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REVIEW PAPER

SYNERGISTIC RESPIRATORY RISKS IN COFFEE PROCESSING: A SYSTEMATIC REVIEW OF ALPHA-DIKETONE, CARBON MONOXIDE, AND DUST CO-EXPOSURE

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Highlights

- Coffee processing shows a synergistic risks due to carbon monoxide, diketones, and dust.
- Coffee grinding is the hazard epicenter for peak toxic gas emissions.
- Task-specific ventilation is key to control peak emissions in coffee plants.

Abstract

Modern coffee processing generates a complex, co-localized mixture of respiratory hazards, such as carbon monoxide (CO), volatile α-diketones (principally diacetyl and 2,3-pentanedione), and bio-reactive coffee dust, that challenge single-agent risk paradigms. This systematic review synthesizes quantitative exposure data, exposure-response relationships, mechanistic plausibility, and control efficacy to characterize the occupational respiratory burden associated with simultaneous exposure to these agents. A systematic search of literature (in accordance with Preferred Reporting Items for Systematic Reviews and Meta-Analyses – PRISMA 2020) was conducted choosing eligible studies reported quantitative exposure metrics (CO in ppm, α-diketones in ppb, particulate matter in mg/m^3) and respiratory health outcomes among coffee industry workers. Task-based monitoring identified grinding, flavoring, and packaging as peak emission events. Epidemiological findings included sentinel clusters of obliterative bronchiolitis, increased prevalence of obstructive spirometric abnormalities, and measurable forced expiratory volume (FEV_1) declines associated with cumulative α-diketone exposure; organic dust exposure correlated with chronic cough, dyspnea, and reduced FEV_1/forced vital capacity. Co-exposure to CO, α-diketones, and coffee dust in roasting, grinding, and packaging operations produces a substantive occupational respiratory risk profile, including both acute systemic hazards and chronic small-airway injury. Preventing occupational risk requires prioritizing engineering controls, task-specific ventilation design for peak emissions, and longitudinal exposure and health surveillance to define dose-response relationships for complex mixtures and to inform evidence-based occupational exposure limits. Med Pr Work Health Saf. 2026;77(3)

Key words: dyspnea, particulate matter, occupational exposure, carbon monoxide, coffee processing, ketones

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INTRODUCTION

Modern coffee processing creates a multi-hazard environment through the simultaneous release of carbon monoxide (CO), α-diketones, and bio-reactive dust. These complex mixtures render traditional single-hazard paradigms inadequate [1]. The National Institute for Occupational Safety and Health (NIOSH) Health Hazard Evaluations (HHEs) confirm that high-risk tasks involve concurrent exposure to all 3 contaminants, necessitating an integrated, multi-hazard analysis [1,2]. Coffee industry hazards transition from primary organic dusts and allergens to mixtures of dust, gases, and α-diketones during secondary production [3]. The recent UK Surveillance of Work-related and Occupational Respiratory Disease scheme identifies "green coffee" dust as an emerging factor in work-related respiratory disease [4]. The exposure profile begins at primary processing (green bean handling), where workers encounter high levels of organic dust, endotoxins, and allergens [5]. Cumulative organic dust exposure is linked to chronic respiratory symptoms [6] and measurable lung function impairment [7,8]. The risk profile shifts during secondary processing (roasting, grinding, packaging), where high-temperature mechanical stages release CO and α-diketones [1]. Roasting (>200°C) also generates polycyclic aromatic hydrocarbons (PAHs) and acrylamide, which are beyond the scope of this review [9]. Carbon monoxide is a pervasive toxin in coffee processing due to a dual-source mechanism: combustion emissions from roasting and the rapid off-gassing of CO trapped within beans [10,11]. Grinding fractures the beans, accelerating gas release and creating peak exposures distant from the roaster [10]. This risk can reach lethal concentrations in unventilated silos or storage tanks [10,11]. Alpha-diketones, primarily diacetyl (DA) and 2,3-pentanedione (2,3-PD), are volatile roasting by products [12] linked to bronchiolitis obliterans (BO), a severe obstructive disease caused by small airway scarring [13]. Because DA and 2,3-PD induce similar respiratory injury patterns, NIOSH categorizes them within the "α-dicarbonyl class" as a functional toxic grouping [13]. Despite NIOSH-recommended limits, evaluations consistently document exceedances [2]. This persists due to a major regulatory gap: Occupational Safety and Health Administration (OSHA) lacks specific, enforceable standards for these compounds [13,14]. This integrated multi-hazard environment suggests synergistic injury mechanisms: fine coffee dust may vector volatile α-diketones into distal airways [15,16], while CO-induced systemic hypoxia [11] may potentially compromise pulmonary repair capacity. These mechanisms collectively support a cumulative insult hypothesis and are examined mechanistically below. While literature often focuses on single agents, NIOSH evaluations confirm simultaneous exposure to all 3 during tasks like grinding [1,12], by pooling health outcome data, this review offers a focused synthesis of the risks unique to co-exposure from coffee dust, CO, and α-diketones.

The aim of this systematic review is to synthesize quantitative evidence on exposure levels, exposure-response relationships, and control efficacy for CO, α-diketones, and coffee dust among coffee workers.

METHODS

Registration and adherence to reporting guidelines

This systematic review protocol adheres to stringent methodological standards, following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020) guidelines for comprehensive reporting [17]. The protocol was not prospectively registered in a public database (e.g., PROSPERO, an international prospective register of systematic reviews). However, to ensure maximum transparency and minimize the risk of reporting bias, the full search strategy, inclusion criteria, and methodological approach are fully detailed in the following sections. Given the necessary reliance on observational epidemiological studies, such as cohort and cross-sectional studies documented in HHEs, rigorous and transparent reporting of study inclusion, exposure assessment, and data synthesis is maintained throughout this review.

Data extraction and exposure metric standardization

For comparative synthesis, all quantitative exposures were standardized to common units: CO in ppm [18], α-diketones in ppb [13], and particulate matter (PM) in mg/m^3 [19]. Data prioritized 8-hour time-weighted average (TWA) or 15-minute short-term exposure limit (STEL) for regulatory comparison. For α-diketones, a mixture index (sum of DA and PD) was calculated to account for combined exposure to structurally similar compounds, preventing underestimation of toxicological risk [13]. A systematic search of academic databases and grey literature ensured comprehensive evidence was captured, including material 2000–2025. The primary electronic databases searched were: MEDLINE (via PubMed), Scopus, and Web of Science (Core Collection).

Targeted searches for grey literature were conducted on NIOSH and OSHA websites to capture HHEs and criteria documents. The final literature search was executed on November 15, 2025. Two independent reviewers collaboratively developed the search strategy using controlled vocabulary (e.g., Medical Subject Headings [MeSH]) and free-text keywords related to the 3 core hazards and the occupational setting. A 2-concept Boolean model ensured retrieval of literature covering both hazard categories (CO, α-diketones, and coffee dust) and the industrial environment.

All records were managed using Zotero (Roy Rosenzweig Center for History and New Media, Fairfax, VA, USA), and duplicates were removed. Two independent reviewers screened titles and abstracts against pre-defined inclusion criteria, specifically studies reporting quantitative exposure levels and epidemiological data on coffee workers. Studies from chemically-related industries were included if they provided quantitative exposure-response models (e.g., for α-diketones) applicable to the coffee industry (Table 1). Data were then systematically extracted by the same 2 reviewers using a standardized form, capturing study characteristics, exposure metrics (TWA and STEL), quantitative health outcomes (e.g., forced expiratory volume [FEV_1], mean difference, odds ratios [OR]), and control efficacy rates. Any discrepancies were resolved through consensus, with a senior reviewer arbitrating unresolved cases. Study investigators were not contacted for additional data. No enforceable U.S. OSHA permissible exposure limit exists for DA or 2,3-PD, though regulations have been proposed [14]. A formal risk-of-bias assessment was conducted for the observational studies, focusing on cumulative exposures, ppm-years for α-diketones and mg/m^3-years for coffee dust, which are critical for assessing diseases like BO [13,20].

RESULTS

The study selection process is illustrated in the PRISMA 2020 flow diagram (Figure 1), which was generated using the PRISMA 2020 R package (R Foundation for Statistical Computing, Vienna, Austria) and associated web-based interface [21].

Exposure-response modeling and quantitative health outcomes

A central finding of this systematic synthesis is the routine failure of exposure controls, leading to worker exposures to CO, α-diketones, and coffee dust that far exceed occupational limits (Table 1 and 2).

Peak personal exposures confirm that CO exceedances arise from a dual-source mechanism: roasting combustion and bean off-gassing. Exhaust stack concentrations reaching 2121 ppm [22] underscore the extreme hazard of thermal generation. Routine operations create an acute risk of high-flux CO peaks. Harvey et al. [1] documented concentrations of 584 ppm, nearly triple the NIOSH ceiling limit, during flavoring and grinding tasks. Fatalities and near-fatal collapses have been reported: CO levels in coffee extract storage tanks have reached 10 000–100 000 ppm [10]. One worker was found dead with a carboxyhemoglobin (COHb) of 26%, and another collapsed in a tank containing 7500 ppm, arriving at the hospital with a COHb of 27% [10]. Recent surveillance shows dangerous CO levels extend beyond silos. At a small grinding station, a 10-second peak of 1521 ppm, exceeding the NIOSH immediately dangerous to life or health (IDLH) of 1200 ppm, identifies even small grinders as high-flux sources of acute hazard [2]. Additional evidence of acute CO risk was found where 5 production workers had personal exposures of 304–836 ppm [23,24]. Short-term tasks can lead to high CO exposures: personal levels for roaster operators peaked at 267 ppm, while area levels near a production grinder reached 338 ppm, both exceeding the NIOSH ceiling limit of 200 ppm [23,24]. Task-based monitoring highlights acute CO risk: in a Finnish facility, real-time sampling during grinder cleaning showed a peak of 141 ppm at the operator's breathing zone, exceeding the Finnish 15-minute ceiling of 100 ppm. Although full-shift TWA levels were low (e.g., 2.0 ppm), short-term peaks reached 480 ppm for the grinder and 270 ppm for the roaster, confirming significant acute risk during specific tasks [25]. Case reports and studies highlight severe systemic risks from CO overexposure. Acute CO poisoning can cause long-term harm, with large-scale data linking it to a sustained increase in ischemic heart disease (IHD) risk, even in patients aged <40 years. This risk remains significant years later, with an adjusted hazard ratio of 1.55 (95% CI: 1.27–1.89) persisting ≥6 years after the initial exposure [26]. Acute severe CO poisoning carries a significant long-term risk of delayed neurological sequelae, with exposures >5.5 h predicting later cognitive and motor deficits [27]. Carbon monoxide is also classified as a reproductive toxicant, posing fetal risk [28]. Meta-analysis of chronic low-level CO exposure in the coffee industry is limited by fragmented data, preventing an exposure-response assessment.

Table 1. Characteristics and key findings of epidemiological, exposure, and mechanistic studies on occupational hazards in the coffee industry included in the systematic review (global scope, 2003–2025)

Study	Study design and setting	Contaminant(s) assessed	Key quantitative finding/Result	Country
Exposure quantification and exceedances				
Harvey et al., 2019 [1]	NIOSH HHE: roasting and packaging	CO, α-diketones, coffee dust	peak: CO 584 ppm, DA 2127.7 ppb, PD 1444.7 ppb; TWA: DA 420.9 ppb; 100% REL exceedance	USA
Martin et al., 2025 [2]	NIOSH HHE: roasting and packaging	CO, α-diketones (DA, 2,3-PD)	peak: CO 1521 ppm (>IDLH); TWA: 86% > DA REL (max 12.5 ppb); area samples: 77.3 ppb DA (15.5 × REL), 50.5 ppb PD (5.4 × REL) at small grinder	USA
Oldenburg et al., 2009 [5]	cross-sectional: coffee haulage, silo, and decaffeinating	green coffee dust	exposure: inhalable dust >10 mg/m^3 during unloading	Germany
Bailey et al., 2020 [33]	cross-sectional and longitudinal follow-up	α-diketones (DA, 2,3-PD), CO, dust, VOCs	peak: PD 2060.3 ppb (mezzanine), CO >200 ppm (enclosures); exceedance: 98% DA/71% PD (2012) vs. 97% of samples exceeded the REL despite control implementation (2017)	USA
Sakwari et al., 2013 [7]	cross-sectional: factories	coffee dust	Arabica: GM 2.10 mg/m^3 (range 0.24–30.0); Robusta: GM 3.42 mg/m^3 (range 1.2–6.67)	Tanzania
Abaya et al., 2018 [45]	cross-sectional: primary processing	coffee dust	exceedance: 84% > OEL; GM: 12.5 mg/m^3 (machine/transport)	Ethiopia
Hawley et al., 2017 [10]	NIOSH HHE synthesis	CO, α-diketones	storage tanks: CO 7500–100 000 ppm; health: fatality (26% COHb) and near-fatal collapse (27% COHb)	USA
Gaffney et al., 2015 [12]	exposure measurement: commercial roasting	α-diketones (DA, 2,3-PD)	grinding peaks: DA 390 ppb (15.6 × STEL), PD 210 ppb (6.8 × STEL) from unflavored beans	USA
Samsidar et al., 2019 [22]	source measurement: roasting exhaust	CO	exhaust peak: CO 2121 ppm (1.77 × IDLH), confirms thermal generation during dark roasting (Liberica)	Indonesia
Martin et al., 2018 [30]	NIOSH HHE: roasting and café	α-diketones, CO, CO_2	peaks: DA 458 ppb (18.3 × STEL), PD 431 ppb (13.9 × STEL); café: CO_2 1969 ppm (ventilation failure)	USA
LeBouf et al., 2020 [48]	cross-sectional synthesis (17 facilities and 10 cafés)	α-diketones, VOCs	exceedance: 96% flavored/72% non-flavored > mixture index (sum DA + PD); TWA (GM): flavoring (34 ppb DA), packaging (27 ppb DA), grinding (26 ppb DA); café: 67% > mixture index; Barista: P95 (11.0 ppb) > REL	USA
Pengelly et al., 2019 [38]	personal and area sampling: 8 Irish roasteries	α-diketones (DA, 2,3-PD)	TWA: 40% > 20 ppb OEL; peak: 400 ppb (grinding)	Ireland
Sakwari et al., 2013 [7]	personal air sampling: Tanzanian factories	endotoxin	endotoxin (GM): Robusta 10 800 vs. Arabica 1400 EU/m^3; dust: GM 2.5 mg/m^3; peaks to 36 mg/m^3	Tanzania
The Finnish Institute of Occupational Health, 2025 [25]	occupational hygiene survey: Finnish roastery	CO, α-diketones (DA)	peak: CO 141 ppm (cleaning) > Finnish ceiling; area: DA 65.3 ppb (329% of limit) at grinder	Finland
Rim, 2019 [34]	literature review: NIOSH HHEs synthesis	coffee dust, α-diketones	unflavored grinding can reach 93 ppb DA; clinical: BO often misdiagnosed as asthma, bronchitis, or emphysema	South Korea
Fortner et al., 2020 [32]	NIOSH HHE: roasting, flavoring and packaging	CO, α-diketones (DA, 2,3-PD)	TWA: 100% > RELs; max DA 185.4 ppb (grinder), PD 279.9 ppb (packaging); peak: DA 2463 ppb, PD 2067 ppb, CO 234 ppm	USA

Bailey et al., 2015 [35]	cross-sectional and hygiene survey: U.S. roastery and flavoring	DA, 2,3-PD	peak "burst": DA 14 300 ppb, PD 18 400 ppb at open hopper hatches	USA
Cheung et al., 2024 [31]	personal sampling: New Zealand roastery	α-diketones (DA, 2,3-PD)	peak: grinding DA 27 ppb (> STEL); TWA: roaster/packager 6.6 ppb DA (> REL)	New Zealand
Boylstein et al., 2018 [23]	NIOSH HHE: roasting and packaging	α-diketones (DA, PD), CO	TWA: 100% > DA REL (max 40.5 ppb); peak: CO 836 ppm, source DA 9062 ppb during grinding	USA
LeBouf et al., 2017 [36]	NIOSH HHE: roasting and packaging	α-diketones (DA, PD), CO, VOCs	peak: DA 33.4 ppb during manual hand-blending and 37.6 ppb during grinding	USA
Harvey et al., 2018 [24]	NIOSH HHE: roasting and packaging	α-diketones (DA, PD), CO, VOCs	peak CO: 203–267 ppm on roasters, quality control/area near grinder >200 ppm; source: max DA 4505 ppb, PD 1841 ppb during transfer	USA
Health outcomes and exposure-response modeling				
Harvey et al., 2019 [1]	NIOSH HHE: roasting and packaging	α-diketones, CO	health: 1 confirmed BO case, 6% abnormal spirometry, 11% cohort with >15% FEV_1 decline/year	USA
Bailey et al., 2020 [33]	cross-sectional and longitudinal follow-up	α-diketones, green coffee, castor bean	morbidity: 5 BO cases, risk: 2.7 × obstruction, 1.6 × dyspnea vs. US pop; sensitization: 100% green coffee IgG, 1 castor bean IgE	USA
Bråtveit et al., 2021 [8]	cross-sectional: primary processing workers	organic dust, endotoxin	exposure: GM 12 mg/m^3, 84% > OEL; health: adjusted FEV_1 mean difference: –0.26 l, significantly higher OR for: cough (11.3), dyspnea (3.2), wheeze (2.4); decline: 0.9 ml/s FEV_1 per 1 mg/m^3-year	Tanzania/ Ethiopia
Park and Gilbert, 2018 [20]	longitudinal risk assessment: microwave popcorn plant (mechanistic proxy)	DA	decline: 0.40% predicted FEV_1 per ppm-year; risk: 1/1000 lifetime impairment at 2 ppb (0.002 ppm) DA	USA
Abaya et al., 2019 [42]	cross-sectional: hand pickers	organic dust	prevalence ratio: cough (3.0), dyspnea (2.5); small airways: reduction in $FEF_{25–75}$ in hand pickers	Ethiopia
Virji et al., 2022 [40]	pooled cross-sectional synthesis	α-diketones (DA, 2,3-PD)	metric: P95 most sensitive, decline 0.30% ppFEV_1, 0.25% ppFVC per 10 ppb 2,3-PD increase; impulse oscillometry: 27.5% abnormal; linked to P95 sum of DA and PD despite normal spirometry	USA
Abaya et al., 2018 [45]	cross-sectional: primary processing	coffee dust	obstructive lung disease (FEV_1/FVC < 0.70) in older workers: 27%	Ethiopia
Sakwari et al., 2011 [43]	cross-sectional: curing workers	coffee dust	prevalence ratio: cough with sputum 2.5 (23% vs. 10%), chest tightness 2.4 (27% vs. 13%)	Tanzania
Harvey et al., 2020 [46]	pooled cross-sectional: 17 U.S. facilities	α-diketones, green coffee dust, VOCs	SMRs (vs. U.S. population): wheeze (2.0), phlegm (1.9), asthma (1.4), post-hire asthma: 16%	USA
Mechanistic, source characterization, and determinants				
Martin et al., 2018 [30]	NIOSH HHE: roasting and café	α-diketones (DA, 2,3-PD)	headspace: roasted DA 1035 ppb, PD 1670 ppb; green beans: undetectable	USA
Echt et al., 2021 [37]	cross-sectional and lab emissions: craft roastery and café	α-diketones (DA, 2,3-PD), TVOCs, CO, CO_2	TWA: 71% > REL for DA; hierarchy: bagging > grinding > roasting; roast: dark emits 35 times more DA than light; surrogates: DA links to TVOC (R^2 = 0.95), not CO/CO_2	USA

Table 1. Characteristics and key findings of epidemiological, exposure, and mechanistic studies on occupational hazards in the coffee industry included in the systematic review (global scope, 2003–2025) – cont.

Study	Study design and setting	Contaminant(s) assessed	Key quantitative finding/Result	Country
Davey et al., 2022 [44]	measurement: roasteries and breweries	α-diketones, VOCs, CO, CO_2	cooling tray peaks: DA 21 ppb, PD 14 ppb; surrogates: DA shows exact temporal coincidence with particulate matter (≤ 2.5 µm) "smoke events"	Canada
LeBouf et al., 2022 [49]	exposure modeling	α-diketones (DA, 2,3-PD)	specific emission rate: fine > coarse > whole bean; modeling: near-field grinding peaks of 123.5 ppb; storage: dark roast emissions decayed, light roast increased >10 days	USA
Blackley et al., 2022 [50], Stanton et al., 2022 [55]	exposure modeling (Bayesian)	α-diketones (DA, 2,3-PD)	drivers: grinding tasks increased DA by 508% vs. roasting, flavoring tasks increased 2,3-PD by 806%, flavoring ground coffee increased risk by >300%	USA
LeBouf and Aldridge, 2019 [47]	lab modeling	CO	grinding significantly increases CO flux ($p < 0.0001$); dark roasts and fresh grounds (69.5 mg/kg × h) emit >2 times more CO than light or pre-packaged coffee (30.9 mg/kg × h)	USA
Blackley et al., 2019 [51]	NIOSH HHE	α-diketones, CO	exposures < RELs: all full-shift TWAs below limits; grinding peak: 106 ppb DA	USA
Mitigation and control efficacy				
Martin et al., 2025 [2]	NIOSH HHE: roasting and packaging	DA, 2,3-PD, CO	ventilation enclosures: reduced diketones 16 times and CO 7–12 times; fans: 5 times CO reduction	USA
LeBouf et al., 2020 [48]	exposure modeling and control analysis	α-diketones in general ventilation	standardized benchmarks: recommends adherence to ANSI/ASHRAE Standard 62.1-2019 for dilution ventilation; rates: small/medium spaces – 7.5 cfm/p + 0.12 cfm/ft², medium/large areas – 10 cfm/p + 0.18 cfm/ft²	USA
Blackley et al., 2022 [40]	Bayesian model averaging	DA, 2,3-PD	GEV reduces α-diketones by approx. 65% facility-wide, while flavoring LEV cuts downstream 2,3-PD by 38–51% in packaging and up to 82% in grinding	USA
Stanton et al., 2022 [55]	case study: HHE follow-up	α-diketones (DA, 2,3-PD)	enclosures: 95% external DA reduction despite 58% production growth; internal risk: 907.2 ppb DA "trapping effect" requires strict entry protocols	USA
Lee et al., 2023 [56]	process modification: lab study	α-diketones (total α-DCs)	Robusta pre-treatment: 3% tartaric acid and sonication reduced α-dicarbonyls by up to 58% during roasting	South Korea
U.S. EPA, 2003 [57]	thermal oxidizer	VOCs (α-diketones)	achieves 98–99.99% VOC destruction efficiency	USA

ANSI/ASHRAE – American National Standards Institute/American Society of Heating, Refrigerating and Air-Conditioning Engineers, BO – bronchiolitis obliterans, CO – carbon monoxide, COHb – carboxyhemoglobin, DA – diacetyl, $FEF_{25–75}$ – forced expiratory flow between 25% and 75% of vital capacity, FEV_1 – forced expiratory volume in 1 s, FVC – forced vital capacity, GEV – general exhaust ventilation, GM – geometric mean, HHE – health hazard evaluation, IDLH – immediately dangerous to life or health, LEV – local exhaust ventilation, NIOSH – National Institute for Occupational Safety and Health, OEL – occupational exposure limit, OR – odds ratio, P95 – 95th percentile, $ppFEV_1$ – percent of predicted FEV_1, PR – prevalence ratio, R^2 – coefficient of determination, REL – NIOSH recommended exposure limit, SMR – standardized morbidity ratio, STEL – 15-minute short-term exposure limit, TWA – 8-hour time-weighted average, TVOC – total volatile organic compounds, VOCs – volatile organic compounds, 2,3-PD/PD – 2,3-pentanedione/pentanedione.

Table 2. Occupational exposure limits for key hazardous substances in the coffee industry, including international reference values and acute exposure thresholds (global scope, 2003–2025)

Substance	Source/Compound	NIOSH REL (8-h TWA)	ACGIH TLV (8-h TWA)	Acute risk level (ceiling/IDLH/STEL)	Alternative/Lower OEL (8-h TWA)
Carbon monoxide	gas (systemic toxin)	35 ppm	25 ppm [18]	200 ppm (ceiling) [18]	20 ppm (Finnish binding OEL) [25]
				1200 ppm (IDLH) [18]	6.4 ppm (7.5 mg/m^3) (Dutch HBR-OEL) [29]
Diacetyl	α-diketone (vapor)	5 ppb [13]	10 ppb [61]	25 ppb (STEL) [13]	20 ppb (0.07 mg/m^3) (Finnish HTP-8h) [25]
2,3-pentanedione	α-diketone (vapor)	9.3 ppb [13]	under study (no TLV) [13]	31 ppb (STEL) [62]	n.a.
Coffee dust (respirable)	particulate (asthmagen)	5 mg/m^3 [19]	3 mg/m^3 (guideline) [19]	n.a.	n.a.

ACGIH – American Conference of Governmental Industrial Hygienists, HBR – health-based recommended, HBR-OEL – health-based recommended occupational exposure limit, HTP – Finnish occupational exposure limit, IDLH – immediately dangerous to life or health, NIOSH – National Institute for Occupational Safety and Health, n.a. – not applicable, OEL – occupational exposure limit, REL – recommended exposure limit, STEL – short-term exposure limit, TLV – threshold limit value, TWA – time-weighted average.

This gap is compounded by differing international standards: the Dutch Health Council's 8-hour TWA of 6.4 ppm [29] is 3.9–5.5 times lower than U.S. limits of 25–35 ppm [18].

Health Hazard Evaluations consistently document severe α-diketone exceedances despite NIOSH RELs intended to reduce BO risk [13]. Headspace analysis detected up to 1035 ppb DA and 1670 ppb 2,3-PD in freshly roasted beans, while green beans were undetectable [30]. Grinding unflavored coffee produced peaks of 390 ppb DA (15.6 × STEL) and 210 ppb 2,3-PD (6.8 × STEL) [12]. These findings were corroborated by measurements from an unflavored coffee facility in New Zealand, which documented grinding-related DA peaks of 27 ppb [31]. Flavoring operations pose particularly high risks: full-shift sampling showed universal DA REL exceedances, with peaks up to 420.9 ppb in flavoring and grinding areas, and manual flavoring generating bursts of 2127.7 ppb DA and 1444.7 ppb 2,3-PD, far above short-term limits [1]. Instantaneous monitoring revealed extreme short-term spikes, with DA and 2,3-PD reaching 9062 and 6897 ppb at the bag mouth during grinding [23,24]. Health Hazard Evaluations 2018 demonstrated that exposures extend beyond grinders, with the highest DA on a grinder operator (185.4 ppb) but the highest 2,3-PD on a packaging worker (279.9 ppb) [32]. Similarly, 86% of workers in a secondary processing facility exceeded the DA REL, with area levels near a small grinder reaching 77.3 ppb DA and 50.5 ppb 2,3-PD [2]. Industrial scaling further overwhelmed controls, with 2,3-PD peaking at 2060.3 ppb, underscoring the need to design ventilation for peak, not average, exposures [33]. At a single facility, unflavored grinding and packing averaged 93 ppb DA, while flavoring and grinding produced personal exposures up to 84 × NIOSH limits [34]. Even in unflavored production, a 15-minute air sample taken at the open hatch of a mezzanine hopper above an active grinding/packaging line measured 14 300 ppb DA (>500 × STEL) and 18 400 ppb 2,3-PD, levels to which workers are briefly exposed when opening the hatch to check coffee levels [35]. Spikes up to 4505 ppb DA and 1841 ppb 2,3-PD occur when grinding fresh coffee, among the highest in unflavored production [23,24]. A NIOSH HHE and Technical Assistance (HETA 2016) [30] identified freshly roasted beans as the primary source, with headspace concentrations up to 1035 ppb DA and 1670 ppb 2,3-PD; grinding generated short-term peaks of 458 ppb and 431 ppb, respectively, substantially elevating full-shift exposures [30]. At a facility, hand-blending roasted beans produced DA peaks of 33.4 ppb, comparable to active grinding (37.6 ppb) [36]. Field studies confirm exposure epicenters extend beyond grinders. In craft roasteries, 71% of personal DA samples exceeded the REL, with highest levels at bagging, followed by grinding and roasting [37]. A cross-sectional analysis of 17 facilities revealed that 96% of flavored and 72% of non-flavored production operations exceeded the combined mixture index for DA and 2,3-PD [38]. The highest exposures were documented among workers in flavoring, packaging, and grinding roles [39]. Furthermore, 67% of café samples exceeded these thresholds, with barista 95th percentile DA levels reaching 11 ppb [37]. European data align with these findings: Finnish samples reached 65.3 ppb near grinders with moderate TWAs across tasks [25], and Irish facilities reported 40% of full-shift samples

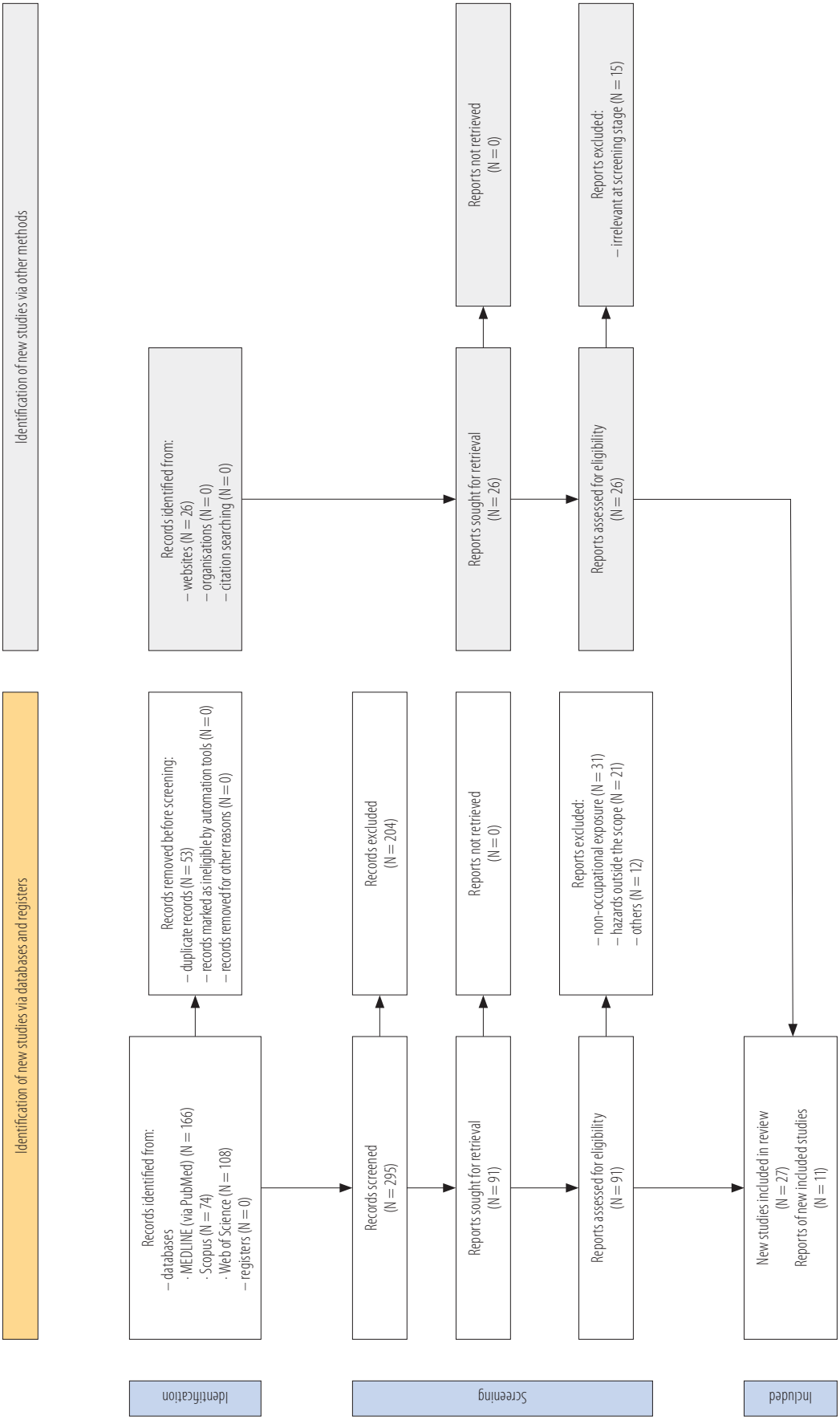

Figure 1. Flow diagram of study selection according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020) guidelines in a systematic review of occupational exposure to carbon monoxide, α-diketones, and coffee dust in the coffee industry

above the EU OEL, with grinding peaks up to 400 ppb, confirming grinding of warm, freshly roasted beans as a peak-emission event [39].

According to NIOSH, exposure–response analyses link cumulative α-diketone exposure to lung function decline and BO [13,20]. This is exemplified by a sentinel cluster of 5 BO cases from a single U.S. coffee facility, where current workers also showed markedly elevated morbidity, 2.7-fold higher spirometric obstruction and 1.6-fold higher dyspnea prevalence than the U.S. population after adjustment for age, sex, and smoking [33]. Similar findings have been reported elsewhere: a roasting and flavoring worker was diagnosed with BO, and 11% of coworkers lost ≥15% lung function within 1 year, exceeding expected age-related decline [1]. Dose–response evidence further supports causality. In a DA-exposed workforce from a microwave popcorn plant, percent predicted FEV_1 declined 0.40%/ppm-year, with chronic obstructive pulmonary disease mortality 5 times higher than expected; modeled risk estimates suggest measurable impairment at concentrations as low as 2 ppb, likely underestimated due to survivor bias [20]. Subclinical disease is common: exposed workers show reduced mid-expiratory (forced expiratory flow at 25–75% of forced vital capacity [FEF_{25-75}%]) despite normal FEV_1/forced vital capacity (FVC) ratios [35]. A pooled analysis of 384 workers found peak exposures to be the strongest predictors of damage, with each 10 ppb increase in peak 2,3-PD associated with a 0.30% predicted FEV_1 (ppFEV_1) decline, and small-airway abnormalities detected by impulse oscillometry in 27.5% of workers with otherwise normal spirometry [40]. A healthy-worker survivor effect was evident, indicating cross-sectional studies likely underestimate true risk [40].

Concerning the PM, analysis of 16 East African factories revealed extreme exposure: 12 mg/m³ mean dust (with 84% of samples exceeding limits) [8,41]. Exposed workers suffered respiratory deficits, reductions of 0.26 l (FEV_1) and 0.21 l (FVC), with obstruction prevalence 7 times higher than controls (6.3% vs. 0.9%) [8]. Key clinical risks included chronic cough (OR 11.3) and breathlessness (OR 3.2), with FEV_1 declining by 0.9 ml/s per cumulative mg/m³-year [8]. Ethiopian female hand pickers were 3 times more likely to have chronic cough (prevalence ratio [PR] 3.0) than unexposed controls and showed small airway impairment (FEF_{25-75}%) [42]. Sakwari et al. [43] reported a significantly higher prevalence of cough with sputum in coffee workers than in controls (23% vs. 10%; PR 2.5, 95% CI: 1.0–5.9) and chest tightness (27% vs. 13%; PR 2.4, 95% CI: 1.1–5.2). Seasonal peaks in Tanzania reached 36 mg/m³, 7 times the NIOSH limit [39]. Grinding and cleanup create multi-phase hazards by co-locating respirable dust, CO, and α-diketone vapors, especially during grinder cleaning when dust re-entrainment and gas off-gassing occur together [12,41]; the cooling tray is characterized by episodic "smoke events" where DA bursts correlate exactly with PM (≤2.5 μm) [44]. In 764 female workers, organic dust (coffee, tea, spices, grains) caused acute cross-shift declines of 9.9% (FEV_1) and 3.7% (FVC), indicating immediate airway hyper-responsiveness [6]. Endotoxin exposure in Robusta machine rooms reached 10 800 EU/m³ geometric mean (GM), exceeding the recommended health limit of 90 EU/m³ by >100-fold and contributing to increased systemic inflammation and respiratory morbidity [39]. Workers handling Robusta beans had higher dust exposure (3.42 vs. 2.10 mg/m³), more chronic cough, and more asthma symptoms (38% vs. 18%, OR 3.5). Greater cumulative dust exposure was also linked to reduced FEV_1/FVC [7]. Coffee workers showed reduced FEV_1 and FVC, suggesting mixed lung impairment; 27% of the oldest had obstructive disease (FEV_1/FVC <0.70) [45]. Beyond respiratory effects, Oldenburg et al. [5] reported that inhalable dust levels >10 mg/m³ were associated with increased erythema and rhino-conjunctivitis.

To conclude, the 17-facility synthesis demonstrates a significant occupational respiratory burden. Coffee workers were 2 times more likely than the general U.S. population to experience wheeze (standardized morbidity ratio [SMR] 2.0) and chronic phlegm (SMR 1.9). Notably, the prevalence of current asthma was 1.4 times the national average, with 16% of these cases diagnosed following the commencement of coffee-related employment [46].

Hazard co-localization, mechanistic plausibility, and critical gaps

This review highlights the grinding task as a key hazard point. This process serves as an epicenter for simultaneous peak exposures, generating all 3 primary hazards: CO from accelerated off-gassing, with concentrations reaching up to 1521 ppm [2]; α-diketones, with peak instantaneous source samples recorded during grinding, with DA and 2,3-PD reaching 9062 ppb and 6897 ppb [23,24]. Gaffney et al. [12] confirmed peak emissions during grinding, with respirable dust (up to 1.7 mg/m³) and α-diketone levels exceeding

those during roasting. Emissions modeling identifies dark-roast cycles as peak-risk periods, releasing up to 35 × higer DA emission (per g) than light roasts [37]. Fresh, dark roasts pose a higher toxicological burden, as CO emission factors decline by approx. 50% within 7 days of package opening [47]. Grinding further amplifies risk by fracturing bean pore structure and increasing the CO-diffusing surface area approx. 9-fold [47]. Instantaneous source measurements confirm that ground coffee, even in non-flavored facilities, is a primary emission source, exhibiting geometric mean concentrations of 488 ppb for DA and 251 ppb for 2,3-PD. This flux is driven by mechanical grinding, which increases bean surface area and accelerates the off-gassing of roasting-generated volatile organic compounds (VOCs) [48]. However, the multi-hazard environment is not limited to grinding; it encompasses a multi-phase exposure profile where thermal combustion during roasting [22], chemical "bursts" during manual flavoring [1], and gas entrapment release during packaging [31,36], converge with high-flux organic dust levels [8,39], to create a cumulative respiratory and systemic burden. The geographical distribution of the included studies reveals a distinct bifurcation of risk profiles based on the stage of production. Studies from East Africa (Ethiopia, Tanzania) predominantly characterize the primary processing stage, where the burden is defined by extreme organic dust and endotoxin concentrations, often exceeding international limits. Conversely, data from North America, Europe, Asia and Oceania focus almost exclusively on secondary processing. In these regions, the risk shifts toward volatile chemical hazards and combustion-related gases; specifically, CO emerges as a critical secondary-stage hazard due to thermal generation during roasting and high-flux off-gassing during grinding. This global disparity suggests that while the "coffee hazard" is universal, the specific pathological driver is highly dependent on the regional industrial infrastructure. This regional bifurcation carries significant implications for the global burden of occupational disease. In primary processing centers, the pathology is dominated by high-mass lung burdens of organic dust and endotoxins, leading to classic obstructive and restrictive deficits. Conversely, in the secondary processing hubs, the risk profile is dominated by the sub-clinical, irreversible "small-airway" injury characteristic of volatile α-diketones. This suggests that a "one-size-fits-all" international surveillance strategy is inadequate; instead, occupational health monitoring must be calibrated to the specific stage of the global coffee supply chain. International policy should prioritize dust suppression and endotoxin monitoring in primary regions, while secondary processing nations must pivot toward high-resolution VOC monitoring and peak-emission engineering controls.

LeBouf et al. [49] quantified that fine grinding maximizes DA flux (specific emission rate: 3.60 mg × kg^{-1} × h^{-1}, predicting near-field peaks of 123.5 ppb. This modeling confirms that exposure is driven by particle size and proximity, noting that while dark roast emissions decay during storage, light roast emissions increase >10 days [49]. A large-scale Bayesian analysis of 17 facilities (N = 606) established grinding as the primary exposure driver, associated with a 508% increase in DA concentrations compared to roasting, while flavoring tasks drove the highest relative increases for 2,3-PD at 806% [49]. In contrast, a NIOSH HHE [51] establishes a baseline showing that full-shift exposures can remain below safety limits despite grinding peaks of 106 ppb, identifying sporadic task frequency and ventilation as critical risk moderators. For example ceiling fans can spread α-diketones facility-wide beyond source zones, such as green bean storage, facilitating facility-wide background contamination [1]. Multiple regression modeling identified the sum of open storage sources, flavoring of ground coffee, and high production volume (>10 000 lbs/day) as consistent predictors of α-diketone overexposure [50].

A central hypothesis of this review is the particulate carrier effect, where respirable coffee dust may serve as a vector for α-diketones to reach the susceptible small airway region [16]. While this hypothesis is mechanistically plausible, it remains empirically unvalidated and should be distinguished from the evidence-based findings presented elsewhere in this review. Evidence supporting the biological plausibility of this "Trojan Horse" effect is derived from the physicochemical properties of coffee matter. While these properties were characterized in spent grounds, Kim and Kim [15] demonstrated that coffee grounds possess a high specific surface area (approx. 7.5 m^2 × g^{-1}) and a density of reactive functional groups, specifically polyphenols and carboxyls, which enhance adsorption capacity. Additionally, this hypothesis is supported by real-time proton-transfer reaction time-of-flight mass spectrometry identifies cooling tray "smoke events" as hazard hotspots, showing a temporal correlation between α-diketone bursts and $PM_{2.5}$ levels [44]. However, critical empirical evidence remains absent. Direct experimental validation of gas-phase adsorption of volatile α-diketones onto respirable coffee dust particles has not been demonstrated.

Similarly, *in vivo* or *in vitro* evidence that dust-bound α-diketones penetrate distal airways more effectively than gas-phase molecules alone has not been established. Therefore, while the "Trojan Horse" mechanism is biologically plausible based on physicochemical reasoning, it constitutes a speculative framework requiring targeted mechanistic studies for validation. The inclusion of this hypothesis should not be interpreted as evidence of its occurrence in occupational settings. Because α-diketones induce direct epithelial fibrosis [13] while organic dust is linked to chronic respiratory irritation [8], this synergy suggests a biologically plausible mechanism to accelerate the onset of fixed obstructive lung disease, resulting in a more rapid FEV_1 reduction than exposure to either agent alone. At the same time, CO exposure, while primarily systemic, induces tissue hypoxia [11] and cardiovascular stress [26]. This generalized stress is the primary driver of the systemic impairment co-factor theory: it compromises the body's repair mechanisms or compensation capacity for the severe, localized pulmonary injury caused by diketones and dust. The documented long-term elevation of IHD risk after acute CO exposure [27], coupled with persistent neurocognitive sequelae [27], confirms that CO induces systemic stress that could influence chronic inflammatory and fibrotic lung outcomes. The impairment scenario complexity is amplified by biological hazards, too; mycotoxins (e.g., Ochratoxin A) present in organic dust introduce a secondary systemic toxic load that may compromise general health [52]. Overlapping clinical features frequently lead to BO being misdiagnosed as asthma, chronic bronchitis, or emphysema, obscuring occupational disease attribution [34]. Histopathology in a 37-year-old coffee worker showed lung injury beyond classic BO, suggesting α-diketones can cause a broader airway-centric disease spectrum [53]. This diagnostic gap is highlighted by a case in India involving a worker with a 10-year history of respiratory symptoms who was managed for bronchial asthma before BO was finally suspected [54].

Despite compelling quantitative evidence, a major limitation of this study concerns the chronic outcomes related to coffee industry hazards: it relies on cross-sectional data, primarily from NIOSH HHE reports [3], whose interpretation is subject to significant methodological challenges. The primary biases identified are: selection bias, resulting from the Healthy Worker Effect or survivor bias [20,40], and non-differential exposure misclassification, stemming from the reliance on short-term air samples to estimate chronic cumulative dose [3]. Critically, these methodological errors inherently dilute the apparent strength of the exposure-response relationship [20].

This review likely underestimates the cumulative toxicological burden by omitting biological risks and secondary chemical pollutants. Specifically, Robusta processing exhibits >100-fold endotoxin exceedances [39] and systemic Ochratoxin A toxicity [52], while excluded thermally-generated PAHs and acrylamide [9] suggest higher actual inflammatory stress. Key research gaps include the absence of longitudinal cohorts tracking cumulative exposure (ppm-years and mg/m^3-years) against annual FEV_1 decline and a lack of inhalation studies validating the particulate carrier hypothesis. Such robust mechanistic data are essential to develop the precise dose-response models required for setting scientifically defensible OELs for these synergistic mixtures.

Integrated hazard mitigation and control implementation

Effective hazard mitigation must follow the hierarchy of controls, prioritizing source-point engineering to abate multi-phase emissions. Local exhaust ventilation (LEV) is essential at high-flux points: roasters for thermal CO [23] and grinding/packaging stations for simultaneous CO and α-diketone off-gassing [2,49,50]. Modeling confirms that general exhaust ventilation (GEV) provides approx. 65% facility-wide reduction for both DA and 2,3-PD, while targeted LEV during flavoring significantly reduces downstream 2,3-PD exposures, by 38–51% during packaging and up to 82% during grinding [50]. Replacing open storage with closed containers is a high-impact, low-cost way to reduce ambient background concentrations of α-diketones [50]. Furthermore, increasing physical distance between machines limits cumulative exposure from nearby sources, while ensuring that automated processes are paired with dedicated ventilation addresses the higher emission volumes typical of large-scale facilities [50]. Local exhaust ventilation design is critical. Standard extraction hoods may fail due to limited suction range; a Finnish report (FIOH 2025) [25] recommends a "push-pull" configuration, using a 10 m/s air jet to blow CO away from the breathing zone into the exhaust.

Furthermore, ventilated enclosures outperform administrative measures, offering 16-fold DA and 7–12-fold CO reductions, while enclosures are preferred long term, temporary fans at grinding stations can cut average CO by fivefold [2]. These systems keep risk low regardless of production volume, maintaining a 92–95% reduction

in external hazards under standard conditions and sustaining a 75–84% reduction even during a 349% surge in throughput [55]. This "halo effect" extends facility-wide, reducing concentrations in shipping areas by approx. 60% [55]. However, a "trapping effect" within enclosures can concentrate DA to 907.2 ppb (180 × REL), necessitating strict "purge" protocols and mandatory organic vapor personal protective equipment during maintenance [55]. Primary prevention through process modification also shows promise. Pre-treating Robusta beans with 3% tartaric acid and sonication diverts Maillard reaction pathways, reducing total α-dicarbonyl formation (including DA, 2,3-PD) by <58% [56]. This approach demonstrates potential to mitigate the entire α-dicarbonyl class during roasting.

Complementary strategies include maintaining production areas under negative pressure [1] and ensuring dilution ventilation, 7.5–10 cfm/person plus area-based flow rates, in accordance with American National Standards Institute (ANSI)/American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) Standard 62.1-2019 [48]. Continuous rooftop air-handling and improved seals (e.g., door sweeps) are necessary to prevent contaminant migration into non-production areas like cafés, where CO_2 levels can otherwise exceed 1900 ppm, demonstrating ventilation inadequacy [30]. Multi-phase emissions can be managed via thermal oxidizers, achieving up to 99.99% VOC and 98% PM_{10} destruction [57]. For monitoring, real-time total VOC (TVOC) sensors serve as reliable DA surrogates ($R^2 = 0.95$), whereas CO and CO_2 sensors are poor indicators of α-diketone risk [37].

Total VOC environmental monitoring is commonly conducted using photoionization detector (PID) sensors integrated into fixed monitors or networked online platforms. These systems provide continuous, low-cost, high-resolution detection of transient emission spikes, making them well suited for real-time surveillance. However, PID sensors lack chemical speciation and exhibit compound-dependent responses based on ionization potential, which limits their interpretability when individual analytes must be identified or quantified.

In contrast, analytical methods such as gas chromatography-mass spectrometry offer definitive chemical identification and accurate quantification of volatile compounds. These methods, however, require time-integrated sampling, laboratory infrastructure, and substantially higher operational costs, making them less suitable for continuous monitoring. Carbon monoxide monitoring is typically accomplished using electrochemical sensors incorporated into commercially available industrial detectors (e.g., Honeywell BW series, Dräger PAC series, Industrial Scientific Tango), which provide reliable, real-time measurements appropriate for occupational environments.

Together, these approaches support a tiered monitoring framework that balances continuous environmental surveillance with targeted chemical confirmation. Although personal real-time monitoring of α-diketones and other VOCs would be ideal, current technologies, such as portable Fourier transform infrared spectrometers, are designed for area sampling and are not suitable for personal use. Advances in sensor technology may enable future deployment of wearable, real-time monitors capable of capturing instantaneous, short-duration, and full-shift exposures using a single sampler, thereby significantly reducing monitoring burden. Ideally, such systems would simultaneously measure α-diketones, VOCs, dust, and CO.

Mitigation must also address potential castor bean (CB) contamination; data from dockworkers link respiratory symptoms to handling green coffee sacks, with ≥1 case of sensitization specifically tied to CB-contaminated bags [58]. This is corroborated with 100% of workers (N = 60) green coffee immunoglobulin G (IgG) prevalence and sentinel CB immunoglobulin E (IgE) sensitization in processing cohorts [33]. In other studies, cross-sectional analyses identified CB–specific IgE sensitization in 17% of workers [5].

Administrative policies must mandate respiratory protection programs in accordance with OSHA 29 Code of Federal Regulation (CFR) 1910.134, including training and annual fit-testing [59]. Until engineering controls achieve REL compliance, workers require NIOSH-approved half-mask respirators with organic vapor and particulate cartridges for cleaning and non-routine tasks, especially during periods of industrial scaling that overwhelm existing ventilation [33]. Furthermore, strict confined space protocols are vital for silos, where CO levels can reach 100 000 ppm [10]. Per Finnish regulations, employers should also maintain a 5-year registry of CO-exposed personnel [25]. A mandatory medical monitoring program is essential for detecting sub-clinical damage. This should incorporate annual symptom questionnaires, COHb biomonitoring [10], and longitudinal spirometry that prioritizes annual FEV_1 decline rates over static population norms [60]. Finally, clinical findings must be paired with post-implementation air sampling to verify that controls maintain safety as industrial throughput scales [33]. This review has some limitations that should be considered when interpreting the findings. The studies included

showed heterogeneous methodological quality and variable risks of bias. These issues were mainly related to observational study designs, limited sample sizes, incomplete control of confounding factors, and clinical and methodological differences across study populations, outcomes, and measurement tools. Overall, these factors reduced the comparability of results and, in some cases, limited the feasibility of robust quantitative syntheses. Most of the available evidence derives from cross-sectional studies and HHEs. Although these sources provide reliable quantitative data and high-resolution exposure measurements in real-world occupational settings, they do not allow a comprehensive characterisation of long-term cumulative exposures. In addition, the potential influence of the healthy worker survivor effect may have led to an underestimation of risks associated with chronic exposures, particularly in the context of multi-hazard exposure mixtures. Taking together, these limitations do not undermine the main conclusions of the review but support a cautious interpretation of the findings and highlight the need for future, methodologically robust longitudinal studies.

CONCLUSIONS

This systematic review confirms coffee workers face severe multi-hazard risks from co-exposure to CO, α-diketones, and dust. Grinding is the established hazard epicenter, generating peak exposures that routinely exceed safety limits [32,50]. This profile correlates with measurable damage: α-diketones are linked to FEV_1 declines indicative of BO [20,40], while organic dust is associated with a mean FEV_1 deficit of –0.26 l [8]. Systemically, CO poses lethal risks in confined spaces [10] and long-term risks for IHD [26] and neurological sequelae [27]. Protecting this workforce requires integrated engineering controls and primary prevention, such as green bean pre-treatment [56]. Regulatory agencies must prioritize longitudinal research to quantify dose-response relationships and validate synergistic effects to establish defensible health-based exposure limits for these complex mixtures.

AUTHOR CONTRIBUTIONS

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REFERENCES

1. Harvey RR, Blackley BH, Martin SB, Stanton ML, King BA. Evaluation of exposures and respiratory health at a coffee roasting and packaging facility. NIOSH HHE Report No. 2016-0164-3341. Cincinnati: National Institute for Occupational Safety and Health; 2019.
2. Martin SB, Stanton ML, Beaty M, Blackley BH, Hines S. Evaluation of exposures and health concerns at a coffee roasting and packaging facility. NIOSH HHE Report No. 2023-0027-3417. Cincinnati: National Institute for Occupational Safety and Health; 2025.
3. Virji MA, Cummings KJ, Cox-Ganser JM. Editorial: Investigating exposures and respiratory health in coffee workers. Front Public Health. 2022;10:1026430. https://doi.org/10.3389/fpubh.2022.1026430.
4. Zhou AY, Seed M, Carder M, Money A, Van Tongeren M, Agius R. Sentinel approach to detect emerging causes of work-related respiratory diseases. Occup Med Oxf Engl. 2020;70(1):52–9. https://doi.org/10.1093/occmed/kqz159.
5. Oldenburg M, Bittner C, Baur X. Health risks due to coffee dust. Chest. 2009;136(2):536–44. https://doi.org/10.1378/chest.08-1965.
6. Žuškin E, Mustajbegović J, Schachter EN, Kern J, Macan J, Ebling Z. Respiratory function and immunological status in paper-recycling workers. J Occup Environ Med. 1998;40(11):986–93. https://doi.org/10.1097/00043764-199811000-00009.
7. Sakwari G, Mamuya SHD, Bråtveit M, Moen BE. Respiratory symptoms, exhaled nitric oxide, and lung function among workers in Tanzanian coffee factories. J Occup Environ Med. 2013;55(5):544–51. https://doi.org/10.1097/JOM.0b013e318285f453.
8. Bråtveit M, Abaya SW, Sakwari G, Moen BE. Dust exposure and respiratory health among workers in primary coffee processing factories in Tanzania and Ethiopia. Front Public Health. 2021;9:730201. https://doi.org/10.3389/fpubh.2021.730201.
9. Kung HC, Hung CH, Lin KH, Huang BW, Cheruiyot NK, Chang-Chien GP. Fast and slow roasting of alcohol- and honey-infused coffee blends: a comparative study of PAH and N-PAH emissions. Aerosol Air Qual Res. 2024;24(2):230221. https://doi.org/10.4209/aaqr.230221.
10. Hawley B, Cox-Ganser JM, Cummings KJ. Carbon monoxide exposure in workplaces, including coffee processing

facilities. Am J Respir Crit Care Med. 2017;196(8):1080–1. https://doi.org/10.1164/rccm.201703-0513LE.
11. Rose JJ, Wang L, Xu Q, McTiernan CF, Shiva S, Tejero J, et al. Carbon monoxide poisoning: pathogenesis, management, and future directions of therapy. Am J Respir Crit Care Med. 2017;195(5):596–606. https://doi.org/10.1164/rccm.201606-1275CI.
12. Gaffney SH, Abelmann A, Pierce JS, Glynn ME, Henshaw JL, McCarthy LA, et al. Naturally occurring diacetyl and 2,3-pentanedione concentrations associated with roasting and grinding unflavored coffee beans in a commercial setting. Toxicol Rep. 2015;2:1171–81. https://doi.org/10.1016/j.toxrep.2015.08.003.
13. National Institute for Occupational Safety and Health. Criteria for a recommended standard: occupational exposure to diacetyl and 2,3-pentanedione (Publication No. 2016-111). Cincinnati: NIOSH; 2016.
14. Occupational Safety and Health Administration. Flavorings-related lung disease – standards [Internet]. Washington: United STATES Department of Labor, OSHA, 2016 [cited 2025 Dec 20]. Available from: https://www.osha.gov/flavorings-related-lung-disease/standards.
15. Kim MS, Kim JG. Adsorption characteristics of spent coffee grounds as an alternative adsorbent for cadmium in solution. Environments. 2020;7(4):24. https://doi.org/10.3390/environments7040024.
16. Fransen LFH, Leonard MO. Small airway susceptibility to chemical and particle injury. Respiration. 2022;101(3):321–33. https://doi.org/10.1159/000519344.
17. Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ. 2021;372: n71. https://doi.org/10.1136/bmj.n71.
18. National Institute for Occupational Safety and Health. NIOSH pocket guide to chemical hazards: Carbon monoxide [Internet]. Cincinnati: NIOSH; 2019 [cited 2025 Dec 21]. Available from: https://www.cdc.gov/niosh/npg/npgd0105.html.
19. Occupational Safety and Health Administration. Permissible exposure limits – OSHA annotated Table Z-1 [Internet]. Washington: U.S. Department of Labor, OSHA [cited 2025 Dec 21]. Available from: https://www.osha.gov/annotated-pels/table-z-1.
20. Park RM, Gilbert SJ. Pulmonary impairment and risk assessment in a diacetyl-exposed population: microwave popcorn workers. J Occup Environ Med. 2018;60(6):496–506. https://doi.org/10.1097/JOM.0000000000001303.
21. Haddaway NR, Page MJ, Pritchard CC, McGuinness LA. PRISMA 2020: an R package and Shiny app for producing PRISMA 2020-compliant flow diagrams, with interactivity for optimised digital transparency and Open Synthesis. Campbell Syst Rev. 2022;18(2):e1230. https://doi.org/10.1002/cl2.1230.
22. Samsidar, Sawitri KN, Purbakawaca R, Ridho M, Heriyanti, Marlinda L, et al. Integration of gas sensors as an exhaust gas monitoring device from green roasting process of various coffee beans. J Phys Conf Ser. 2019;1282(1):012024. https://doi.org/10.1088/1742-6596/1282/1/012024.
23. Boylstein R, McClelland T, White S. Evaluation of exposures and respiratory health at a coffee roasting and packaging facility. HHE report No. HETA-2016-0141-3329 [Internet]. Cincinnati: National Institute for Occupational Safety and Health [cited 2026 Jan 9]. Available from: https://www.cdc.gov/niosh/hhe/reports/pdfs/2016-0141-3329.pdf
24. Harvey RR, Hawley B, Martin SB Jr. Evaluation of exposures and respiratory health at a coffee roasting and packaging facility. HHE report No. HETA-2016-0144-3305 [Internet]. Cincinnati: National Institute for Occupational Safety and Health; 2018 [cited 2026 Jan 9]. Available from: https://www.cdc.gov/niosh/hhe/reports/pdfs/2016-0144-3305.pdf.
25. The Finnish Institute of Occupational Health. Unpublished report/statement TYHYG 2025, TTL24-06023; 2025.
26. Bahng Y, Baek K, Park JT, Choi WJ, Kwak K. Carbon monoxide poisoning and developing ischemic heart disease: a nationwide population-based nested case-control study. Toxics. 2021;9(10):239. https://doi.org/10.3390/toxics9100239.
27. Gao X, Wei W, Yang GD. Clinical factors for delayed neuropsychiatric sequelae from acute carbon monoxide poisoning: a retrospective study. Front Med. 2024;11:1333197. https://doi.org/10.3389/fmed.2024.1333197.
28. Place E, Wareing H, Herigstad M. Carbon monoxide exposure in pregnant women in the UK. BMC Pregnancy Childbirth. 2025;25:1063. https://doi.org/10.1186/s12884-025-08126-6.
29. Health Council of the Netherlands. Carbon monoxide: health-based recommended occupational exposure limit. Advisory Report No. 2024/09 [Internet]. The Hague: Health Council of the Netherlands; 2024 [cited 2025 Dec 27]. Available from: https://www.healthcouncil.nl/documents/2024/09/09/carbon-monoxide.
30. Martin SB Jr, LeBouf RF, Stanton ML, Bailey RL. Evaluation of exposures and respiratory health at a coffee roasting and packaging facility and associated café. HHE report No. HETA-2016-0067-3313 [Internet]. Cincitnnati: National Institute for Occupational Safety and Health; 2018 [cited 2025 Dec 21]. Available from: https://www.cdc.gov/niosh/hhe/reports/pdfs/2016-0067-3313.pdf.
31. Cheung K, Whitelaw J, Apthorpe L. Exposures and health effects of diacetyl and 2,3-pentanedione in a coffee pro-

cessing facility. N Z J Health Saf Pract. 2024;1(1). https://doi.org/10.26686/nzjhsp.v1i1.8679.
32. Fortner AR, Beaty MC. Evaluation of exposures at a coffee roasting, flavoring, and packaging facility. HHE report No. 2018-0134-3373 [Internet]. Cincinnati: National Institute for Occupational Safety and Health; 2020 [cited 2025 Dec 21]. Available from: https://www.cdc.gov/niosh/hhe/reports/pdfs/2018-0134-3373.pdf.
33. Bailey RL, Duling MG, Martin SB Jr, Stanton ML, McClelland TL, LeBouf RF, et al. Evaluation of exposures and respiratory health at a coffee roasting, flavoring, and packaging facility. HHE report No. 2012-0170-3372 [Internet]. Cincinnati: National Institute for Occupational Safety and Health; 2020 [cited 2025 Dec 23]. Available from: https://www.cdc.gov/niosh/hhe/reports/pdfs/2012-0170-3372.pdf.
34. Rim K. Coffee roasters and their occupational lung disease: a literature review. Toxicol Environ Health Sci. 2019;11(3):175–84. https://doi.org/10.1007/s13530-019-0403-7.
35. Bailey RL, Cox-Ganser JM, Duling MG, LeBouf RF, Martin SBJ, Bledsoe TA, et al. Respiratory morbidity in a coffee processing workplace with sentinel obliterative bronchiolitis cases. Am J Ind Med. 2015;58(12):1235–45. https://doi.org/10.1002/ajim.22533.
36. LeBouf RF, Martin SB Jr, Mugford C, Stanton ML, Bailey RL. Evaluation of exposures and respiratory health at a coffee roasting and packaging facility. HHE report No. HETA-2015-0082-3287 [Internet]. Cincinnati: National Institute for Occupational Safety and Health; 2017 [cited 2026 Jan 9]. Available from: https://www.cdc.gov/niosh/hhe/reports/pdfs/2015-0082-3287.pdf.
37. Echt H, Dittmore M, Coker M, Beaudet N, Croteau GA, Cohen M, et al. Characterization of naturally occurring alpha-diketone emissions and exposures at a coffee roasting facility and associated retail café. Ann Work Expo Health. 2021;65(6):715–26. https://doi.org/10.1093/annweh/wxaa148.
38. Pengelly I, O'Shea H, Smith G, Coggins MA. Measurement of diacetyl and 2,3-pentanedione in the coffee industry using thermal desorption tubes and gas chromatography-mass spectrometry. Ann Work Expo Health. 2019;63(4):415–25. https://doi.org/10.1093/annweh/wxz015.
39. Sakwari G, Mamuya SHD, Bråtveit M, Larsson L, Pehrson C, Moen BE. Personal exposure to dust and endotoxin in Robusta and Arabica coffee processing factories in Tanzania. Ann Occup Hyg. 2013;57(2):173–83. https://doi.org/10.1093/annhyg/mes064.
40. Virji MA, Fechter-Leggett ED, Groth CP, Liang X, Blackley BH, Stanton ML, et al. Decrements in lung function and respiratory abnormalities associated with exposure to diacetyl and 2,3-pentanedione in coffee production workers. Front Public Health. 2022;10:966374. https://doi.org/10.3389/fpubh.2022.966374.
41. Abaya SW, Bråtveit M, Deressa W, Kumie A, Moen BE. Personal dust exposure and its determinants among workers in primary coffee processing in Ethiopia. Ann Work Expo Health. 2018;62(9):1087–95. https://doi.org/10.1093/annweh/wxy079.
42. Abaya SW, Bråtveit M, Deressa W, Kumie A, Moen BE. Respiratory health among hand pickers in primary coffee-processing factories of Ethiopia. J Occup Environ Med. 2019;61(7):565–71. https://doi.org/10.1097/JOM.0000000000001613.
43. Sakwari G, Bråtveit M, Mamuya SHD, Moen BE. Dust exposure and chronic respiratory symptoms among coffee curing workers in Kilimanjaro: a cross sectional study. BMC Pulm Med. 2011;11:54. https://doi.org/10.1186/1471-2466-11-54.
44. Davey NG, Richards LC, Davidson J, Michalchuk T, Gill CG, Krogh ET, et al. Measurement of diacetyl and related compounds in coffee roasteries and breweries. Ann Work Expo Health. 2022;66(5):618–31. https://doi.org/10.1093/annweh/wxab101.
45. Abaya SW, Bråtveit M, Deressa W, Kumie A, Moen BE. Reduced lung function among workers in primary coffee processing factories in Ethiopia: a cross sectional study. Int J Environ Res Public Health. 2018;15(11). https://doi.org/10.3390/ijerph15112415.
46. Harvey RR, Fechter-Leggett ED, Bailey RL, Edwards NT, Fedan KB, Virji MA, et al. The burden of respiratory abnormalities among workers at coffee roasting and packaging facilities. Front Public Health. 2020;8:5. https://doi.org/10.3389/fpubh.2020.00005.
47. LeBouf RF, Aldridge M. Carbon monoxide emission rates from roasted whole bean and ground coffee. J Air Waste Manag Assoc. 2019;69(1):89–96. https://doi.org/10.1080/10962247.2018.1515125.
48. LeBouf RF, Blackley BH, Fortner AR, Stanton M, Martin SB, Groth CP, et al. Exposures and emissions in coffee roasting facilities and cafés: diacetyl, 2,3-pentanedione, and other volatile organic compounds. Front Public Health. 2020;8:561740. https://doi.org/10.3389/fpubh.2020.561740.
49. LeBouf RF, Ranpara A, Fernandez E, Burns DA, Fortner AR. Model predictions of occupational exposures to diacetyl and 2,3-pentanedione emitted from roasted whole bean and ground coffee: influence of roast level and physical form on specific emission rates. Front Public Health. 2022;10:786924. https://doi.org/10.3389/fpubh.2022.786924.
50. Blackley BH, Groth CP, Cox-Ganser JM, Fortner AR, LeBouf RF, Liang X, et al. Determinants of task-based exposures to alpha-diketones in coffee roasting and pack-

aging facilities using a Bayesian model averaging approach. Front Public Health. 2022;10:878907. https://doi.org/10.3389/fpubh.2022.878907.
51. Blackley BH, Fortner AJ, Duling MG. Evaluation of exposures at a coffee roasting, flavoring, and packaging facility. HHE report Report No. HETA-2018-0071-3342 [Internet]. Cincinnati: National Institute for Occupational Safety and Health; 2019 [cited 2025 Dec 29]. Available from: https://www.cdc.gov/niosh/hhe/reports/pdfs/2018-0071-3342.pdf.
52. Oliveira FDSD, De Andrade ET, Viegas C, De Souza JRSC, Rabelo GF, Viegas S. Hidden hazards: a literature review on occupational exposure to fungi and mycotoxins in the coffee industry. Aerobiology. 2025;3(2):3. https://doi.org/10.3390/aerobiology3020003.
53. Harvey RR, Blackley BH, Korbach EJ, Rawal AX, Roggli VL, Bailey RL, et al. Case report: Flavoring-related lung disease in a coffee roasting and packaging facility worker with unique lung histopathology compared with previously described cases of obliterative bronchiolitis. Front Public Health. 2021;9:657987. https://doi.org/10.3389/fpubh.2021.657987.
54. Chetambath R. Popcorn lung – report of a rare case and its significance in a coffee-growing district of Kerala. Lung India. 2019;36(4):367. https://doi.org/10.4103/lungindia.lungindia_441_18.
55. Stanton ML, McClelland TL, Beaty M, Ranpara A, Martin SBJ. Case study: efficacy of engineering controls in mitigating diacetyl and 2,3-pentanedione emissions during coffee grinding. Front Public Health. 2022;10:750289. https://doi.org/10.3389/fpubh.2022.750289.
56. Lee H, Yim J, Lee Y, Lee KG. Effect of organic acid-soaking and sonication on the formation of volatile compounds and α-dicarbonyl compounds in Robusta coffee. Ultrason Sonochem. 2023;99:106580. https://doi.org/10.1016/j.ultsonch.2023.106580.
57. U.S. Environmental Protection Agency. Air pollution control technology fact sheet: thermal incinerator (EPA-452/F-03-022) [Internet]. Research Triangle Park: U.S. Environmental Protection Agency; 2003 [cited 2025 Dec 27]. Available from: https://www.epa.gov/sites/default/files/2020-11/documents/fthermal.pdf.
58. Gasperazzo A, Toffanin P, Larese Filon F. Green coffee been exposure and symptoms in dock workers in Trieste (Italy). Med Lav. 2017;108(5):4. https://doi.org/10.23749/mdl.v108i5.6224.
59. Occupational Safety and Health Administration. Respiratory protection (1910.134) [Internet]. Washington: U.S. Department of Labor, OSHA; 2023 [cited 2025 Dec 27]. Available from: https://www.osha.gov/laws-regs/regulations/standardnumber/1910/1910.134.
60. Redlich CA, Tarlo SM, Hankinson JL, Townsend MC, Eschenbacher WL, Von Essen SG, et al. Official American Thoracic Society technical standards: spirometry in the occupational setting. Am J Respir Crit Care Med. 2014;189(8):983–93. https://doi.org/10.1164/rccm.201402-0337ST.
61. American Conference of Governmental Industrial Hygienists. Diacetyl: TLV® chemical substances documentation [Internet]. Cincinnati: ACGIH; 2023 [cited 2025 Dec 22]. Available from: https://www.acgih.org/diacetyl/.
62. Occupational Safety and Health Administration. 2,3-pentanedione [Internet]. Washington: U.S. Department of Labor, OSHA; 2016 [cited 2025 Dec 22]. Available from: https://www.osha.gov/chemicaldata/822.

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