitative data could not be extracted. Nine studies met overall eligibility and were included in the narrative synthesis: eight met the primary (blood-based biomarker) tier defined in Section 2.2, and one (Feng et al., whose only cytokine outcomes were urinary) met only the supporting tier. The harvest-plot synthesis (Section 3.5, Figure 2) and the biomarker-by-biomarker primary synthesis (Section 3.4) are restricted to the eight primary-tier studies; Feng et al.'s findings are reported narratively as secondary, supportive evidence (Section 3.4.4b).

3.2 Characteristics of included studies

The nine included studies were conducted across six countries: India (three studies), Peru (two studies), China, Nigeria, Honduras, and South Africa (one study each). Study designs comprised eight cross-sectional studies including one multi-site study (Burroughs Peña et al.) in which exposure was assigned at the household rather than individual level and one randomised controlled trial (an ethanol-cookstove intervention in pregnant Nigerian women, Olopade et al.). Sample sizes and units of analysis differed considerably across studies and are reported as given in each source rather than aggregated into a single total, to avoid conflating individually sampled adults with households and avoid conflating enrolled with analyzed participants: seven studies reported a clear individual-level analytic sample size, summing to at least 2,362 adults (Dutta et al., 1,087; Banerjee et al., 268; Feng et al., 75; Olopade et al., 271 analysed of 324 enrolled; Benka-Coker et al., 146; Misra et al., 415; Mitra et al., 100); Burroughs Peña et al. enrolled 617 households rather than individually sampled adults; and Caravedo et al. did not clearly specify a precise analytic sample size in the source abstract available to us, which should be confirmed from the full text before this figure is finalised. Exposure contrasts were most commonly biomass (wood, dung, or crop residue) versus LPG or electricity; two studies used continuous measures of personal or kitchen PM2.5/carbon monoxide; one study additionally distinguished coal from biomass users. Populations were predominantly women identified as the household's primary cook, reflecting the sex-differentiated exposure pattern that characterises solid-fuel HAP globally. Two studies enrolled pregnant women specifically. Table 1 summarises study characteristics, exposure contrasts, biomarkers assessed (categorised as primary blood-based inflammatory markers, vascular/endothelial markers, or secondary/mechanistic and other physiological measures — see the note beneath Table 1), and the direction of key findings.

Table 1. Characteristics of included studies (n = 9: 8 meeting the primary blood-based-biomarker eligibility tier, plus 1 — Feng et al. — meeting only the supporting, urinary-biomarker tier; see Section 2.2).

Study, year (country)	Design	Population (n)	Exposure contrast	Biomarkers assessed	Key direction of effect
Dutta et al., 2012 (India)	Cross-sectional	635 biomass users vs 452 LPG users; premenopausal, non-smoking rural women	Biomass (wood/dung/crop residue) vs LPG for cooking	[Primary] serum IL-6, CRP, TNF- α , IL-8 (ELISA) [Oxidative-stress] ROS, SOD	$\uparrow$ IL-6, CRP, TNF- α , IL-8 (primary); $\uparrow$ ROS, $\downarrow$ SOD (secondary); $\uparrow$ hypertension/tachycardia prevalence
Banerjee et al., 2012 (India)	Cross-sectional	142 biomass users vs 126 LPG users; premenopausal women	Biomass vs LPG	[Primary] serum TNF- α , IL-6, IL-12, IL-8 [Other/mechanistic] neutrophil surface markers (CD35, CD16, CD11b/CD18)	$\uparrow$ TNF- α (+72%), IL-6 (+67%), IL-12 (+54%), IL-8 (doubled) (primary); $\uparrow$ neutrophil activation (secondary)
Caravedo et al., 2016 (Peru)	Cross-sectional	Biomass-exposed vs non-exposed adults (Puno region); precise analytic n not clearly stated in the available abstract	Chronic biomass fuel smoke exposure vs none	[Primary] hs-CRP [Vascular/endothelial] ICAM-1, VCAM-1, E-selectin, von Willebrand factor	$\downarrow$ hs-CRP (primary, counter-directional); $\uparrow$ ICAM-1, VCAM-1, E-selectin (vascular/endothelial)
Feng et al., 2021 (China, Fenwei Plain)	Cross-sectional, 3-group, 2-season	75 female homemakers: biomass, coal, and non-solid-fuel control groups	Biomass or coal vs clean/non-solid fuel	[Secondary – urinary, not blood-based] IL-6, TNF- α , 8-OHdG [Other/physiological] blood pressure, heart rate	$\uparrow$ urinary IL-6 in solid-fuel users (both seasons, more marked in winter); urinary TNF- α not consistently elevated (secondary/supportive evidence only)
Olopade et al., 2017 (Nigeria)	Randomised controlled trial	324 pregnant women (162 ethanol-stove intervention vs 162 firewood/kerosene controls); 271 analyzed	Bioethanol (“CleanCook”) stove vs traditional firewood/kerosene	[Primary] serum TNF- α , IL-6, IL-8 [Other/mechanistic] retinol-binding protein, malondialdehyde	$\downarrow$ TNF- α trajectory (1st $\rightarrow$ 3rd trimester) in intervention arm (primary); IL-6, IL-8 not significantly different (primary)
Benka-Coker et al., 2022 (Honduras)	Cross-sectional	146 women (traditional vs Justa wood-burning stoves)	Personal/kitchen PM2.5 and black carbon (continuous), by stove type	[Primary] dried-blood-spot CRP, SAA, IL-1 β , IL-8, TNF- α [Vascular/endothelial] ICAM-1, VCAM-1	$\uparrow$ CRP with PM2.5/BC (10.5% $\uparrow$ CRP per 25% $\uparrow$ personal PM2.5) (primary); other primary and vascular markers largely null
Misra et al., 2018 (South Africa)	Cross-sectional (SOWB cohort)	415 reproductive-age women	Wood vs electricity as main cooking fuel	[Primary] plasma interleukins, TNF- α , CRP, serum amyloid A (SAA)	No significant association for most primary markers; $\downarrow$ SAA with indoor wood use; imprecise $\uparrow$ wheezing odds (respiratory outcome, not synthesized here)
Burroughs Peña et al., 2020 (Peru)	Cross-sectional, multi-site (household-level exposure assignment)	617 households (not individually sampled adults) across 4 settings varying in urbanization/altitude/biomass use	Indoor PM2.5 and CO concentration (continuous), stratified by biomass cookstove use	[Primary] CRP [Other/cardiometabolic] HbA1c, blood pressure, exhaled CO	$\uparrow$ CRP associated with indoor PM2.5/CO (primary); associations attenuated among habitual biomass users
Mitra et al., 2025 (India)	Cross-sectional	100 rural women (biomass vs LPG/clean fuel)	Biomass vs clean cooking fuel	[Primary] serum CRP, IL-8 [Other/mechanistic] 8-OHdG, lactate dehydrogenase (LDH), Clara cell protein (CC16)	$\uparrow$ CRP (7.96 vs 1.68 mg/L), $\uparrow$ IL-8 (51.3 vs 9.6 pg/mL) (primary); $\downarrow$ CC16, a lung-epithelial-injury marker, and $\uparrow$ 8-OHdG/LDH (secondary/mechanistic)

Abbreviations: LPG, liquefied petroleum gas; CRP, C-reactive protein; IL, interleukin; TNF- α , tumor necrosis factor-alpha; ROS, reactive oxygen species; SOD, superoxide dismutase; 8-OHdG, 8-hydroxy-2'-deoxyguanosine; SAA, serum amyloid A; ICAM-1, intercellular adhesion molecule-1; VCAM-1, vascular cell adhesion molecule-1; vWF, von Willebrand factor; HbA1c, glycated hemoglobin; LDH, lactate dehydrogenase; CC16, Clara cell 16 protein.

Marker categories, as bracketed in the “Biomarkers assessed” column: [Primary] blood-based (serum, plasma, or dried-blood-spot) pro-inflammatory cytokines and acute-phase proteins, which form the core outcome of this review and the basis of the harvest-plot synthesis (Figure 2; Section 3.5); [Vascular/endothelial] soluble adhesion molecules and coagulation-related markers, synthesized separately (Section 3.4.5); [Oxidative-stress/other

mechanistic] markers of oxidative damage, tissue injury, or cellular activation that are biologically related but conceptually distinct from cytokine-mediated inflammation, treated as supportive mechanistic evidence only; and [Other/cardiometabolic or physiological] outcomes (HbA1c, blood pressure, heart rate, exhaled CO) that are not inflammatory biomarkers and are reported in Table 1 for completeness but excluded from the primary synthesis. Feng et al. measured IL-6 and TNF- α in urine rather than blood; because our eligibility criteria define the primary outcome as blood-based systemic inflammatory biomarkers (Section 2.2), these urinary results are treated throughout as secondary, supportive evidence rather than primary findings.

3.3 Risk of bias within studies

Risk-of-bias appraisal (Table 2) classified one study (the randomised trial) as low risk of bias, three as low-to-moderate risk, and five as moderate or moderate-to-high risk. The most common limitations across cross-sectional studies were incomplete adjustment for confounders beyond age (particularly household socioeconomic position, secondhand tobacco smoke exposure, and body mass index), single time-point exposure and biomarker assessment despite biomarkers such as CRP being sensitive to acute intercurrent illness, and, in several smaller studies, limited statistical power to detect modest effect sizes. No study was rated as critically high risk of bias warranting exclusion from synthesis.

Table 2. Risk-of-bias assessment of included studies (adapted Newcastle-Ottawa Scale).

Study	Selection (0–4)	Comparability (0–2)	Exposure/ Outcome ascertainment (0–3)	Total (0–9)	Risk of bias	Domain of greatest concern
Dutta et al., 2012	3	1	2	6	Moderate	Limited confounder adjustment; single-day exposure and biomarker snapshot
Banerjee et al., 2012	3	1	2	6	Moderate	Comparability (partial adjustment); cross-sectional design
Caravedo et al., 2016	3	1	2	6	Moderate	Unexpected direction for CRP/vWF suggests possible residual confounding or reverse causation
Feng et al., 2021	3	1	2	6	Moderate	Small per-group n (~25); urinary rather than serum biomarkers
Olopade et al., 2017	4	2	3	9	Low	Randomised design; strongest internal validity in this evidence base
Benka-Coker et al., 2022	3	2	2	7	Low–Moderate	Cross-sectional; dried-blood-spot method still being validated against serum
Misra et al., 2018	3	2	2	7	Low–Moderate	Possible non-differential exposure misclassification (wood use may be intermittent)
Burroughs Peña et al., 2020	3	1	2	6	Moderate	Household- rather than individual-level exposure assignment
Mitra et al., 2025	2	1	2	5	Moderate–High	Limited reporting of confounder adjustment; small sample (n=100)

3.4 Findings by biomarker

3.4.1 C-reactive protein (CRP)

CRP was the most frequently reported biomarker, assessed in six of nine studies. Four studies reported statistically significant elevation of CRP among solid-fuel-exposed adults relative to comparators: Dutta et al. reported significantly higher serum CRP among 635 biomass users versus 452 LPG users in rural India [13]; Benka-Coker et al. found a 10.5% increase in dried-blood-spot CRP per 25% increase in personal PM2.5 among Honduran women using wood-burning stoves [17]; Burroughs Peña et al. reported a positive association between indoor PM2.5/CO and CRP across four Peruvian settings, although this association was attenuated among habitual biomass users, suggesting possible adaptive or survivor-population effects [18]; and Mitra et al. reported markedly higher CRP among Indian biomass users compared with clean-fuel users (7.96 vs. 1.68 mg/L) [20]. In contrast, Caravedo et al. reported significantly lower hs-CRP among chronically biomass-exposed Peruvian adults compared with non-exposed adults, a counterintuitive finding the authors themselves highlighted, occurring alongside significantly elevated

endothelial adhesion molecules in the same population [15]. Misra et al., in the largest single study (n = 415), found no significant association between wood use and CRP [19].

3.4.2 Interleukin-6 (IL-6)

Blood-based (serum/plasma) IL-6 was reported in three studies, two of which found significant elevation among solid-fuel-exposed adults: Dutta et al. [13] and Banerjee et al. (67% higher IL-6 among biomass users) [14], both in India. The Nigerian ethanol-cookstove trial found no significant between-arm difference in serum IL-6 [23], and the South African study found no association in plasma IL-6 [19]. As secondary, supportive evidence outside the primary blood-based synthesis, Feng et al. reported significantly higher urinary IL-6 among biomass and coal users compared with non-solid-fuel users in both winter and summer, with a more pronounced difference in winter, coincident with higher ambient and indoor PM_{2.5} [16]; because this outcome was measured in urine rather than blood, it is not counted toward the primary IL-6 synthesis in Figure 2 or in the summary statement in Section 3.5, but is directionally consistent with the blood-based findings.

3.4.3 Tumor necrosis factor-alpha (TNF-α)

Blood-based TNF-α was reported in four studies. Two cross-sectional Indian studies found higher serum TNF-α among solid-fuel users (Dutta et al. [13], Banerjee et al., 72% higher [14]). Notably, the sole randomised trial found a favorable trajectory, a decrease in serum TNF-α from the first to third trimester of pregnancy in the ethanol-stove intervention arm relative to the firewood/kerosene control arm, which the trial authors interpreted as consistent with reduced cardiovascular and prothrombotic stress, though we note this is the trial authors' interpretation rather than a directly demonstrated clinical outcome in that trial [23]. The South African [19] and Honduran [17] studies found no significant association in serum/plasma TNF-α. As secondary, supportive evidence, Feng et al. found no significant difference in urinary TNF-α between fuel groups [16]; this null secondary finding is not counted toward the primary TNF-α synthesis.

3.4.4 Interleukin-8 (IL-8)

Blood-based IL-8 was elevated among solid-fuel users in three studies: Banerjee et al. (doubled) [14], Dutta et al. [13], and Mitra et al. [20], but was not significantly different between arms in the Nigerian trial [23] or in the Honduran secondary analysis [17]. Banerjee et al. additionally reported increased surface expression of neutrophil activation markers (CD35, CD16, CD11b/CD18) among biomass users, a secondary/mechanistic finding consistent with innate immune cell activation beyond soluble cytokine elevation, though this was not itself a blood cytokine concentration and is reported as supportive evidence [14].

3.4.4b Secondary and mechanistic markers (oxidative stress, tissue injury, and other physiological measures)

Several included studies additionally reported markers that are biologically related to, but conceptually distinct from, cytokine-mediated systemic inflammation, and are summarised here as supportive rather than primary evidence (see the marker-category note beneath Table 1). Dutta et al. reported elevated reactive oxygen species (ROS) and reduced superoxide dismutase (SOD) activity among biomass users, indicating heightened oxidative stress [13]. Mitra et al. reported elevated 8-hydroxy-2'-deoxyguanosine (8-OHdG, an oxidative DNA-damage marker) and lactate dehydrogenase (LDH, a marker of cellular injury), alongside reduced Clara cell protein (CC16, a marker of lung epithelial integrity) [20]. Olopade et al. additionally measured retinol-binding protein and malondialdehyde, an oxidative-stress marker, without a significant intervention effect reported for these secondary measures [23]. Burroughs Peña et al. reported that indoor PM_{2.5}/CO exposure was associated with higher glycated hemoglobin (HbA1c) and blood pressure in addition to CRP, situating systemic inflammation within a broader cardiometabolic risk picture in that population, though HbA1c and blood pressure are cardiometabolic rather than inflammatory outcomes and are not part of the primary synthesis [18]. These secondary findings are broadly consistent with, and mechanistically compatible with, the primary cytokine and CRP findings, but were not used to determine study eligibility or counted in the harvest-plot synthesis (Section 3.5).

3.4.5 Endothelial adhesion molecules and vascular markers

Only one study, Caravedo et al., directly assessed endothelial adhesion molecules, finding significantly higher soluble ICAM-1, VCAM-1, and E-selectin among chronically biomass-exposed adults, alongside the aforementioned reduction in hs-CRP and von Willebrand factor [15]. The authors proposed that biomass smoke exposure may preferentially activate endothelial inflammatory pathways distinct from the classical hepatic acute-phase response captured by CRP, a hypothesis that, if correct, would have important implications for biomarker selection in future HAP research (Section 4.3).

3.5 Semi-quantitative harvest-plot synthesis

Figure 2 displays the direction and statistical significance of each primary, blood-based study-biomarker combination extracted (secondary/urinary and mechanistic markers, Section 3.4.4b, are not plotted). Most reported study-biomarker comparisons were directionally consistent with higher inflammatory biomarker levels among participants exposed to solid-fuel HAP, although the magnitude and statistical significance of associations varied substantially across studies. For descriptive completeness, of the $k = 22$ primary-biomarker study-cells with a reported result, 13 (59%) showed a statistically significant elevation in the solid-fuel-exposed or higher-exposure group, 1 (5%) showed a statistically significant reduction, and 8 (36%) were null. We report this tally only as a descriptive summary of the pattern visible in Figure 2, not as a quantitative effect estimate: vote-counting by direction and statistical significance does not weight studies by sample size or precision, treats a small study with a large effect the same as a large study with a small effect, and does not establish that a null result reflects a true absence of association rather than limited power. Significant elevations were concentrated in the Indian and Chinese cross-sectional studies and in the Peruvian CRP study; null findings were concentrated in the South African study and, for secondary biomarkers, the Honduran study. The single inverse finding CRP in the Peruvian endothelial-marker study occurred alongside positive findings for a different biomarker class (adhesion molecules) in the same study population. Finding: Caravedo et al. observed significantly lower hs-CRP but significantly higher soluble adhesion molecules among biomass-exposed adults in the same sample [15]. Interpretation: one possible explanation is that biomass smoke exposure in that population preferentially activated endothelial inflammatory pathways distinct from the hepatic acute-phase response captured by CRP. Uncertainty: this single cross-sectional study cannot establish that mechanism, and alternative explanations including chance, residual confounding, or population-specific factors affecting CRP metabolism cannot be excluded on the available evidence.

3.6 Sources of heterogeneity

Several factors plausibly contribute to the heterogeneity observed across studies. First, exposure intensity varied substantially: indoor PM_{2.5} concentrations reported across included studies ranged from roughly 35–60 $\mu\text{g}/\text{m}^3$ in some Peruvian settings to over 900 $\mu\text{g}/\text{m}^3$ in some Indian biomass-cooking kitchens, and the South African study which found the least evidence of association explicitly noted that wood-burning exposures in that setting may have been lower and more intermittent than in the Indian and Central American studies, given cooking practices and, in some households, use of a separate cooking structure [19]. Second, biomarker assay matrix differed (serum, plasma, dried blood spot, urine), which affects both absolute concentrations and measurement precision. Third, populations differed in physiological state (pregnant vs. non-pregnant), age structure, and background inflammatory burden (e.g., prevalence of infection, anemia, or obesity), all of which independently influence CRP and cytokine levels. Fourth, only one study was a randomised trial; the remainder were cross-sectional, precluding causal inference and leaving open the possibility of residual confounding or reverse causation (e.g., healthier individuals self-selecting into clean-fuel use).

4. Discussion

4.1 Summary of findings

This systematic review synthesised evidence from nine studies examining inflammatory biomarkers among adults exposed to solid-fuel household air pollution: eight met our primary, blood-based-biomarker eligibility tier, and one met only a supporting, urinary-biomarker tier. Among the eight primary-tier studies, most reported study-biomarker comparisons were directionally consistent with higher levels of pro-inflammatory markers most consistently CRP, IL-6, and TNF- α among solid-fuel-exposed adults relative to clean-fuel or lower-exposure comparators; some studies reported effect magnitudes that, if confirmed in larger and more rigorous studies, would be clinically notable (e.g., a roughly five-fold difference in mean CRP reported in one Indian study [20], and 50–70% relative differences in IL-6 and TNF- α reported in others [14]). This pattern is directionally consistent with the more extensive literature on ambient particulate air pollution and CRP [9,10], and with experimental data showing that biomass particulate matter robustly activates NF- κB signaling and pro-inflammatory cytokine release in airway epithelial cells and in animal models [29]. However, the evidence is not uniform: the largest single study included here ($n = 415$) found little evidence of association for most biomarkers [19], and one otherwise well-conducted study found a significant reduction in CRP alongside elevated endothelial adhesion molecules in the same exposed population [15]. The single randomised trial provides the strongest evidence available for a causal interpretation and found a favorable effect of a clean-fuel intervention on TNF- α trajectory in pregnancy, though not on IL-6 or IL-8 [23]; because the remaining eight studies were cross-sectional, this review's findings for those studies should be read as describing observed associations rather than demonstrated causal effects (Section 4.6).

4.2 Biological plausibility and mechanisms

The biological plausibility of an association between solid-fuel HAP and systemic inflammation rests on well-characterised pathways. Inhaled fine and ultrafine particulate matter can deposit deep in the alveoli, where it is phagocytosed by macrophages that release pro-inflammatory

mediators (TNF- α , IL-6, IL-1 β) locally; these mediators, together with particulate constituents that translocate into the systemic circulation, are thought to stimulate hepatic synthesis of acute-phase proteins including CRP and serum amyloid A, and to activate circulating leukocytes and vascular endothelium [6,8,29]. Experimental work directly exposing mice and human airway epithelial cells to biomass particulate matter has demonstrated robust induction of a broad panel of neutrophil and macrophage chemokines and cytokines, with some differences in cytokine profile between wood-derived and dung-derived biomass particulate matter, suggesting fuel-source-specific toxicity that may partly explain between-study heterogeneity [29,30]. As already noted in Section 3.5: Caravedo et al. observed elevated endothelial adhesion molecules without concomitant CRP elevation [15] (finding); one possible explanation is that biomass smoke may, in some populations or exposure regimes, act more directly on the vascular endothelium than through the classical hepatic acute-phase pathway (interpretation); however, this remains a speculative hypothesis derived from a single cross-sectional study, and cannot be distinguished from chance, residual confounding, or other population-specific explanations on the available evidence (uncertainty). Mechanistic follow-up combining systemic biomarkers with direct measures of vascular function (e.g., flow-mediated dilation) would be needed to evaluate this hypothesis properly.

4.3 Comparison with the ambient air pollution literature

Our findings can be usefully compared with the more extensive ambient (outdoor) PM_{2.5}-CRP literature. An earlier narrative review of epidemiologic studies concluded that the evidence for ambient particulate air pollution and CRP was generally consistent in children and in healthy adults, but less consistent among adults with chronic inflammatory conditions, potentially reflecting anti-inflammatory medication use or altered baseline inflammatory regulation in that subgroup [11]; because this was a narrative review rather than a formal meta-analysis, we cite it as descriptive context rather than as a quantitative benchmark. Separately, two more recent systematic reviews and meta-analyses that did pool quantitative estimates concluded that ambient particulate air pollution is associated with elevated circulating CRP [9,10]. Notably, at least one large study of outdoor air pollution and systemic inflammation in the general English population found no association between ambient PM₁₀ and either CRP or fibrinogen, illustrating that null findings are not unique to the HAP literature and may partly reflect the comparatively modest and intermittent nature of ambient, as opposed to household, exposure in some settings [31]. The much higher personal exposure concentrations, longer daily exposure durations, and closer physical proximity to the combustion source that characterise solid-fuel cooking, relative to ambient pollution exposure, would plausibly be associated with a larger and more readily detectable difference in inflammatory biomarkers between higher- and lower-exposure groups; this is broadly consistent with, though not proof of, the relatively large effect sizes reported in several of the included HAP studies conducted in high-exposure settings (e.g., rural India) [13,14,20].

4.4 Clinical and public health implications

If confirmed by more rigorous longitudinal and interventional research, a reproducible association between solid-fuel HAP and higher systemic inflammatory biomarker levels would strengthen the mechanistic rationale for cardiovascular disease as a major, and possibly underappreciated, component of the HAP-attributable disease burden, alongside the more extensively documented respiratory outcomes [1,3]. It would also support the use of inflammatory biomarkers, particularly CRP measured via minimally invasive dried-blood-spot methods that have shown promise in field settings [17], as feasible intermediate endpoints for evaluating clean-cookstove and clean-fuel-transition interventions in future trials, where hard clinical cardiovascular endpoints typically require sample sizes and follow-up durations that are difficult to achieve. At the same time, the mixed findings summarised here, and particularly the null result in the largest included study [19] and the counterintuitive CRP finding in another [15], caution against overstating the certainty or universality of this relationship, and argue against using any single biomarker in isolation as a definitive marker of HAP-related cardiovascular risk, and against describing the present, largely cross-sectional evidence as having established that HAP causes or produces this inflammatory response, rather than being associated with it.

4.5 Limitations

The heterogeneity in exposure metrics, biomarker assays, and reporting formats meant that no biomarker met our pre-specified criteria for quantitative pooling; the harvest-plot vote-counting synthesis presented here is a transparent but comparatively low-resolution alternative that does not account for study precision, sample size, or the magnitude of effect, does not distinguish a small imprecise study from a large precise one, and does not establish that a null result reflects a true absence of association rather than limited statistical power. Eight of the nine included studies (seven of the eight primary-tier studies, plus the one supporting-tier study) were cross-sectional, precluding causal inference for those studies. Findings from these studies are therefore described throughout as associations (“was associated with,” “was observed to be higher among”) rather than as causal effects (“caused,” “raised,” “led to”), and the direction of causality particularly for the counterintuitive CRP finding in Caravedo et al. [15]; remains open to alternative explanations including reverse causation, selective uptake of clean fuels by healthier or wealthier households, and residual confounding by socioeconomic position, secondhand tobacco smoke, diet, and infection/parasite burden, all of which independently affect inflammatory biomarkers and are imperfectly measured or adjusted for across the included studies. These remain plausible interpretations rather than demonstrated mechanisms. Finally, nearly all included studies enrolled women, reflecting the sex-

differentiated real-world exposure pattern for cooking-related HAP but limiting generalizability of these findings to men, whose personal exposure profile and, in some settings, comorbidity profile (e.g., concurrent active tobacco smoking) may differ substantially.

4.6 Recommendations for future research

Future research would benefit from: (i) larger, multi-country, longitudinal cohort studies with repeated biomarker measurement to characterise within-person change in relation to changing exposure and to reduce the influence of acute, incidental illness on single-time-point CRP measurement; (ii) standardisation of biomarker assay platform and sample matrix (or, at minimum, transparent cross-matrix calibration) to enable future pooling; (iii) nesting inflammatory biomarker panels within existing and future randomised clean-cookstove and clean-fuel-transition trials, following the model established by the Household Air Pollution Intervention Network (HAPIN) trial and the Nigerian ethanol-cookstove trial included here [23,32], to obtain causally interpretable estimates and (iv) inclusion of men and of non-pregnant and pregnant women as pre-specified subgroups.

5. Conclusion

Available evidence, drawn from nine studies conducted across six countries (eight meeting a primary, blood-based-biomarker eligibility tier), is broadly, though not uniformly, consistent with a possible association between solid-fuel household air pollution exposure and higher systemic inflammatory biomarker levels in adults particularly CRP, IL-6, and TNF- α and this is biologically plausible given a well-characterised mechanistic pathway linking inhaled particulate matter to systemic inflammation and, potentially, cardiovascular and metabolic disease. This should be read as a possible and not-yet-quantified association rather than a robust, quantitatively established effect. Larger, standardised, and ideally interventional studies are needed before systemic inflammatory biomarkers can be confidently used either as mechanistic evidence in policy discussions about clean-fuel transition or as validated intermediate endpoints in cookstove-intervention trials.

Declarations

Funding: This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Conflicts of interest: The authors declare that they have no competing interests.

Data availability: The data extraction table underlying this review (Table 1) is provided in the manuscript. A full extraction spreadsheet with verbatim quotations and page/table locators from each included study is prepared and deposited alongside journal submission, per current transparency expectations for systematic reviews.

Author contributions: OJA and KSPA conceived and designed the study. KSPA and OJA developed the search strategy, conducted the literature search, and performed data extraction. AAO, OEO, OBJ, and SSE contributed to screening, data interpretation, and critical review of the manuscript. KSPA drafted the manuscript, while OJA and KSPA revised the manuscript critically for important intellectual content. All authors reviewed and approved the final version of the manuscript.

PROSPERO registration: A revised protocol was registered on PROSPERO consistent with PROSPERO's current guidance on systematic review.

Acknowledgements: The authors acknowledge the support and contributions of colleagues and reviewers who provided valuable feedback during the development and refinement of this manuscript.

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Figures



Figure 1

PRISMA 2020 flow diagram of study identification and selection.

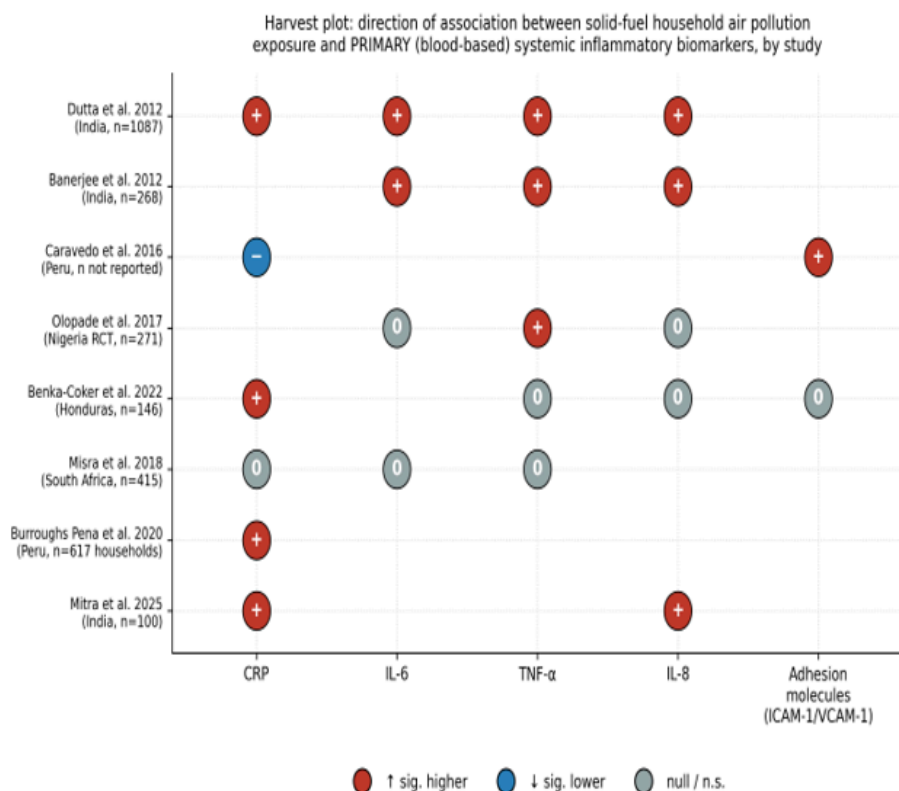


Figure 2

Harvest plot summarizing the direction and statistical significance of the association between solid-fuel household air pollution exposure and primary, blood-based systemic inflammatory biomarkers, by included study. Red circles (+) indicate a statistically significant elevation among solid-fuel-exposed/higher-exposure participants; blue circles (-) indicate a statistically significant reduction; grey circles (0) indicate a null or non-significant association. Cells are blank where a study did not report that biomarker. Feng et al. (2021) is not plotted here because its cytokine outcomes (IL-6, TNF-α) were measured in urine rather than blood and are therefore treated as secondary, supportive evidence (Section 3.4.4b) rather than primary outcomes under our pre-specified eligibility criteria (Section 2.2).