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Review Article

Respiratory Hazard from Burning Imported Second-Hand Clothing in Accra, Ghana: An Exposure-Chain Review

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Abstract

Background: Open waste burning emits fine particulate matter (PM2.5) and polycyclic aromatic hydrocarbons (PAHs), but the respiratory hazard from burning discarded clothing has not been assessed. In Accra, Ghana, unsellable imported garments are burned on dumpsites and as washhouse fuel.

Methods: A narrative review of OpenAlex, PubMed, grey literature, and trade data, organised as a graded exposure chain.

Findings: 89% of sampled discarded garments contained synthetic fibre, mostly polyester. In one small stove study a cotton-polyester textile mix emitted PAHs at highly variable rates (21 ± 19 mg/kg) similar to common plastics and about 60 times those of wood. In three mostly open-air washhouses sampled by an advocacy group, near-hearth air held two- and three-ring PAHs at 73 to 294 μg/m^3 and benzene at 250 to 887 μg/m^3 while only clothing burned, and benzene at 217 to 5,250 μg/m^3 over a week of clothing with kindling wood; PM2.5 was not measured. No peer-reviewed study has measured respiratory outcomes in people exposed to textile smoke. The closest analogue, household solid-fuel smoke, is associated with chronic obstructive pulmonary disease (COPD) and chronic bronchitis in women and with acute respiratory infection in children, although stove trials have not reduced child pneumonia. Evidence for open waste burning is weaker, resting on self-reported childhood wheeze and a modest COPD association in veterans deployed near burn pits.

Conclusion: The respiratory hazard is plausible, most clearly for washhouse workers and by inference for waste pickers and nearby children, but unquantified. The priority is a panel study with personal exposure monitoring, small-airway and gas-transfer measures, and urinary PAH biomarkers.

***Keywords**: Chronic Obstructive Pulmonary Disease; Household Air Pollution; Lung Function; Open Burning; Particulate Matter; Polycyclic Aromatic Hydrocarbons; Synthetic Fibres; Textile Waste*

Introduction

The problem

Open burning of domestic waste releases PM2.5 and PAHs close to where people live and work, and it has been estimated to cause 270,000 premature adult deaths a year worldwide [1]. In Accra, unsold clothing from the Kantamanto market is burned on dumpsites and as fuel in public washhouses [2]. The people closest to these fires are washhouse workers, waste pickers, and children in informal settlements. This review asks whether burning clothing poses a respiratory hazard and what evidence would settle the question.

Why clothing may differ

Clothing differs from biomass and paper-rich refuse in two ways that matter for the lung. Most discarded garments contain synthetic fibre, mostly polyester [3], and a burned mix of cotton, polyester, and polyamide textiles gave PAH yields closer to plastic than to wood [4]. Synthetic fibres could also be inhaled as particles in their own right. The review therefore builds an exposure chain from imported clothing to emissions, measured concentrations, and dose-response evidence (Figure 1), grades each link, and specifies the study that would close the largest gap.

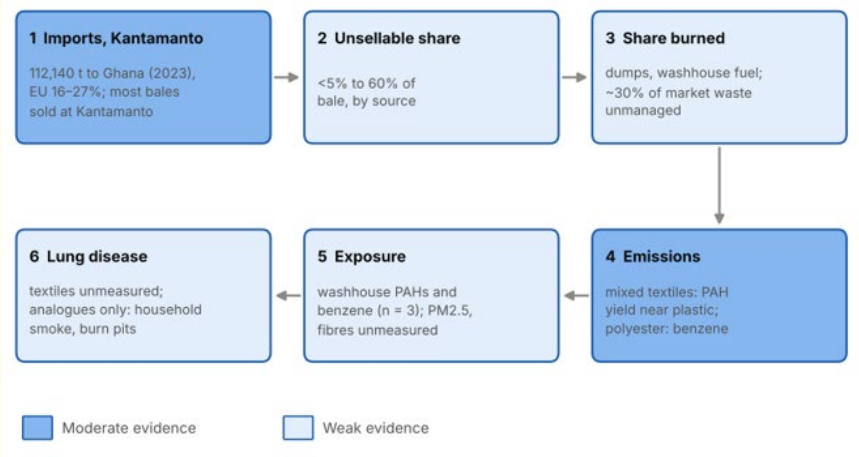


Figure 1: *Exposure chain from imported clothing to respiratory disease in Accra. Shading grades the evidence at each link as in table 1.*

Policy context

The EU's textile extended producer responsibility (EPR) directive is unlikely to change the hazard on its own, because it does not price synthetic fibre content and the EU supplies only 16% to 27% of Ghana's imports [5,6].

Review approach

This is a narrative review with a targeted search. OpenAlex (41 queries) and PubMed (16 queries) were searched in October 2026 without a start-date limit, using terms for textile waste, open burning, waste pickers, burn pits, household air pollution, PAHs, benzene, and lung function, covering each link of the exposure chain, respiratory outcomes in analogous settings, and textile EPR. Targeted searches added grey literature with primary data, WHO guidelines, and key syntheses on household air pollution and deployment-related lung disease. Of 207 candidates assessed in full, 147 were excluded, most often as duplicates or superseded sources (57) or general reviews without specific evidence (31). Sixty sources were retained; the 40 that bear most directly on the respiratory argument are cited here, with five further sources (trade data, a field-study annex, a human-rights report, a news report, and EU occupational law). Each link was graded Strong (several consistent quantitative sources), Moderate (at least one direct quantitative measurement of the link from a peer-reviewed study or official statistics), Weak (small, preliminary, grey, or interested-party data, or evidence from analogous exposures only), or Unmeasured (no measurement and no close analogue). Grades refer to textile-specific evidence, so a link supported only by measurements of other waste is graded Weak.

Source: From imported bales to Accra's fires

Ghana reported importing 112,140 tonnes of worn clothing in 2023. The United Kingdom and China were the largest suppliers [6]. Kantamanto market in central Accra received most of these bales, about 15 million items a week, with around 30,000 workers [7,8]. Fire destroyed much of the market on 2 January 2025, after these figures were collected [9].

Estimates of the unsellable share of a bale come from interested parties. A survey of 370 retailers by the Ghana Used Clothing Dealers Association put waste in new bales at under 5%, The Or Foundation reports that about 40% of bale contents leaves the market as waste, and stallholders told Greenpeace that up to 60% was unsellable [2,8]. The Accra Metropolitan Assembly manages at best about 70% of Kantamanto's waste, and the rest is mismanaged or burned at the market's edges [10]. Unsold clothing also fuels public washhouses in the Old Fadama settlement, where it heats bathing water [2]. No study has measured the share of the unsellable fraction that is burned.

What burning clothes emits

Using plastic and paper emission factors as proxies for polyester and cotton, Bayramova., *et al*. [11] modelled 10 to 20 million tonnes of textile waste burned each year across 137 countries, including 1.36 million tonnes in sub-Saharan Africa. Three of its authors declare an affiliation with a cotton industry body.

Greenpeace Africa [3] scanned 164 garments from three Accra dumpsites and 608 garments rejected at Kantamanto. In both sets 89% contained synthetic fibre, mostly polyester; in the market set the figure was 85% without elastane, and a quarter were wholly synthetic. The synthetic share by mass was not measured. Hoffer., *et al*. [4] burned a textile mix of 46% cotton and 43% polyester-based material, with some polyamide, in a household stove rather than an open fire. It emitted 21 mg/kg of PAHs (standard deviation 19) against 0.34 mg/kg for wood, similar to polyethylene terephthalate and polyethylene and below polystyrene and ABS plastic, and its benzo[a]pyrene toxic equivalent was 390 times that of wood. Its PM10 emission factor was 8.7 g/kg against 2.1 g/kg for wood. In crucible burns at 450°C that simulated open burning of South African household waste, mixed fabrics emitted 54 g/kg of PM10 and 47 g/kg of PM2.5, more PM2.5 than plastic bags (34 g/kg) or dry vegetation (3.2 g/kg) but far less than rubber or smouldering plastic bottles [12]. Textile PM10 factors therefore differ about sixfold between the two laboratory studies, a measure of how uncertain textile emissions remain. The fabrics also had the highest nitrogen and sulfur content of any waste and the highest NOx (11.6 g/kg) and SO_2 (3.7 g/kg) factors, so textile smoke carries airway irritants as well as particles [12,13]. Moltó., *et al*. [14] identified more than 160 compounds from burning polyester fabric, with benzene as the organic compound of highest yield, and measured 17.9 pg WHO-TEQ/g of dioxins and dioxin-like compounds under fuel-rich combustion. No study has burned polyester and cotton garments side by side under open conditions, and no study has measured how prints, flame retardants, or fluorinated finishes change the smoke.

Who breathes it: exposure in Accra and analogous sites

Accra's ambient air

Alli., *et al*. [15] monitored 146 sites in 2019 and 2020 and measured annual mean PM2.5 of 37 μg/m^3 across fixed sites (26 to 43 μg/m^3), more than five times the WHO guideline of 5 μg/m^3 [13]. Lange., *et al*. [16] reanalysed the filters and found that the share of elemental PM2.5 from solid and electronic waste burning rose from 0-11% to 21-35% at four legacy sites over a decade. The city-wide pooled share was about 7%, and the rise was concentrated in dense, low-income neighbourhoods such as Nima and Jamestown. No study has apportioned a textile fraction within the waste-burning factor.

Washhouses, dumpsites, and fires

The strongest textile-specific evidence comes from washhouses that burn clothing as fuel. Old Fadama has about 15, each with four to six bathing cabins [3]. Most are open-air hearths, sometimes under a tin roof, lit three times a day in narrow alleys beside homes, where smoke lingers [3]. Greenpeace Africa [3] sampled three Old Fadama washhouses whose hearths burned only clothing during 20-minute active sampling and clothing with kindling wood during one-week passive sampling. Gas-phase two- and three-ring PAHs, mostly naphthalene and unlike the particle-bound PAHs in the stove study, reached 73, 294, and 80 μg/m^3 in the active samples (Figure 2), against the German indoor guide value II of 30 μg/m^3. Benzene measured 250, 887, and 552 μg/m^3 in the active samples and 217, 5,250, and 1,059 μg/m^3 in the passive samples, against a German indoor guide value of 4.5 μg/m^3 (Figure 2). For washhouse workers the fairer

comparator is the EU occupational limit of 660 µg/m³ (0.2 ppm), an 8-hour average in force from April 2026 [17]; two week-long area values exceed it, although area samples are not personal exposures. The air samples captured only volatile two- and three-ring PAHs, but wall soot near the hearths held the heavier carcinogenic PAHs, including benzo[a]pyrene, with the sum of 16 PAHs reaching 2160 µg/m² against a 10 µg/m² remediation target. PM2.5 and carbon monoxide were not measured. The samplers sat near the hearth, so these are source-proximal concentrations rather than breathing-zone doses, and soot buried the Washhouse 1 passive sampler, so 217 µg/m³ likely understates it. For the sampling, managers agreed not to burn plastics, which are otherwise common washhouse fuels, so the values give a cleaner textile signal but probably understate routine exposure, including to chlorinated dioxins [3].

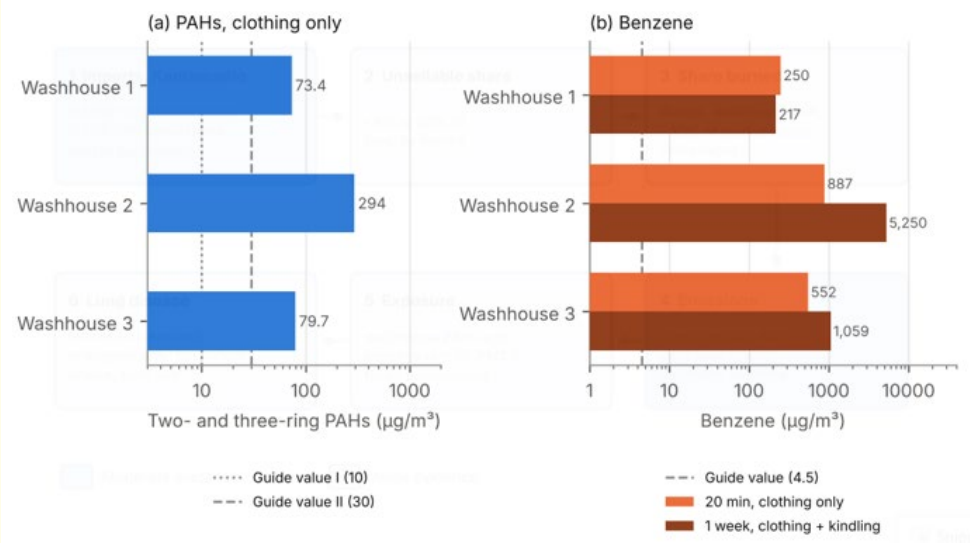


Figure 2: *Near-hearth air in three Accra washhouses burning clothing [3]: (a) two- and three-ring PAHs (20-minute samples, clothing only); (b) benzene (20-minute samples, clothing only; one-week samples, clothing and kindling). Dashed lines: German indoor guide values. Log scale.*

Agbogbloshie e-waste burning is Accra's nearest open-burning setting with peer-reviewed data. Kwarteng., *et al*. [18] measured median gravimetric personal PM2.5 of 93 µg/m³ among workers against 43 µg/m³ in a reference group, and 124 µg/m³ among burners against 88 µg/m³ among other workers. Amoabeng Nti., *et al*. [19] found that working as a burner was associated with a 14.2% lower peak expiratory flow. Laskaris., *et al*. [20] reported wheeze in 23.6% of workers against 8.5% of controls, but shift PM2.5 did not predict cross-shift lung function. These sites burn cable and plastic, so they set an order of magnitude rather than a textile estimate.

Fibres as inhaled particles

Synthetic garments may add a pathway that does not need combustion. Jenner., *et al*. [21] found 39 microplastic particles in 11 of 13 lung tissue samples from living surgical patients; 49% were fibres, and polyethylene terephthalate, the polymer of polyester, made up 18%. Song., *et al*. [22] showed that inhalable nylon fibres reduced airway organoid formation, mainly through a leached chemical, while polyester fibres had much milder effects. Nylon flock workers developed interstitial lung disease at 48 or more times the expected rate [23], but industrially cut respirable flock differs from fibres shed by worn clothing. No study has measured airborne fibre concentrations where second-hand clothing is sorted, sold, or burned.

Respiratory effects

Population burden

Gordon., *et al*. [24] attributed 11,200 deaths in Africa in 2017 to trash burning. Such models cannot resolve neighbourhood hotspots, and Kodros., *et al*. [1] assume that all PM2.5 is equally toxic per microgram.

Household solid-fuel smoke: The closest analogue

A washhouse hearth is an open solid-fuel fire tended several times a day in sheltered alleys, so household air pollution is the closest analogue with a strong evidence base, although washhouses are less enclosed than kitchens. In a meta-analysis of 25 studies, Po., *et al*. [25] found that biomass fuel exposure was associated with acute respiratory infection in children (odds ratio 3.53, 95% CI 1.94 to 6.43), chronic bronchitis in women (2.52, 1.88 to 3.38), and COPD in women (2.40, 1.47 to 3.93), but not with asthma. The Lancet Respiratory Medicine Commission [26] judged the evidence linking household air pollution to COPD strong and noted that plastics used to light stoves may add PAHs and other co-pollutants. It also concluded that exposure must be measured on the person, because fixed monitors misclassify those who tend fires. Randomised trials have not confirmed the effect on child pneumonia. Cleaner biomass stoves in Malawi did not reduce pneumonia (incidence rate ratio 1.01) [27], and in a four-country trial infants in homes given liquefied petroleum gas stoves had estimated median personal PM2.5 of 24 $\mu g/m^3$ against 66 $\mu g/m^3$ in biomass homes, yet no less severe pneumonia (incidence rate ratio 0.96) [28]. Baseline kitchen PM2.5 in stove-intervention studies ranged from several hundred to several thousand $\mu g/m^3$, and the pooled COPD odds ratio of 2.70 in women corresponded to an exposure contrast of 300 versus 100 $\mu g/m^3$ [26]. Whether washhouse PM2.5 reaches that range is unknown, but benzene offers a rough benchmark. Indian cooks' personal benzene while cooking ranged from 11.7 $\mu g/m^3$ in open kitchens to 114 $\mu g/m^3$ in enclosed dung-burning kitchens [29], against 250 to 887 $\mu g/m^3$ beside washhouse hearths burning clothing alone. This marks intense combustion near the hearth, but benzene is not the agent behind the COPD associations and area samples are not personal doses, so the comparison cannot place washhouse workers on the kitchen exposure-response curve. Washhouse fuel, unlike wood, commonly contains synthetic fibre, which should make its smoke more PAH-rich per kilogram.

Living and working near open burning

In Nairobi, Meme., *et al*. [30] studied 2373 schoolchildren and found that refuse burning within sight of the home was associated with current wheeze (adjusted odds ratio 1.47, 95% CI 1.09 to 2.00). The exposure was self-reported, estimated personal PM2.5 was not associated with symptoms, and spirometry did not differ between the informal settlement and the affluent area. Among Delhi ragpickers working on open dumps, Ray., *et al*. [31] recorded respiratory symptoms in 94% and lung function decrement in 52%, against 56% and 34% of matched controls. In Cambodia, brick-kiln workers burning garment-factory waste reported respiratory problems in 2023, but nothing was measured [32].

Military burn pits

Savitz., *et al*. [33] followed 459,381 US veterans for a median of 10.9 years and found that each 100 days of deployment near burn pits raised the adjusted odds of COPD by 4% (odds ratio 1.04, 95% CI 1.02 to 1.07) and of asthma by about 1%. King., *et al*. [34] biopsied 49 soldiers with unexplained exertional dyspnoea after deployment and found constrictive bronchiolitis in 38. Of these 38, 28 had been exposed to a sulfur-mine fire and 24 reported burn-pit exposure, so the disease cannot be attributed to burn pits. Chest radiographs were normal in 37, and 19 had an isolated low diffusing capacity with normal spirometry and lung volumes, although group FEV1 averaged 86.7% of predicted against 99.1% in military controls. The National Academies [35] judged the evidence for chronic cough, shortness of breath, and wheezing after deployment limited or suggestive and found no respiratory outcome with sufficient evidence. It called biopsy interpretation controversial and found no epidemiological link specific to burn pits.

Plastic-rich smoke is probably more toxic per unit mass

Kim., *et al*. [36] burned cardboard, plywood, plastic, and mixed waste and found that, on an equal mass basis, smoke particles from flaming plastic-containing waste caused more lung inflammation and injury in mice and were more mutagenic, in step with higher PAH content. Wu., *et al*. [37] found that aerosols from burning plastic foam, the most toxic plastic type tested, were 1.1 to 2.7 times more toxic to human lung cells than aerosols from other waste types. Neither study burned clothing.

A plausible but unproven pathway links these findings: PAH-laden combustion particles cause oxidative stress and airway inflammation [36,37], and repeated inhalational injury can produce small-airway disease that spirometry misses [34]. This hypothesis is why small-airway measures belong in any study of exposed workers.

PAHs, benzene, and lung function

PM2.5 carries most of the evidence for COPD, infection, and lung function, and PAHs add specific lung evidence. Armstrong., *et al*. [38] pooled 39 cohorts and estimated a lung cancer relative risk of 1.20 per 100 μg/m^3-years of benzo[a]pyrene, but no airborne benzo[a]pyrene has been measured in a washhouse, so this slope cannot yet be applied. Non-cancer effects appear sooner. In 2747 adults, Zhou., *et al*. [39] found that each log-unit rise in total urinary PAH metabolites was associated with a 37 ml lower FEV1, and Chen., *et al*. [40] linked urinary PAH metabolites to lower FEV1 and FEF25% in children near coal-chemical plants. Benzene's established endpoint is acute myeloid leukaemia, with positive associations for lung cancer [41], and the washhouse samples sit 13 to 309 times above the WHO concentration for a 1 in 10,000 lifetime leukaemia risk [42]; these are source-proximal area concentrations whose relation to personal dose is uncertain in both directions. Benzene therefore marks combustion intensity, and the respiratory case rests on PAHs and on PM2.5, which no washhouse study has measured.

The missing epidemiology

No cohort, panel, or biomonitoring study has measured the health of people exposed to textile burning. Glenn., *et al*. [43] found only 14 studies of ambient air pollution and non-communicable respiratory illness in sub-Saharan Africa, and none examined trash burning. Vinti., *et al*. [44] reviewed 29 studies of communities near waste sites and found that none of the dumpsite and open-burning studies measured respiratory outcomes. Velis and Cook [45] found only 20 eligible studies of open plastic burning and judged population risk from PAHs almost impossible to model. Table 1 grades the evidence at each link, and table 2 sets each respiratory analogue against washhouse conditions. The respiratory risk is plausible, most clearly for washhouse workers and by inference for waste pickers and children in informal settlements, but it is unquantified.

Link	Best evidence	Strength
Imports and Kantamanto	UN Comtrade tonnage [6] (official statistics); market throughput from grey sources [7,8]	Moderate for tonnage, Weak for throughput
Unsellable share	Interested-party estimates [2,8]	Weak
Share burned	Unmanaged share of market waste [10]; field reports [2]	Weak
Textile emissions	Stove and laboratory burns [4,12,14]	Moderate
Exposure in Accra	Washhouse air (n = 3) [3]; source apportionment [16]; fibres unmeasured	Weak
Respiratory effect	Analogues only (Table 2); none for textiles	Weak
Inhaled fibres (not drawn in figure 1)	Lung tissue, organoid, and occupational analogues [21-23]; no airborne measurement	Weak

Table 1: *Evidence at each link of the exposure chain, graded by the criteria in section I-D (Figure 1 shows the tonnage grade for link 1).*

Analogue	Exposure measure	Respiratory finding	Difference from washhouses
Household biomass smoke [25,27,28]	Fuel type; trials with personal PM2.5	Child ARI OR 3.53; COPD in women OR 2.40; stove trials null for pneumonia	Kitchens more enclosed; wood, not synthetic fuel
E-waste burning, Accra [18-20]	Personal PM2.5 93 μg/m^3 (workers), 124 μg/m^3 (burners)	Lower peak flow (burners); more wheeze	Outdoor; cable and plastic
Refuse burning near homes [30]	Self-report	Child wheeze aOR 1.47	Outdoor, intermittent; mixed refuse
Military burn pits [33,34]	Days deployed	COPD aOR 1.04 per 100 days; constrictive bronchiolitis on biopsy	Mixed military waste; co-exposures
Washhouses, Accra [3]	PAHs 73 to 294 μg/m^3; benzene 250 to 887 μg/m^3 (clothing only), 217 to 5,250 μg/m^3 (week, with kindling)	Not measured	Target setting

Table 2: *Analogue evidence and its distance from washhouse conditions.*

Conclusion

Burning discarded clothing in Accra likely exposes washhouse workers, and by inference waste pickers and nearby children, to smoke from mixed-fibre textiles whose PAH yields, in the one stove study available, resemble common plastics more than wood, and synthetic fibres may add an inhaled-particle pathway. The closest analogue, household solid-fuel smoke, is associated with COPD and chronic bronchitis and with childhood respiratory infection, which stove trials have not reduced, while the evidence for open waste burning is weaker and rests on self-reported wheeze and a modest COPD association in deployed veterans. No peer-reviewed study has yet measured respiratory outcomes in people exposed to textile smoke, so the hazard is plausible but unquantified. The first priority is a panel study in Accra's washhouses and markets with personal exposure monitoring and sensitive lung function measures.

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