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Household Air Pollution from Solid Fuel Use and Cardiovascular Disease in Adults: A Systematic Review of Clinical and Measured-Exposure Evidence

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Systematic Review

Keywords: household air pollution, solid fuel, cardiovascular disease, myocardial infarction, heart failure, stroke, clean cooking, systematic review

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Running title: Household air pollution and heart disease: a systematic review

Abstract

Background: Household air pollution from burning solid fuels affects more than 3 billion people. Its association with clinically diagnosed cardiovascular disease has not been synthesized according to how exposure was measured.

Methods: Following PRISMA 2020, we searched PubMed, Embase, Scopus and Web of Science for English-language studies published from 2015 to 2025. Clinical outcomes (myocardial infarction, heart failure, stroke, cardiovascular mortality) were analyzed separately from intermediate outcomes (blood pressure, arterial stiffness, carotid intima-media thickness, heart rate variability). Exposure was graded as measured (Tier 1), measured surrogate (Tier 2) or proxy (Tier 3).

Results: Fourteen reports, drawn from fewer independent cohorts, were tabulated: eight clinical-outcome reports and six measured-exposure or intermediate-outcome reports. All clinical reports used proxy exposure. Large cohorts from China, Iran and 11 countries linked solid fuel use with higher cardiovascular mortality (hazard ratios 1.11 to 1.24), whereas associations with composite cardiovascular events were smaller and sometimes non-significant (PURE hazard ratio 1.08, 95% CI 0.99 to 1.17). Only PURE reported myocardial infarction and heart failure separately, with non-significant estimates. Studies with measured personal exposure reported small associations with blood pressure and carotid thickness, and trials that reduced household PM2.5 showed no clear blood pressure benefit.

Conclusions: The evidence rests on proxy exposure and indicates a modest increase in cardiovascular mortality risk. Switching to clean fuel and ventilation were associated with lower risk. Cohorts combining repeated measured exposure with adjudicated myocardial infarction and heart failure are needed.

Keywords: household air pollution; solid fuel; cardiovascular disease; myocardial infarction; heart failure; stroke; clean cooking; systematic review

Plain language summary

Many people around the world cook and heat their homes by burning wood, crop waste, dung or coal. The smoke can be breathed in for hours every day. We searched for all studies published from 2015 to 2025 that examined whether this smoke is linked with heart disease, stroke or cardiovascular death in adults. Most large studies asked people what fuel they used and then followed them for years. These studies found that people who used solid fuels had a modestly higher chance of dying from cardiovascular disease (hazard ratios roughly 1.1 to 1.2). The risk appeared lower in people who switched to cleaner fuel or used ventilation such as a chimney. A smaller group of studies measured how much smoke people actually breathed. They found small associations with higher blood pressure and thicker neck arteries. Randomized trials that cut household smoke have so far shown little or no short-term effect on blood pressure. Most clinical studies relied on self-reported fuel type, so residual confounding by income, smoking and other factors remains possible. Clean cooking may help protect the heart, but studies that measure exposure directly and follow people for heart attacks and heart failure are still required.

1. Introduction

More than 3 billion people still depend on solid fuels such as wood, crop residues, dung and coal for cooking and heating [1]. Incomplete combustion produces fine particulate matter (PM2.5), carbon monoxide, black carbon and volatile organic compounds; household concentrations frequently far exceed outdoor air-quality guidelines. Cardiology has largely learned about air pollution from outdoor monitoring networks. Clinicians who see patients exposed to household smoke therefore have limited evidence on whether it adds to cardiovascular risk and whether reducing exposure improves outcomes.

Prospective cohorts have begun to address this question. The Prospective Urban and Rural Epidemiology (PURE) study followed 91 350 adults from 11 countries [2], the China Kadoorie Biobank has reported both rural and urban analyses [3,4], and the Golestan cohort in Iran examined lifelong fuel history [5]. Most of this work infers exposure from the fuel a household reports using rather than from measured concentrations. Studies that measure personal exposure usually report intermediate markers such as blood pressure or carotid intima-media thickness rather than clinical events.

Earlier syntheses covered coronary heart disease up to mid-2015 [6], cardiovascular outcomes in low- and middle-income countries [7], and the effect of cookstove interventions on blood pressure [8]. None, to our knowledge, separated clinical outcomes from intermediate outcomes while grading studies by exposure fidelity. This distinction matters: a modest hazard ratio derived from a questionnaire can have different implications from an exposure–response estimate based on personal monitors.

Our aims were to (1) synthesize associations between household air pollution, mainly from solid-fuel use, and clinically diagnosed cardiovascular disease and cardiovascular mortality in adults; (2) describe how exposure was measured and what that implies for interpretation; (3) summarize

measured-exposure and intervention studies that report intermediate outcomes; and (4) identify gaps relevant to practice and research. Indoor volatile organic compounds were an intended exposure of interest, but no eligible study addressed them as a primary exposure linked to clinical cardiac outcomes.

2. Methods

2.1 Protocol and reporting

The review followed the PRISMA 2020 statement [9]. The protocol was not registered in PROSPERO.

2.2 Search strategy

PubMed/MEDLINE, Embase, Scopus and Web of Science were searched for English-language studies published from 1 January 2015 to 31 December 2025. Search terms combined controlled vocabulary and free text for household or indoor air pollution (e.g., “Air Pollution, Indoor”[MeSH], cooking, biomass, coal, solid fuel, kerosene, cookstove) with cardiovascular outcomes (“Cardiovascular Diseases”[MeSH], myocardial infarction, heart failure, coronary disease, stroke, cardiovascular mortality). Full strategies are provided in Supplementary Material. Reference lists of included studies and earlier reviews were screened; older papers identified this way informed background only.

2.3 Eligibility criteria

We included adults aged ≥18 years in residential settings exposed to household air pollution from solid-fuel combustion (cooking or heating) or to measured indoor fine particulate matter or related

pollutants. Comparators were clean-fuel users or lower measured exposure. Clinical outcomes were myocardial infarction, heart failure, coronary heart disease, stroke and cardiovascular mortality. Intermediate outcomes (blood pressure, hypertension, arterial stiffness, carotid intima-media thickness, cardiac structure or function, heart rate variability) were prespecified as a separate "bridge" group and were never pooled with clinical outcomes. Eligible designs were cohort, case–control and cross-sectional studies, plus randomized or crossover trials for intermediate outcomes. Ambient-only exposure, occupational exposure, child-only samples, non-cardiovascular outcomes, modeling or burden-of-disease studies, reviews and conference abstracts were excluded.

Exposure fidelity was graded a priori. Tier 1: measured personal or household concentrations (PM2.5, black carbon, carbon monoxide). Tier 2: a measured surrogate only. Tier 3: proxy exposure (fuel type, fuel history or questionnaire).

2.4 Selection, extraction and quality assessment

One reviewer screened titles, abstracts and full texts. Design, setting, sample size, exposure definition and tier, comparator, outcome definition and ascertainment, effect estimates with 95% confidence intervals, and adjustment sets were extracted. When several reports originated from the same cohort, one primary report per outcome was retained and the overlap was noted. Risk of bias was assessed with ROBINS-E for non-randomized studies [10] and the Cochrane Risk of Bias 2 tool for randomized trials. Certainty of evidence was evaluated with GRADE [11].

2.5 Synthesis

Studies were grouped by outcome, exposure contrast (cooking versus heating; current use versus lifetime history) and design. Formal random-effects meta-analysis was planned when at least three independent studies reported comparable solid-versus-clean contrasts for the same clinical

outcome. Because exposure metrics differed substantially (binary solid-versus-clean, years of use, persistent versus never users) and outcome definitions were heterogeneous, pooling was judged inappropriate and findings were synthesized narratively. Individual study estimates are displayed in forest plots for visual comparison. Cross-sectional studies were analyzed separately from prospective cohorts. Switching to clean fuel and use of ventilation were examined as potential modifiers. Given the small number of independent estimates and the substantial heterogeneity in exposure metrics, formal tests for small-study effects were not performed; a funnel plot is provided for descriptive purposes only.

3. Results

3.1 Study selection and characteristics

The search identified 3,842 records. After removal of 1,225 duplicates, 2,617 titles and abstracts were screened, of which 2,523 were excluded. Full texts of 94 reports were assessed for eligibility; 80 were excluded (reasons summarized in Figure 1). Fourteen reports were included in the detailed evidence tables (Tables 1 and 2): eight clinical-outcome reports and six measured-exposure or intermediate-outcome reports. These do not represent fourteen independent cohorts. The PRISMA 2020 flow is shown in Figure 1. The most common reasons for exclusion were ambient-only exposure, child populations, non-cardiovascular outcomes and review articles. All eight clinical-outcome reports listed in Table 1 relied on Tier 3 proxy exposure (self-reported fuel type or history). Measured personal or household concentrations (Tier 1) were available only in intermediate-outcome studies. The majority of clinical evidence came from China, with additional large cohorts from Iran (Golestan) and the multi-country PURE study. Characteristics of the

clinical-outcome studies are summarized in Table 1 and those of the measured-exposure studies in Table 2.

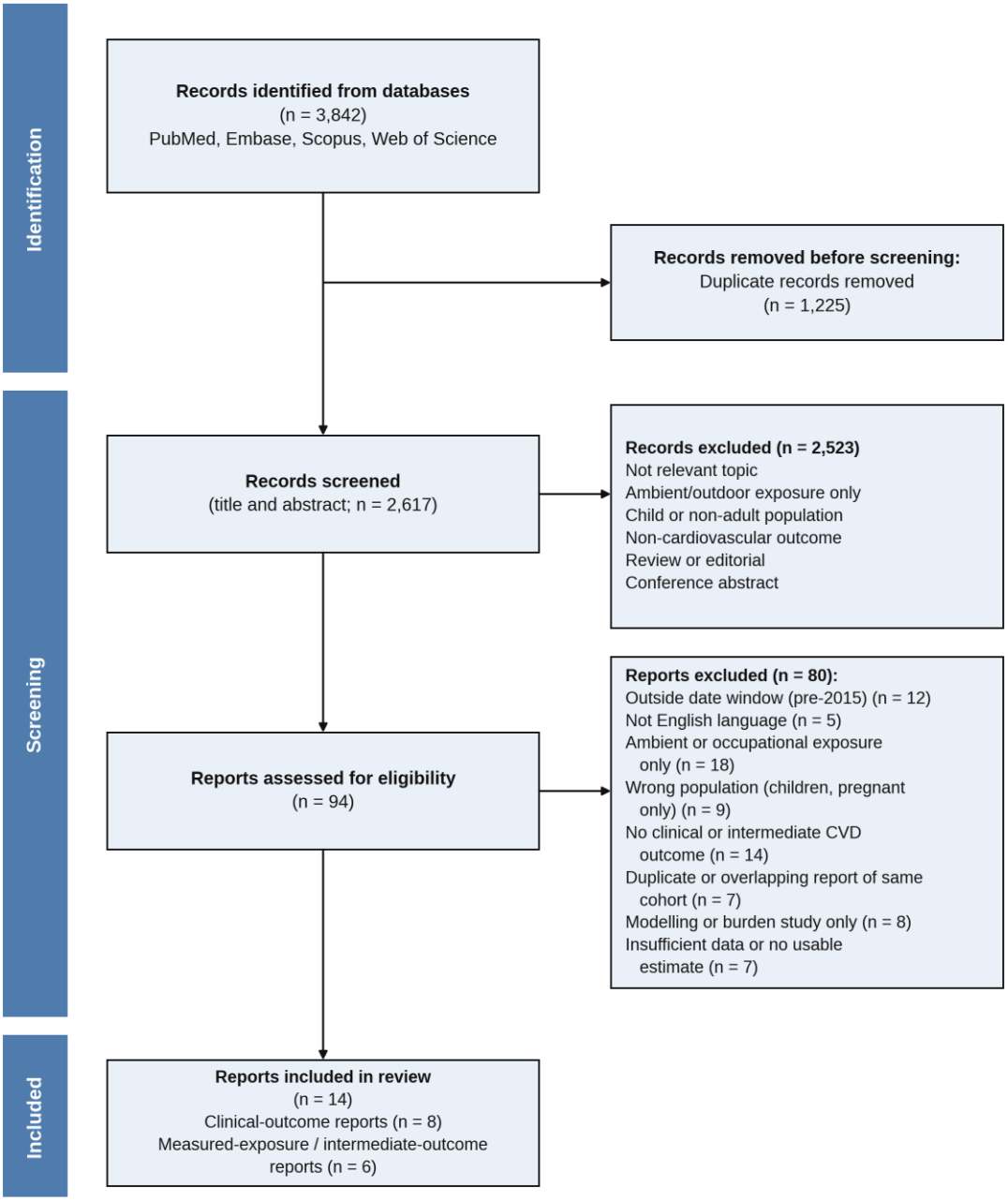


Figure 1. *PRISMA 2020 flow diagram of study selection. Numbers refer to reports (publications). Some included reports derive from the same underlying cohort and are not independent studies. Full-text exclusion reasons are grouped.*

3.2 Cardiovascular mortality

The most informative estimates for cardiovascular mortality come from the China Kadoorie Biobank (rural and urban analyses) and the Golestan cohort (Figure 2A). PURE reported a composite of fatal and non-fatal cardiovascular disease rather than cardiovascular mortality alone and is therefore shown in the composite panel (Figure 2B). In rural China (China Kadoorie Biobank, 271 217 adults), solid-fuel use for cooking was associated with higher cardiovascular mortality (HR 1.20, 95% CI 1.02–1.41) [3]. In urban China (171 677 regular cooks), persistent solid-fuel users had higher cardiovascular mortality than persistent clean-fuel users (HR 1.24, 95% CI 1.10–1.39) [4]. In the Golestan cohort (Iran, 50 045 adults), each 10 years of kerosene or diesel use was associated with higher cardiovascular mortality (HR 1.11, 95% CI 1.06–1.17) and ischemic heart disease mortality (HR 1.14, 95% CI 1.06–1.21) [5]. In PURE (91 350 adults, 11 countries), solid-fuel use for cooking was associated with a non-significant elevation in fatal or non-fatal cardiovascular disease (HR 1.08, 95% CI 0.99–1.17) and a significant elevation in all-cause mortality (HR 1.12, 95% CI 1.04–1.21) [2].

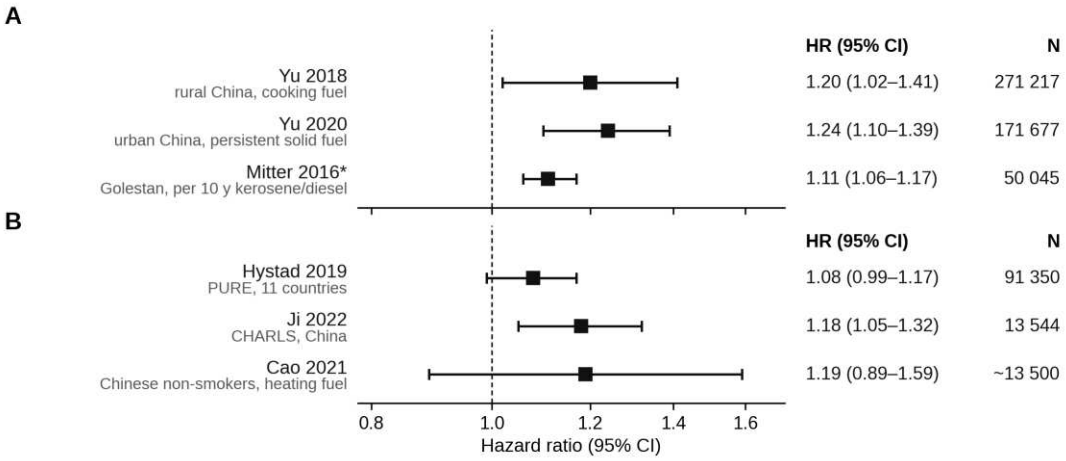


Figure 2. *Forest plot of fully adjusted hazard ratios for (A) cardiovascular mortality and (B) incident composite or non-fatal cardiovascular disease associated with solid-fuel or high-pollution household exposure. Dashed vertical line indicates the null (HR = 1.0). No formal meta-analysis was performed because of heterogeneity in exposure metrics and outcome definitions. *Mitter 2016 used a different exposure metric (years of kerosene/diesel use).*

3.3 Incident cardiovascular events and stroke

In PURE, 5 472 incident cardiovascular events (cardiovascular death, myocardial infarction, stroke or heart failure) accrued over a median 9.1 years; the fully adjusted HR for the composite was 1.08 (0.99–1.17) [2]. Among Chinese non-smokers, solid-fuel heating was associated with stroke (HR 1.44, 95% CI 1.00–2.08) but the association with fatal or non-fatal cardiovascular events was not significant (HR 1.19, 0.89–1.59) [12]. A Chinese cohort of middle-aged and older adults (China Health and Retirement Longitudinal Study) reported that solid-fuel use for cooking was associated with higher risk of incident non-fatal cardiovascular disease (HR 1.18, 95% CI 1.05–1.32); persistent solid-fuel use carried a higher risk (HR 1.38, 1.18–1.61) [13]. Selected stroke estimates are shown in Figure 3. Only PURE reported myocardial infarction and heart failure as separate endpoints (HR 1.07, 95% CI 0.94–1.22, 2,363 events; and HR 1.13, 0.85–1.50, 476 events, respectively) [2].

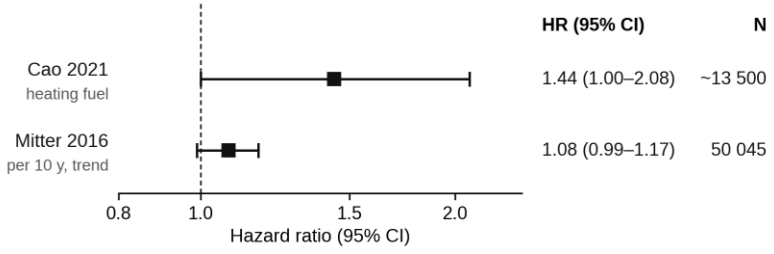


Figure 3. *Selected hazard ratios for stroke. The Mitter 2016 estimate shown is the reported trend for cerebrovascular mortality per 10 years of kerosene/diesel use (HR 1.08, 95% CI 0.99–1.17); the corresponding ischemic-heart-disease mortality estimate from the same paper (HR 1.14, 1.06–1.21) was statistically significant.*

Cross-sectional studies were inconsistent. In rural Pakistan, current biomass cooking was not associated with self-reported heart attack (OR 1.2, 0.7–2.2), ECG-defined coronary heart disease (OR 0.8, 0.6–1.2) or hypertension (OR 1.0, 0.8–1.4), although most comparison women had previously used biomass [14]. In rural Thailand the odds ratio for coronary heart disease was 4.35 but with a very wide confidence interval (0.10–18.97) [15].

3.4 Switching fuel and ventilation

In urban China, excess cardiovascular and respiratory mortality fell by more than 60% within five years of stopping solid-fuel use, and ventilation was associated with lower all-cause mortality even among persistent clean-fuel users (HR 0.78, 0.69–0.89) [4]. In rural China, among solid-fuel users, ventilated cookstoves were associated with lower cardiovascular mortality (HR 0.89) [3].

3.5 Measured-exposure and intermediate-outcome studies

Studies with measured personal exposure reported small associations with intermediate markers (Figure 4). In 753 older Chinese adults, personal PM2.5 was associated with higher blood pressure and greater carotid intima-media thickness and plaque area [16]. In a cross-sectional analysis of 397 women enrolled in the Household Air Pollution Intervention Network (HAPIN) trial across four countries, each 10 μg/m³ increase in personal PM2.5 was associated with 0.001 mm greater mean carotid intima-media thickness (95% CI 0–0.002), with no clear association for plaque [17].

In a crossover trial of 38 patients with coronary artery disease, air purifiers reduced indoor PM2.5 by approximately one-third and lowered a urinary marker of oxidative stress, without changes in blood pressure [18]. In patients with chronic obstructive pulmonary disease, a doubling of household PM2.5 was associated with lower heart-rate variability [19].

In contrast, the HAPIN stove trial lowered median personal PM2.5 from 84 to 24 μg/m³ but found no protective effect on gestational blood pressure [20]; this randomized evaluation is a separate report from the same multi-country platform as the carotid analysis [17]. A randomized trial in Peru likewise found no difference in systolic blood pressure after one year of liquefied petroleum gas use (difference 0.7 mmHg, 95% CI −2.1 to 3.4) [21].

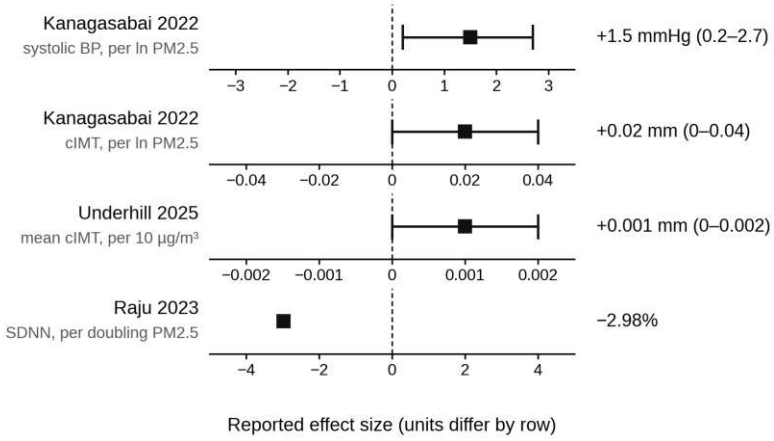


Figure 4. *Selected measured-exposure intermediate outcomes. Effect sizes are reported on different scales and cannot be pooled. Absolute effects are small. Randomized trials that substantially reduced personal PM2.5 (HAPIN and Peru) found no significant short-term blood-pressure benefit.*

3.6 Volatile organic compounds

No eligible study measured indoor volatile organic compounds as a primary exposure and reported clinical cardiac outcomes. A small number of measured-exposure studies examined arterial stiffness or related intermediate markers but did not meet criteria for clinical endpoints.

3.7 Risk of bias, certainty of evidence and small-study effects

Clinical-outcome studies were judged at moderate risk of bias under ROBINS-E, driven primarily by residual confounding (socioeconomic status, smoking, ambient air pollution, diet and healthcare access) and exposure misclassification inherent in self-reported fuel type. Outcome ascertainment was generally stronger in the large Chinese and Iranian cohorts that used death registries or active follow-up. Intermediate-outcome studies that used personal monitors had lower risk of exposure misclassification but were smaller, often cross-sectional or secondary analyses of trials, and subject to selection bias.

Using GRADE (Table 3), we rated the certainty of evidence for cardiovascular mortality as low to moderate. The starting point for observational evidence is low. Consistency of direction across the three principal analyses (China Kadoorie Biobank rural, China Kadoorie Biobank urban, and Golestan) is a potential reason for upgrading, but residual confounding, exposure misclassification, and the limited number of independent cohorts mean that an upgrade to moderate is debatable. We therefore present the rating as low to moderate rather than firmly moderate. Certainty was rated low for incident composite cardiovascular disease, myocardial infarction and stroke as separate endpoints (imprecision and residual confounding). Certainty for heart failure was very low (a single cohort with imprecise estimates). Certainty for intermediate outcomes (blood pressure, carotid thickness) was low to moderate, limited by imprecision, heterogeneity of exposure contrast, and, for some estimates, cross-sectional design.

A formal assessment of small-study effects was not undertaken because only a small number of heterogeneous estimates were available. A descriptive funnel plot is shown in Figure 5 and should be interpreted with caution.

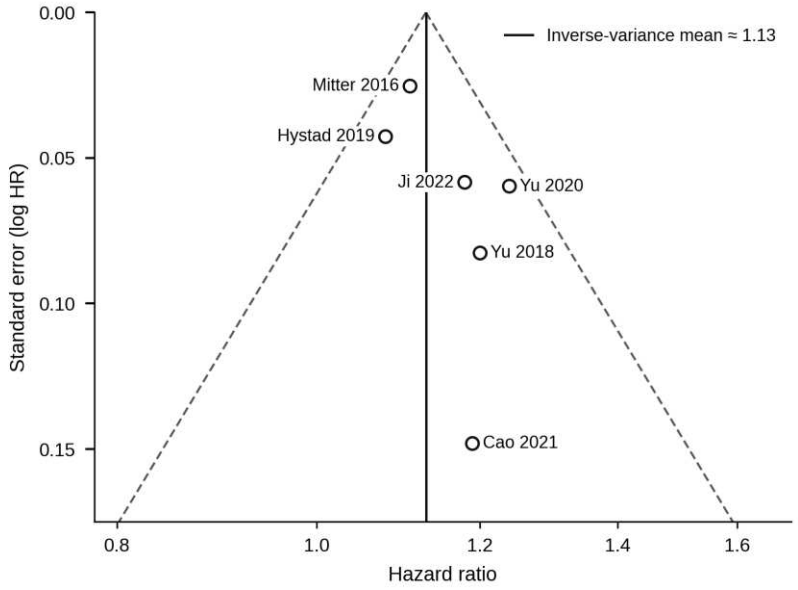


Figure 5. *Funnel plot of hazard ratios for cardiovascular outcomes associated with solid-fuel or high-pollution household exposure. Shown for descriptive purposes only; interpretation is limited by the small number of heterogeneous estimates.*

Table 1. Characteristics of clinical-outcome studies

Study	Cohort and size	Exposure (tier)	Outcome	Main result
Yu 2018 [3]	China Kadoorie Biobank, rural; 271 217	Cooking/heating fuel (3)	CV mortality	Cooking HR 1.20 (1.02–1.41)
Yu 2020 [4]	China Kadoorie Biobank, urban; 171 677	Fuel history, switching, ventilation (3)	CV mortality	Persistent solid vs clean HR 1.24 (1.10–1.39)
Mitter 2016 [5]	Golestan, Iran; 50 045	Years of kerosene/diesel (3)	CVD & IHD mortality	Per 10 y: CVD HR 1.11 (1.06–1.17); IHD 1.14 (1.06–1.21)

Hystad 2019 [2]	PURE, 11 countries; 91 350	Primary cooking fuel (3)	Composite CVD (MI, stroke, HF, CV death)	HR 1.08 (0.99–1.17); all-cause mortality 1.12 (1.04–1.21)
Cao 2021 [12]	Chinese non-smokers; ~13 500	Heating fuel (3)	Stroke; CVD events	Stroke HR 1.44 (1.00–2.08); CVD 1.19 (0.89–1.59)
Ji 2022 [13]	CHARLS, China; 13 544	Cooking fuel (3)	Non-fatal CVD	HR 1.18 (1.05–1.32); persistent use 1.38 (1.18–1.61)
Fatmi 2019 [14]	Rural Pakistan; 850 women	Biomass cooking (3)	Angina, MI, ECG CHD	No association (MI OR 1.2, 0.7–2.2)
Juntarawijit 2020 [15]	Rural Thailand	Biomass cooking (3)	CHD, hypertension	CHD OR 4.35 (0.10–18.97)

Abbreviations: CHD, coronary heart disease; CI, confidence interval; CV, cardiovascular; CVD, cardiovascular disease; HF, heart failure; HR, hazard ratio; IHD, ischemic heart disease; MI, myocardial infarction; OR, odds ratio. Tier indicates exposure fidelity (see Methods).

Table 2. Measured-exposure and intermediate-outcome studies (bridge group)

Study	Population	Exposure (tier)	Outcome	Main result
Kanagasabai 2022 [16]	753 Chinese adults 40–79 y	Personal PM2.5 (1)	BP, cIMT, plaque	Higher BP and cIMT with higher personal PM2.5
Underhill 2025 [17]	397 women, 4 countries (HAPIN)	Personal PM2.5, BC, CO (1)	cIMT, plaque	PM2.5: +0.001 mm mean cIMT per 10 µg/m³ (0–0.002)
Eom 2022 [18]	38 CAD patients	Indoor PM2.5, air purifier (1)	Oxidative stress, BP	PM2.5 ↓33.9%; oxidative marker ↓; BP unchanged
Raju 2023 [19]	85 adults with COPD	In-home PM2.5 (1)	Heart-rate variability	Doubling of PM2.5: SDNN −2.98%
Ye 2022 [20]	HAPIN pregnant women	Personal PM2.5 (1)	Gestational BP	PM2.5 84→24 µg/m³; no BP effect
Checkley 2021 [21]	180 Peruvian women	LPG stove trial	Blood pressure	SBP difference 0.7 mmHg (−2.1 to 3.4)

Abbreviations: BC, black carbon; BP, blood pressure; CAD, coronary artery disease; cIMT, carotid intima-media thickness; CO, carbon monoxide; COPD, chronic obstructive pulmonary

disease; LPG, liquefied petroleum gas; PM2.5, fine particulate matter; SDNN, standard deviation of normal-to-normal intervals; SBP, systolic blood pressure.

Table 3. Summary of risk of bias and certainty of evidence

Outcome	Studies (n)	Dominant ROBINS-E concerns	GRADE starting point & adjustments	Certainty
Cardiovascular mortality	3 principal analyses (CKB rural, CKB urban, Golestan)	Confounding; exposure misclassification	Low; possible upgrade for consistency is debatable	Low to moderate
Incident CVD / stroke	4–5 cohorts	Confounding; variable outcome ascertainment	Low; limited by imprecision	Low
Myocardial infarction (separate)	PURE only (2,363 events)	Same as above + sparse data	Low → downgraded for imprecision	Very low–low
Heart failure	PURE only (476 events)	Confounding; exposure misclassification; imprecision	Low; downgraded for imprecision	Very low
Blood pressure / cIMT (measured exposure)	Multiple small studies	Selection; limited generalizability	Low–moderate	Low to moderate

4. Discussion

4.1 Principal findings

The clinical evidence consistently indicates a modest excess of cardiovascular mortality associated with solid-fuel use, with hazard ratios of approximately 1.11–1.24 across large cohorts from China and Iran (Figure 2A) [3–5]. Evidence for myocardial infarction and especially heart failure as discrete clinical endpoints is substantially thinner. Every clinical-outcome study relied on proxy

(Tier 3) exposure. Measured-exposure studies, which are smaller and focus on intermediate outcomes, show small positive associations with carotid intima-media thickness and blood pressure (Figure 4) [16,17]. Randomized trials that achieved large reductions in household PM2.5 have not demonstrated clear short-term blood-pressure benefit [20,21].

4.2 Biological plausibility

The intermediate findings are consistent with proposed mechanisms including systemic oxidative stress, autonomic imbalance and endothelial or arterial-wall changes [16–19]. The directional consistency between proxy-based mortality studies and measured-exposure vascular studies strengthens biological plausibility. However, the two bodies of evidence do not measure the same construct: effect sizes from personal monitors were small, and a single 24-hour measurement may not capture habitual exposure [17].

4.3 Implications for practice

A brief exposure history (cooking and heating fuel, stove type, ventilation) is quick to obtain and may help contextualize risk in patients from affected communities. Because switching from solid fuel and use of ventilation were associated with lower mortality risk in large Chinese cohort analyses [3,4], advice to transition to cleaner fuel is reasonable when framed as an addition to, rather than a replacement for, established secondary-prevention measures. These observational associations do not by themselves prove that the fuel change caused the risk reduction. The null blood-pressure findings from intervention trials argue against promising short-term hemodynamic gains.

4.4 Comparison with earlier reviews

This review covers 2015–2025 and therefore updates the coronary-heart-disease review that searched to June 2015 [6]. Compared with the broader review of cardiovascular outcomes in low- and middle-income countries [7], we separate clinical from intermediate outcomes, grade exposure fidelity explicitly, and examine fuel switching and ventilation as modifiers.

4.5 Limitations

Exposure was self-reported in all clinical-outcome studies, so misclassification and residual confounding by socioeconomic status, smoking, ambient pollution, diet and healthcare access are likely. Several reports derive from the same underlying cohorts (particularly China Kadoorie Biobank); we mitigated double-counting by selecting one primary report per outcome. Designs and exposure contrasts were heterogeneous, precluding formal meta-analysis (see Figure 2). The search was restricted to English-language publications from 2015 onward, so earlier and non-English studies were not captured. The PRISMA 2020 flow (Figure 1) reports identification, screening, full-text assessment and inclusion numbers, together with grouped reasons for full-text exclusion. Publication bias cannot be excluded; the small number of heterogeneous estimates precluded a meaningful assessment of small-study effects. Indoor volatile organic compounds could not be assessed. Most clinical evidence originates from China; generalizability to sub-Saharan Africa, South Asia and Latin America remains uncertain.

4.6 Research priorities

- Prospective cohorts that combine repeated personal or household exposure measurement with adjudicated myocardial infarction and heart failure.
- Intervention trials powered for clinical cardiovascular events, with verified and sustained reductions in exposure.

- Studies of indoor volatile organic compounds in relation to clinical cardiac outcomes.
- Greater representation of African, South Asian and Latin American populations.

5. Conclusions and take-home messages

Household solid-fuel use is associated with a modest increase in cardiovascular mortality and possibly stroke, but the evidence rests almost entirely on proxy exposure and contains little information on heart failure. Switching to clean fuel and use of ventilation were associated with lower risk in large Chinese cohorts. Clinicians can incorporate a brief household-exposure history into risk counselling. Researchers should prioritize measured-exposure cohorts with adjudicated clinical cardiac events.

Declarations

Ethics and consent: This systematic review used only published data. Ethical approval and participant consent were not required.

Funding: No external funding was received for this work.

Competing interests: The authors declare no relevant conflicts of interest.

Data availability: All data analyzed are contained in this article and its supplementary files. Search strategies, extraction templates and the PRISMA checklist are provided as supplementary material.

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