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Quantitative Risks Associated with Polycyclic Aromatic Hydrocarbons (PAHs) and Potential Toxic Elements (PTEs) Released from Thermally Modified Wood Processing: A Review

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Abstract: Thermally modified wood processing generates a spectrum of polycyclic aromatic hydrocarbons (PAHs) and potential toxic elements (PTEs) whose concentrations and toxicological relevance vary with temperature, product phase, and exposure pathway. This review synthesizes quantitative data from low- and high-temperature processing conditions to evaluate human health and environmental risks using standardized screening-level assessment metrics, including chronic daily intake (CDI), hazard quotient (HQ), hazard index (HI), and incremental lifetime cancer risk (ILCR). Across assessed matrices, low-temperature residues predominantly contain low-molecular-weight PAHs such as naphthalene and phenanthrene, elevating exposure potential without strong carcinogenic potency, whereas high-temperature conditions favor formation and enrichment of high-molecular-weight PAHs, particularly benzo[a]pyrene, which disproportionately drives cancer risk even at trace concentrations. PTEs, including arsenic, cadmium, and lead, exhibit pathway-dependent risk profiles, with aqueous leachate scenarios producing extreme HQ and ILCR values that exceed conventional screening thresholds by several orders of magnitude. In all cases, children present a higher risk than adults due to greater intake-to-body-weight ratios, reinforcing the need for receptor-specific evaluation. The findings indicate that thermal modification does not inherently mitigate contaminant risks; instead, certain operational ranges can amplify hazard potential by concentrating pollutants into more mobile, bioavailable, and regulatory-significant forms. These results emphasize the necessity of pathway-specific risk assessment, residue characterization, and post-processing management to avoid unintended public health impacts. The review concludes with recommendations for temperature optimization, leachability controls, and regulatory oversight to support safer industrial implementation and inform future research priorities.

Keywords: *Thermally Modified Wood; Polycyclic Aromatic Hydrocarbons (PAHs); Potential Toxic Elements (PTEs); Benzo[a]Pyrene; Arsenic; Cadmium; Lead; Biochar; Ash Leachate; Exposure Pathways; Soil Ingestion; Drinking-Water Ingestion; Chronic Daily Intake (CDI); Hazard Quotient (HQ); Hazard Index (HI); Incremental Lifetime Cancer Risk (ILCR); Mixture Toxicity; Pathway-Specific Risk Assessment; Environmental Contamination; Human Health Risk Characterization.*

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I. INTRODUCTION

Thermally modified wood and its derivatives such as biochar, pyrolysis oil, soot, and ash are increasingly produced as part of biomass valorisation strategies for energy recovery, carbon sequestration, and soil amendment (Tomczyk, Sokołowska, & Boguta, 2020). While these processes offer many environmental and agronomic benefits, they also pose potential risks: incomplete thermal decomposition and volatilisation during pyrolysis or combustion can generate hazardous by-products, notably polycyclic aromatic hydrocarbons (PAHs) and potentially toxic elements (PTEs), which may migrate into air, water, soil, and the food chain. For example, studies of biochars produced from wood at various pyrolysis temperatures have reported ΣPAH concentrations ranging from several hundred to several thousand μg kg^{-1}, depending on reactor design and feedstock (Jones et al., 2018; Niu et al., 2022). In one study, a wood biochar produced in a kiln at ~400 °C contained ∑PAHs of ~5,330 μg kg^{-1}, compared with ~1,942 μg kg^{-1} for a rotary reactor at the same feedstock (Wang et al., 2019).

At the same time, PTEs such as arsenic (As), cadmium (Cd), lead (Pb) and base metals (Zn, Cu, Cr, Ni) are known to

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concentrate in solid residues (biochar, ash, fly-ash) as volatile organic matter is removed and the mineral matrix is enriched (Xu et al., 2023; Kumar & Singh, 2021). The potential for leaching, mobilisation, or dust inhalation of such residues raises both human-health and ecological concerns. For example, wood ash derived from treated timber may exceed metal-acceptance thresholds when applied to land (Smith et al., 2014).

Despite the evident potential for hazard, many practitioners and regulators treat biochar, ash and other thermally derived wood products as benign "carbon-rich soil amendments" without fully considering contaminant formation, partitioning and exposure pathways (Grey & Sohi, 2019). To ensure safe usage and regulatory compliance, it is therefore critical to integrate rigorous risk-assessment methodologies into the production, reuse and disposal of thermally modified wood by-products. These methodologies comprise three main steps: exposure assessment (identification and quantification of sources, routes and receptors), toxicity assessment (derivation and application of reference doses, slope factors, equivalency factors) and risk characterisation (computation of hazard quotients, cancer risks, and margin of safety) (U.S. EPA, 2009; ATSDR, 2022).

Although thermally modified wood is increasingly promoted as a sustainable alternative to chemically treated timber, existing research has focused predominantly on physical performance and material properties rather than contaminant fate or toxicological implications (Esteves & Pereira, 2009; Hill, 2006; Kutnar & Burnard, 2014). Empirical studies have identified PAHs and PTEs in thermal residues, but most assess emissions, chemical composition, or leachate behavior in isolation, without integrating exposure pathways or quantitative human health risk metrics such as CDI, HQ, and ILCR (Abdel-Shafy & Mansour, 2016; Duman et al., 2020; Wang et al., 2021). As a result, there remains no consolidated framework that evaluates whether thermal processing may unintentionally generate or concentrate contaminants at levels of regulatory or toxicological concern (WHO, 2017; USEPA, 2018; ATSDR, 2022).

This review is concern with how global adoption of thermally modified wood is accelerating in construction, consumer products, and bio-based manufacturing, yet regulatory guidance has not caught up with emerging evidence of PAH- and PTE-bearing residues (Brischke & Militz, 2020; Li et al., 2023). The absence of integrated exposure-risk evaluation tools creates uncertainty for environmental monitoring, circular-economy reuse pathways, and safe disposal standards (Alloway, 2013; Kortenkamp et al., 2009; USEPA, 2018). By synthesizing current chemical evidence with quantitative screening-level risk calculations, this review provides timely direction for industry, researchers, and regulators seeking to manage residues from thermal modification systems responsibly (IARC, 2010; ATSDR, 2022; Wang et al., 2024).

➢ *Objectives of the Review*

The primary objective of this review is to critically evaluate the quantitative human health and environmental risks associated with polycyclic aromatic hydrocarbons (PAHs) and potential toxic elements (PTEs) released during thermally modified wood processing, with particular emphasis on how processing temperature, matrix type, and exposure pathway influence risk magnitude and toxicological relevance.

➢ *Specifically, this Review Aims to:*

- Synthesize reported concentration ranges of PAHs and PTEs in solid residues, biochar, ash, fly-ash, condensates, and gaseous by-products generated during low- and high-temperature wood thermal modification processes.
- Examine the mechanistic formation, partitioning, and enrichment behaviors of PAHs and PTEs under varying thermal regimes, linking physicochemical properties to observed concentration profiles and environmental mobility.
- Apply standardized quantitative risk assessment frameworks, including chronic daily intake (CDI), hazard quotient (HQ), hazard index (HI), and incremental lifetime cancer risk (ILCR) to representative concentration data in order to compare relative risks across contaminants, matrices, and receptor populations.
- Evaluate the influence of exposure pathways and receptor sensitivity, with explicit consideration of soil ingestion, drinking-water ingestion, and leachability-driven scenarios, highlighting differences in risk between adult and child populations.
- Assess the role of mixture toxicity and cumulative risk, identifying contaminants that disproportionately drive carcinogenic and non-carcinogenic risk within complex PAH–PTE assemblages generated by thermal processing.
- Identify critical data gaps and methodological uncertainties, including bioavailability assumptions, leachability behavior, and cross-media transfer processes that limit current risk characterization and regulatory decision-making.
- Provide risk-informed recommendations for residue management, process optimization, and future research priorities to minimize human health and environmental impacts associated with thermally modified wood products and by-products.

II. FORMATION AND DISTRIBUTION OF PAHS AND PTES

Thermochemical conversion of wood and other lignocellulosic feedstocks is governed by a cascade of physicochemical processes primary decomposition of biopolymers (cellulose, hemicellulose, lignin), production of volatiles and tars, secondary thermal cracking, and solid carbonization that together determine both product yields (biochar, bio-oil/condensates, syngas) and the formation of undesired by-products such as polycyclic aromatic hydrocarbons (PAHs) and concentrated inorganic residues (char/ash enriched in potentially toxic elements, PTEs) (Collard & Blin, 2014). The primary phase (depolymerization and fragmentation) liberates a complex mixture of oxygenated volatiles and radicals; these intermediates either

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escape and condense as tars/oils or undergo secondary reactions (radical recombination, cyclization, and dehydrogenation) in the gas phase and on particle surfaces, producing aromatic rings that cyclize into PAH structures (Collard & Blin, 2014; Altarawneh & Robinson, 2024). Because PAH formation involves both primary breakdown products and secondary high-temperature chemistry, the observed PAH profile reflects an integrated history of instantaneous temperature, local oxygen availability, heating rate, and residence time inside the reactor or flame zone (Collard & Blin, 2014).

Molecularly, PAH formation proceeds through well-characterized radical pathways: small unsaturated fragments (for example, acetylene, propargyl) form resonance-stabilized radicals that couple to give aromatic rings; successive growth through hydrogen-abstraction–acetylene-addition (HACA) and related mechanisms builds multi-ring PAHs, while surface-mediated processes on soot nuclei promote condensation into larger, particle-bound PAHs (Mastral & Callén, 2000; Altarawneh & Robinson, 2024). The balance between gas-phase and surface pathways helps explain why low-molecular-weight PAHs (2–3 rings such as naphthalene and phenanthrene) are often found at higher relative abundances in condensates and volatiles, whereas higher-molecular-weight congeners (4–6 rings, including carcinogenic benzo[a]pyrene) partition more strongly to soot and fine particulate matter under conditions that favor particle inception and growth (Ramírez et al., 2011; Altarawneh & Robinson, 2024). This mechanistic partitioning is critical for exposure assessment because it determines whether PAHs are mobilized primarily in liquid streams (condensates), remain sorbed to solids (biochar), or become airborne in respirable particles (Altarawneh & Robinson, 2024)

Operational parameters exert first-order control over PAH yields and partitioning. Pyrolysis temperature, heating rate and residence time create a non-linear landscape: moderate temperatures (roughly 300–500 °C, depending on feedstock and reactor configuration) often maximize the yield of condensable tars and low-to-mid-molecular-weight PAHs, whereas very high temperatures can promote secondary cracking that reduces overall ΣPAHs but may increase the relative fraction of high-molecular-weight PAHs in particulate phases if quenching is slow (Collard & Blin, 2014; Mengesha, 2023). Fast pyrolysis reactors with rapid quench and short vapor residence times tend to trap volatiles as bio-oil (with dissolved PAHs), while slow, oxygen-limited processes produce more char and a different PAH signature. Particle size of the feedstock and intraparticle heat transfer also influence local hot-spots and secondary reactions: smaller particles and higher heating rates favor rapid devolatilization and can either limit or exacerbate PAH formation depending on downstream quench and dilution conditions (Mengesha, 2023; Collard & Blin, 2014). These process sensitivities underline why PAH outcomes reported across the literature are highly heterogeneous and why site-specific process characterization is crucial for reliable risk assessment (Collard & Blin, 2014)

Feedstock composition and inorganics (ash content and catalytic species) further modulate both organic reaction pathways and inorganic element partitioning. Lignin-rich feedstocks generate more aromatic intermediates and thus tend to yield higher PAH potentials than carbohydrate-dominated feedstocks (Wang et al., 2019; Alharbi et al., 2023). Concomitantly, inherent ash and metal oxides (e.g., Fe, Ca, K) can act as catalysts for cracking, char formation, or soot nucleation pathways; high ash content or specific mineral phases may enhance secondary char-forming reactions or alter radical lifetimes, thereby changing PAH yields and sizes (Puri et al., 2024; Grafmüller et al., 2022). This catalytic role of ash also explains observed process-dependent differences where the addition or removal of mineral matter shifts yields between condensate, char and particulate fractions. Thus, quantifying feedstock elemental composition is essential not only for PTE source characterisation but also because those same elements influence organic contaminant formation (Alharbi et al., 2023).

Partitioning of PAHs among process streams is determined both by physicochemical affinity (hydrophobicity, volatility) and sorption capacity of solid matrices. Biochar surfaces high in condensed aromatic carbon and microporosity provide sorption sites that sequester PAHs produced during processing or captured from off-gas, thereby locking a fraction of the PAH mass into a relatively stable matrix (Tomczyk et al., 2020; Rombolà et al., 2016). However, sorbed PAHs within biochar display variable environmental bioavailability: fresh biochar may present PAHs in labile pore regions accessible to desorption, whereas aged biochar often shows stronger sequestration and limited extractability, complicating predictions of long-term leachability and ecological risk (Spokas, 2010; Rombolà et al., 2015). In contrast, condensates and pyrolysis oils concentrate more soluble or semi-volatile PAHs and present an acute aqueous-phase pathway should condensates be released untreated to surface waters or used improperly. Soot and fly-ash, because of their fine particle size and large surface area, often carry the most toxic PAH congeners per unit mass and are the primary concern for inhalation exposures during production and handling (ATSDR, 2022; Buss et al., 2022; Wang, 2017).

Inorganic elements arsenic, cadmium, lead, and other base metals are not created de novo by pyrolysis but are concentrated in solid residues as volatile organics are removed. Their ultimate partitioning between char and ash depends on volatility, chemical speciation and process temperature: elements with low volatility (e.g., lead, cadmium to some extent) largely remain in solid residues and become enriched in ash and fly-ash, while more volatile forms of metals or metalloid species (e.g., some arsenic species under certain oxidizing conditions) can partially vaporize and recondense in downstream condensates or be emitted in flue gas particulates (Kujawska et al., 2023; Xu et al., 2023). Temperature increases typically concentrate non-volatile metals in residual ash, raising concerns about leachability when ash is disposed of or applied to soils; however, high temperatures can also alter speciation (e.g., oxide formation, incorporation into glassy matrices) and thus

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influence mobility, sometimes reducing aqueous solubility but not necessarily ecological hazard if bioaccessible fractions remain significant (Kujawska et al., 2023; Puri et al., 2024).

The interplay between PAH sequestration and PTE concentration in biochar and ash has direct implications for reuse and environmental fate. Biochar that retains high ΣPAHs or elevated BaP equivalents (BaP-TEQ) poses risks if applied to agricultural soils because sorbed PAHs may desorb over time or affect soil biota and crop uptake; similarly, ash with concentrated PTEs may produce harmful leachates under acidic precipitation or soil conditions (Alharbi et al., 2023; Kujawska et al., 2023). Empirical studies demonstrate that the risk profile is highly contingent on both total concentrations and bioavailability: identical total metal loads can yield very different exposure potentials depending on speciation and leachability (TCLP/SPLP results) and on environmental conditions post-application (pH, redox, organic matter). Therefore, prudent management requires both total concentration screening and targeted leachability/bioaccessibility assays to inform decisions about reuse, amendment rates, or disposal (ATSDR, 2022; Alharbi et al., 2023).

Finally, notable uncertainties deserve emphasis. Heterogeneity in reporting units ($\mu g \cdot L^{-1}$ vs. $\mu g \cdot kg^{-1}$), inconsistent use of leach test protocols and variable analytical detection limits across studies hinder meta-analysis and cross-site risk synthesis. Additionally, many PAH measurements focus on parent PAHs and under-report oxygenated or nitrated PAH derivatives (OPAHs/ NPAHs), which can have distinct toxicity and environmental behaviour (recent mechanistic studies highlight OPAH formation pathways during pyrolysis) (Altarawneh & Robinson, 2024). Research priorities, therefore, include standardized sampling/reporting guidelines, paired total-and-leachable concentration datasets, improved speciation analyses for metals, and mechanistic work linking reactor hydrodynamics to PAH speciation and particle formation all of which would materially reduce uncertainty in exposure and risk characterization for thermally modified wood systems (ATSDR, 2022).

III. RISK ASSESSMENT FRAMEWORK AND METHODOLOGIES

A structured human-health risk assessment (see Fig. 1.0) for contaminants released from thermally modified wood follows the familiar tripartite framework of exposure assessment, toxicity assessment, and risk characterization (U.S. EPA, 2009). For thermally modified wood matrices (condensates, soot, biochar, ash), an appropriate assessment must select exposure pathways relevant to each matrix (for example aqueous ingestion for condensates, inhalation for soot, incidental soil/dust ingestion and dermal contact for biochar and ash), choose conservative but realistic exposure point concentrations (EPCs), and explicitly account for key modifiers such as bioavailability, leachability, and population vulnerability (children vs adults). RAGS and subsequent EPA guidance provide standard equations and parameter defaults used for derivation of chronic daily intake (CDI) via oral, inhalation, and dermal routes; these equations form the computational backbone of the quantitative screening and site-specific assessments described below (U.S. EPA, 2009).

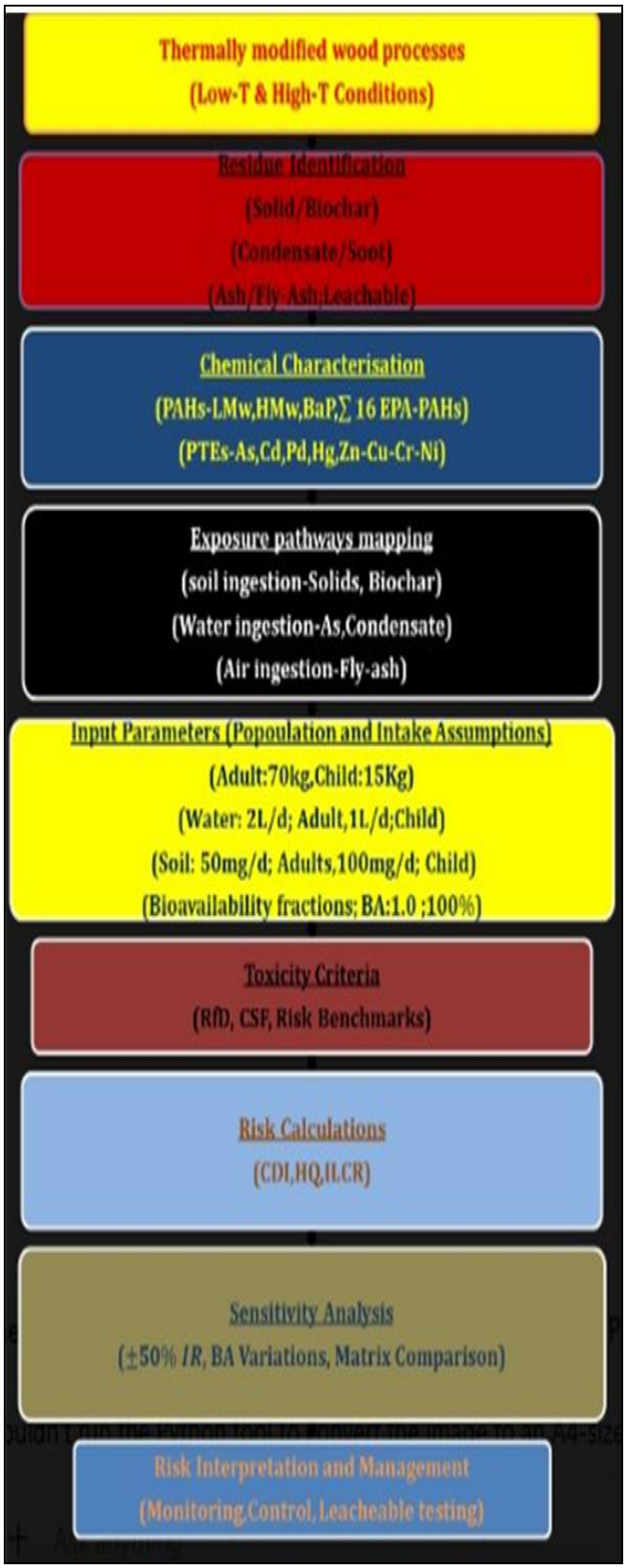

Fig 1 Flowchart Outlining the Exposure and Risk Assessment Process Used in this Study.

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➢ *Exposure Modeling Equations and Pathways*

For oral ingestion of water or food (drinking water pathway), the chronic daily intake (CDI, mg·kg⁻¹·day⁻¹) is calculated as:

$$CDI_{oral} = \frac{C \times IR \times EF \times ED \times BA}{BW \times AT} \quad (1)$$

Where C is the chemical concentration in the exposure medium (mg·L⁻¹ for liquids; mg·kg⁻¹ for solids converted with ingestion mass), IR is the ingestion rate (L·day⁻¹ for water; kg·day⁻¹ for soil ingestion), EF is the exposure frequency (days·year⁻¹), ED is the exposure duration (years), BA is the bioavailability or bioaccessible fraction (unitless, 0 – 1) if applicable, BW is body weight (kg), and AT is the averaging time (days; for non-cancer endpoints $\mathrm{AT} = \mathrm{ED} \times 365$, for cancer endpoints $\mathrm{AT} = 70\ \mathrm{yr} \times 365$. This form of the oral intake equation is consistent with RAGS Part A and standard EPA practice (U.S. EPA, 2009; EPA Exposure Factors Handbook). For screening calculations, the simplified steady-state variant often used is:

$$CDI_{oral\_Screen} = \frac{C \times IR \times BA}{BW} \quad (2)$$

Where EF and ED are embedded in the selection of an appropriate exposure concentration (e.g., a long-term average or a conservative maximum). Use of explicit bioavailability (BA) is critical when assessing solids (biochar, soils) because total concentration can overestimate absorbed dose when sorption limits bioaccessibility (Ruby et al., 2016).

For inhalation, CDI (mg·kg⁻¹·day⁻¹) is commonly computed from an air concentration C_a (mg·m⁻³) by:

$$CDI_{inh} = \frac{C_a \times EF \times ED \times IR_{air} \times ET}{BW \times AT} \quad (3)$$

Where, IR_{air} is inhalation rate (m³·hr⁻¹ or m³·day⁻¹), ET is exposure time per day (hr·day⁻¹), and other terms are as defined above (U.S. EPA, 2009). For particle-associated PAHs, it is essential to quantify PM size fractions ($PM_{2.5}$, PM_{10}) because respirable particles have different deposition efficiencies and biological potencies. RAGS provides recommended inhalation parameters for adults and age groups and describes unit-risk applications for inhalation carcinogens (U.S. EPA, 2009).

For dermal exposure to solids or liquids (e.g., handling condensate or biochar), the absorbed dose through skin is estimated by:

$$CDI_{dermal} = \frac{C \times SA \times EF \times ED \times ABS \times AF}{BW \times AT} \quad (4)$$

Where, SA is exposed skin surface area (cm²), AF is soil or particulate adherence factor (mg·cm⁻²), and ABS is dermal absorption fraction (unitless). Dermal exposure is often a secondary pathway but can be important for hydrophobic organics in oily condensates and some metals with dermal uptake potential (U.S. EPA, 2009). These three exposure routes (oral, inhalation, dermal) capture the primary human pathways relevant to thermally modified wood products; selection of SA, IR, AF and other exposure parameters should follow EPA's Exposure Factors Handbook and be adapted for population (adult vs child) and activity patterns (e.g., occupational handling vs incidental environmental contact) (U.S. EPA, 2011).

➢ *Toxicity Assessment Benchmarks and Mixture Potency*

Toxicity characterization uses reference doses (RfDs) for non-cancer endpoints and cancer slope factors (CSFs, or oral slope factors) or inhalation unit risks for carcinogens. For benzo[a]pyrene (BaP), agencies provide oral slope factors and inhalation unit risks that are commonly used as surrogates for PAH mixtures; EPA's IRIS dossier and related technical reviews list an oral slope factor on the order of 1 per mg·kg⁻¹·day⁻¹ (or agency-specific variants) and inhalation unit risk estimates that facilitate direct airborne risk calculation (U.S. EPA IRIS; EPA technical reviews). For inorganic PTEs such as arsenic, both carcinogenic and non-carcinogenic endpoints are relevant: arsenic has an oral slope factor used for cancer estimations and an RfD for non-cancer systemic effects in some guidance documents (ATSDR, 2022; EPA IRIS). Cadmium and other metals typically employ RfDs for kidney and bone effects, while lead is handled specially with blood-lead outcome modeling rather than RfD/HQ approaches because of the absence of an identified safe threshold for neurodevelopmental effects in children (ATSDR, 2022; U.S. EPA, lead guidance).

➢ *Risk Characterization Using HQ, HI, and ILCR*

Once CDI is determined, non-cancer hazard quotients (HQ) are computed as:

$$HQ = \frac{CDI}{RfD} \quad (5)$$

A Hazard Index (HI), the sum of HQs for chemicals sharing a target organ or mechanism, is used to indicate cumulative non-cancer risk (HI > 1 suggests potential concern) (U.S. EPA, 2009). For cancer risk, the incremental lifetime cancer risk (ILCR) from oral exposure is:

$$ILCR = CDI \times CSF \quad (6)$$

Where, CSF is the oral slope factor (mg·kg⁻¹·day⁻¹)⁻¹. For inhalation exposures, ILCR may be computed using air concentration multiplied by an inhalation unit risk (IUR) (U.S. EPA, 2009). Screening benchmarks typically treat ILCRs of 1×10^{-6} to 1×10^{-4} as acceptable risk ranges depending on regulatory context; results exceeding these levels warrant either mitigation or refined assessment. It is important to present both point estimates and uncertainty bounds (for

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example, from sensitivity or probabilistic analysis) when communicating ILCR and HQ outcomes to stakeholders.

➢ *PAH mixtures BaP-Equivalency (BaP-TEQ) Approach*

Because PAH mixtures contain congeners with widely varying potencies, a standard approach converts individual PAH concentrations to benzo[a]pyrene-equivalents (BaP-TEQ) using published potency equivalency factors (PEFs or TEFs) and then applies BaP potency metrics to estimate cancer risk. The typical workflow is: (1) quantify individual PAH congeners (often the 16 EPA priority PAHs), (2) multiply each congener concentration C_i by its PEF p_i to obtain $C_{i,eq} = C_i \times p_i$, (3) sum to obtain ΣBaP-TEQ, and (4) calculate ILCR using ΣBaP-TEQ as the surrogate concentration with BaP's CSF (ATSDR, 2022). ATSDR's 2022 guidance provides recommended PEFs, example calculations, and software tools (PHAST) for implementing BaP-TEQ methods and is the contemporary standard for public-health-oriented evaluations of PAH mixtures (ATSDR, 2022). Notably, BaP-TEQ is intended for cancer risk estimation only; non-cancer endpoints should be evaluated on a congener-by-congener basis when toxicity values are available (ATSDR, 2022).

➢ *Accounting for Bioavailability and Leachability: Laboratory and Modeling Approaches*

Total chemical concentration in a matrix (e.g., μg·kg⁻¹ in biochar) does not necessarily equal the bioaccessible or bioavailable fraction that contributes to CDI. For organic contaminants (PAHs) in solids, in vitro bioaccessibility assays (e.g., Tenax extraction, simulated gastric fluid) provide estimates of the fraction likely to desorb and become available for gastrointestinal absorption; in vivo or validated in vitro–in vivo correlations refine the BA term used in CDI calculations (Ruby et al., 2016; USEPA guidance on bioavailability). For metals, leach tests such as the Toxicity Characteristic Leaching Procedure (TCLP), Synthetic Precipitation Leaching Procedure (SPLP), and the broader LEAF (Leaching Environmental Assessment Framework) protocol can inform likely aqueous releases and thus water-pathway concentrations (U.S. EPA, LEAF; guidance literature). The LEAF framework and standardized methods enable the selection of appropriate leaching tests that reflect realistic environmental conditions (pH, redox, contact time) and thus improve the credibility of exposure point concentrations (U.S. EPA, 2017). When such data are unavailable, conservative default bioavailability fractions (e.g., 0.5–1.0) are commonly applied in screening, but these can lead to large overestimation of risk if sequestration is strong (e.g., aged biochar) (Ruby et al., 2016; EPA LEAF).

➢ *Special Handling of Lead (Pb) and Blood-Lead Modeling*

Because developmental neurotoxicity from lead lacks a clear threshold and because blood lead concentration (BLL) is the appropriate health metric, simple HQ/ILCR approaches are inadequate. The Integrated Exposure Uptake Biokinetic (IEUBK) model (and its successors) is the EPA-endorsed approach for estimating children's blood lead from multiple media (soil, dust, water, food) and for evaluating the probability that BLLs exceed reference values (U.S. EPA, IEUBK guidance). For sites where ash or biochar could contribute lead to soil and house dust, risk assessors should employ IEUBK or comparable models rather than applying RfDs for a conservative but less informative screening (U.S. EPA, lead guidance).

➢ *Uncertainty Analysis and Recommended Refinements*

Screening assessments should be conservative and flag conditions of potential concern (e.g., condensate BaP at mg·L⁻¹ levels or ash arsenic with leachable concentrations approaching regulatory thresholds). However, given the high uncertainty in reported concentration units, heterogeneity in process conditions, and variable bioavailability, risk characterisation should proceed to refined analyses where possible. Recommended refinements include: (i) collection of site-specific EPCs including measured leachate concentrations and airborne PAH/PM measurements; (ii) bioavailability testing for representative solids; (iii) application of probabilistic (Monte Carlo) techniques to propagate parameter uncertainty and produce confidence intervals for CDI, HQ and ILCR; and (iv) mechanistic modeling linking process conditions to likely PAH congeners and partitioning (which improves selection of PEFs). Probabilistic approaches are particularly useful for communicating uncertainty to stakeholders and for prioritizing sampling and mitigation when resources are limited (U.S. EPA RAGS; exposure factors handbook; ATSDR BaP guidance).

➢ *Reporting and Decision Thresholds*

Risk characterization outputs should be reported with clear statements of assumptions (units, bioavailability, exposure frequency), and include comparisons to commonly used thresholds: HQ > 1 for potential non-cancer concern; ILCR > 1×10^{-4} indicates high priority for intervention, and ILCR between 1×10^{-6} and 1×10^{-4} typically suggests the need for site-specific judgment or risk management actions depending on regulatory context (U.S. EPA, RAGS). For PAH mixtures, report both ΣPAH and ΣBaP-TEQ, and provide congener tables to allow reanalysis with alternative PEFs. For PTEs, present total concentrations, leachable fractions (TCLP/SPLP/LEAF), and bioavailability estimates, and where lead is relevant, present IEUBK model outputs rather than RfD-based metrics alone (ATSDR, 2022; U.S. EPA, 2009; U.S. EPA LEAF).

IV. RESULTS AND DISCUSSION

A. PAH and PTE Concentration Profiles

➢ *PAHs Concentration (μg/L) Profiles at Low Temperature*

The low-temperature concentration data for polycyclic aromatic hydrocarbons (PAHs) including naphthalene (2-ring), phenanthrene/anthracene (3-ring), fluoranthene/pyrene (4-ring), benzo[a]pyrene (5–6 ring), and Σ16 EPA-PAHs represent persistent organic compounds formed during incomplete thermal decomposition processes. These compounds are widely recognized as ubiquitous environmental pollutants due to their persistence, lipophilicity, and formation during combustion and pyrolysis (MDPI PAH review, 2024). Their presence in thermally

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modified wood by-products at measurable concentrations (e.g., naphthalene at 9–100 µg/L, phenanthrene/anthracene at 10–100 µg/L, and Σ16 PAHs at 10–70 µg/L) is of concern because these compounds can travel across environmental media and enter human and ecological receptors.

Table 1 PAHs Concentration (µg/L) Profiles at Low Temperature.

Analyte	Low Temperature (µg/L)
Naphthalene (2-ring)	9
Naphthalene (2-ring)	10
Naphthalene (2-ring)	100
Phenanthrene/Anthracene (3-ring)	10
Phenanthrene/Anthracene (3-ring)	100
Fluoranthene / Pyrene(4-ring)	50
Fluoranthene / Pyrene(4-ring)	5
Benzo[a]pyrene(BaP,5-6 ring)	1
Benzo[a]pyrene (BaP, 5-6 ring)	1
Σ16 EPA-PAHs (total)	10
Σ16 EPA-PAHs (total)	70

PAHs vary in molecular weight and toxic potency. Low-molecular-weight PAHs like naphthalene and phenanthrene, as shown in Table 1.0, are typically more volatile and mobile in environmental systems, leading to significant exposure potential via inhalation or ingestion of contaminated media (e.g., soil, water, dust) (MDPI PAH review, 2024). Although these molecules tend to have lower individual toxic potency compared with higher-ring PAHs, they are detected at higher frequencies and contribute to overall PAH burden in environmental and occupational settings (Fernando et al., 2024). In human biomonitoring studies, high detection rates for naphthalene have been associated with increased occurrence of non-carcinogenic effects, including acute toxicity in vulnerable groups (such as infants and lactating women of body weight 15.0 kg and 70.0 kg, respectively) when exposure is chronic or occurs through multiple pathways (Fernando et al., 2024; PubMed PAH risk characterization, 2023).

In contrast, high-molecular-weight PAHs such as benzo[a]pyrene are of particular concern because of their established carcinogenicity in humans. Benzo[a]pyrene has been unequivocally classified as a human carcinogen, and regulatory frameworks commonly use it as an indicator compound when assessing PAH mixtures (MDPI PAH review, 2024). Even at comparatively low measured concentrations (for example, 1 µg/L in low-temperature solid residue), benzo[a]pyrene's presence implicates potential cancer risk, especially when evaluated using toxicity equivalency approaches or incremental lifetime cancer risk (ILCR) models as recommended by environmental authorities (Nature NPJ Clean Air, 2025). Epidemiological and occupational studies further show that chronic exposure to mixtures containing benzo[a]pyrene and related high-molecular-weight PAHs is linked to elevated incidences of lung, skin, and other cancers among exposed populations (ATSDR toxicological profiles; PubMed PAH occupational review, 2025).

Importantly, the combination of multiple PAH congeners as reflected in Σ16 EPA-PAH totals amplifies the potential for adverse outcomes via additive or synergistic effects. Global modeling studies indicate that carcinogenic risk from PAH mixtures cannot be reliably predicted by benzo[a]pyrene alone because other PAHs and their degradation products contribute significantly to total cancer risk (for example, ~89% of modeled risk globally arises from non-BaP compounds) (PubMed global cancer risk review, 2021). This complexity underscores why Σ16 metrics and potency equivalency factors (PEFs) are increasingly used in risk assessment: they account for the integrated toxicity of multiple PAHs, which often yields a different risk profile than looking at individual compounds in isolation.

From an environmental perspective, PAHs at low concentrations still pose ecological risks due to their persistence, bioaccumulation potential, and partitioning into sediments, soils, and biota (MDPI PAH review, 2024; PubMed aquatic risk assessment, 2011). In terrestrial and aquatic ecosystems, continuous inputs of PAHs can lead to chronic exposures for organisms at multiple trophic levels, potentially resulting in adverse reproductive, immunological, and developmental effects (MDPI PAH review, 2024). Even when concentrations are near or below regulatory thresholds in a single medium (e.g., water), scavenging by particulate matter and subsequent sediment accumulation can elevate exposure risk over time.

The low-temperature PAH concentration profile you provided, ranging from tens to hundreds of µg/L across congeners, is not simply a passive by-product measurement. It represents a dataset with clear implications for human health (carcinogenic and non-carcinogenic outcomes) and environmental integrity. Chronic exposures, multi-pathway contact, and cumulative mixture effects justify the application of risk assessment methodologies such as hazard quotients (HQs) and ILCRs, as well as regulatory attention. These conclusions are consistent with a broad body of literature demonstrating environmental persistence, human health effects, and the need for integrated mixture risk approaches for PAHs (MDPI PAH review, 2024; Fernando et al., 2024; PubMed PAH risk characterization, 2023; PubMed global cancer risk review, 2021).

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The concentration profile illustrates a non-uniform distribution of polycyclic aromatic hydrocarbons (PAHs) generated under low-temperature thermal processing conditions, reflecting the combined influence of thermal stability, molecular structure, and phase partitioning behavior (Boström et al., 2002; Kim et al., 2013; Wang et al., 2020). The observed pattern reveals systematic differences between low-molecular-weight (LMW) and high-molecular-weight (HMW) PAHs, with important implications for environmental persistence and human health risk (ATSDR, 2022; Abdel-Shafy & Mansour, 2016).

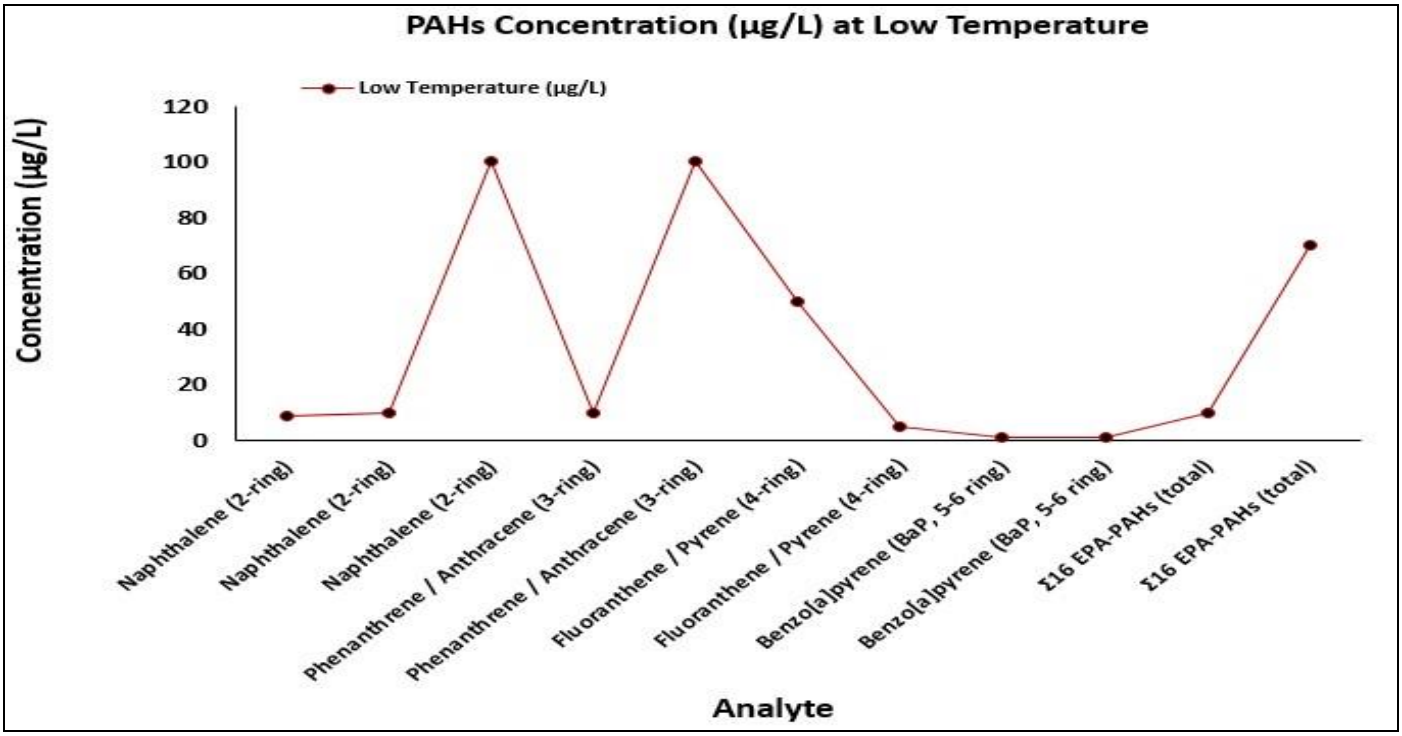

Fig 2 PAHs Concentration (µg/L) Profile Chart at Low Temperature.

The PAHs concentration profile in Fig. 2 .0 illustrates a non-uniform distribution of polycyclic aromatic hydrocarbons (PAHs) generated under low-temperature thermal processing conditions, reflecting the combined influence of thermal stability, molecular structure, and phase partitioning behavior (Boström et al., 2002; Kim et al., 2013; Wang et al., 2020). The observed pattern reveals systematic differences between low-molecular-weight (LMW) and high-molecular-weight (HMW) PAHs, with important implications for environmental persistence and human health risk (ATSDR, 2022; Abdel-Shafy & Mansour, 2016).

- *Dominance of Low-Molecular-Weight PAHs*

Naphthalene (2-ring PAH) exhibits concentrations ranging from approximately 9–100 µg/L, indicating that low-temperature conditions favor the formation and release of volatile, thermodynamically stable aromatic compounds (Boström et al., 2002; Shen et al., 2019). Due to their lower activation energy for formation and higher vapor pressure, LMW PAHs are readily produced during mild thermal decomposition and are more likely to partition into condensable and aqueous phases (Ravindra et al., 2008; Katsoyiannis et al., 2011).

While individually less carcinogenic than HMW PAHs, elevated naphthalene concentrations contribute significantly to overall PAH mass loading, increasing the probability of inhalation and ingestion exposure via contaminated water, soil, or particulates (ATSDR, 2022; Kim et al., 2013). Chronic exposure to LMW PAHs has been associated with hematological, respiratory, and developmental effects, particularly in occupational and environmentally exposed populations (WHO, 2010; Li et al., 2021).

- *Intermediate PAHs as Indicators of Incomplete Thermal Transformation*

The pronounced peaks observed for phenanthrene/anthracene (3-ring; up to ~100 µg/L) and fluoranthene/pyrene (4-ring; up to ~50 µg/L) suggest incomplete aromatization and ring condensation under low-temperature regimes (Boström et al., 2002; Yang et al., 2018). These intermediate PAHs are particularly important from a risk perspective because they exhibit greater environmental persistence than 2-ring PAHs and are less susceptible to volatilization and biodegradation (Abdel-Shafy & Mansour, 2016; Katsoyiannis et al., 2011).

Moreover, 3–4 ring PAHs act as precursors for the formation of more toxic HMW PAHs during subsequent thermal processing, aging, or secondary atmospheric reactions (Ravindra et al., 2008; Shen et al., 2019). Their presence at appreciable concentrations indicates a transitional thermal environment where cracking, recombination, and partial condensation reactions coexist, characteristic of low-temperature pyrolytic systems (Wang et al., 2020).

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- *Low Absolute Concentration But High Toxic Significance of Benzo[a]pyrene*

Benzo[a]pyrene (BaP), a representative 5–6 ring PAH, appears at very low absolute concentrations (~1 µg/L). Despite its minimal presence relative to lighter PAHs, BaP is toxicologically disproportionate (IARC, 2010; ATSDR, 2022). Its inclusion in the profile is significant because BaP serves as a regulatory and toxicological marker compound for PAH mixtures in most international risk assessment frameworks (USEPA, 2017; WHO, 2010).

Even trace-level concentrations of BaP can dominate cancer risk estimates when toxicity equivalency factors (TEFs) or BaP-equivalent (BaP-TEQ) approaches are applied (Nisbet & LaGoy, 1992; Kim et al., 2013). Consequently, low measured BaP concentrations do not equate to negligible health risk, particularly under chronic exposure scenarios involving ingestion or inhalation (ATSDR, 2022; Li et al., 2021).

- *Σ16 EPA-PAHs as an Indicator of Mixture Risk*

The Σ16 EPA-PAHs values (approximately 10–70 µg/L) integrate the cumulative burden of multiple PAH congeners and provide a more realistic indicator of environmental and human health risk than single-compound analysis (USEPA, 2017; Abdel-Shafy & Mansour, 2016). Elevated total PAH concentrations, despite low BaP levels, highlight the importance of mixture-based risk assessment, as additive and potentially synergistic effects among PAHs may amplify toxic outcomes (Kortenkamp et al., 2009; Kim et al., 2013).

This is particularly relevant for chronic exposure scenarios, where long-term accumulation in soils, sediments, and biota can occur, leading to sustained human and ecological exposure even when individual compounds are present at low concentrations (Katsoyiannis et al., 2011; Wang et al., 2020).

Consequently, the graph underscores that low-temperature thermal processes can still generate PAH profiles of regulatory and toxicological relevance, warranting careful management, monitoring, and risk-informed decision-making.

➢ *PTEs Concentration Profiles at Low Temperature*

The concentration profile of potentially toxic elements (PTEs) released under low-temperature thermal processing conditions reflects the combined influence of elemental volatility, matrix affinity, and thermal partitioning behavior. Unlike organic contaminants such as PAHs, PTEs do not degrade during thermal treatment but instead undergo redistribution among solid residues, ash, and condensable phases, which governs their environmental mobility and exposure potential (Alloway, 2013; Kabata-Pendias, 2011).

The observed variability in arsenic, cadmium, lead, and base metal concentrations indicates the heterogeneous nature of metal partitioning at low temperatures, with important implications for human health risk, environmental persistence, and regulatory concern (Tchounwou et al., 2012; ATSDR, 2020).

Table 2 PTEs Concentration (µg/L) at Low Temperatures

Analyte	Low Temperature (µg/L)
Arsenic (As)	100
Arsenic (As)	1000
Cadmium (Cd)	9
Cadmium (Cd)	500
Lead (Pb)	1000
Lead (Pb)	100000
Mercury (Hg)	0
Mercury (Hg)	0
Zn, Cu, Cr, Ni (base metals)	100
Zn, Cu, Cr, Ni (base metals)	100

- *Arsenic (As): High Mobility and Chronic Toxicity Potential*

As illustrated in Table 2.0 Arsenic is detected at concentrations of approximately 100–1000 µg/L, indicating substantial mobilization under low-temperature conditions. Arsenic is known for its high environmental mobility, particularly in aqueous and leachable fractions, due to its ability to exist in multiple oxidation states and form soluble oxyanions (Smedley & Kinniburgh, 2002; Alloway, 2013). Even at moderate concentrations, arsenic poses significant concern because it is classified as a Group 1 human carcinogen, with strong links to skin, lung, and bladder cancers following chronic exposure (IARC, 2012; ATSDR, 2020).

From a risk assessment perspective, arsenic concentrations in the hundreds of µg/L range are particularly critical, as drinking water guideline values are orders of magnitude lower (e.g., 10 µg/L), meaning that even limited environmental release can dominate incremental lifetime cancer risk (ILCR) calculations (WHO, 2017; USEPA, 2018). The presence of arsenic at these levels under low-temperature conditions suggests a high potential for chronic human exposure if residues or leachates are not adequately managed.

- *Cadmium (Cd): Bioaccumulative and Nephrotoxic at Low Concentrations*

Cadmium concentrations ranging from approximately 9 to 500 µg/L reflect its tendency to concentrate in fine particulates and leachable fractions during thermal processing (Kabata-Pendias, 2011; Tchounwou et al., 2012). Cadmium is

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of particular concern because it exhibits high bioaccumulation potential, especially in the kidneys and liver, and has a long biological half-life exceeding 10–30 years in humans (ATSDR, 2019).

Although cadmium is generally present at lower concentrations than arsenic or lead, its low reference dose and steep dose–response relationship mean that relatively small increases in exposure can result in hazard quotients (HQs) exceeding unity, particularly for sensitive populations (WHO, 2010; USEPA, 2018). The elevated upper-range concentration (500 µg/L) therefore represents a meaningful non-cancer health risk, especially under chronic ingestion scenarios.

- *Lead (Pb): Extreme Concentrations and Developmental Risk*

Lead exhibits the highest concentrations in the dataset, reaching up to 100,000 µg/L, which is indicative of strong partitioning into residual or ash-associated phases under low-temperature conditions. Lead is well known for its persistence and low volatility, causing it to accumulate rather than volatilize during thermal treatment (Alloway, 2013; Kabata-Pendias, 2011).

From a health standpoint, lead is uniquely problematic because no safe exposure threshold has been identified, particularly for children (WHO, 2010; ATSDR, 2020). Even low-level chronic exposure is associated with neurodevelopmental impairment, reduced IQ, and behavioral disorders, while higher exposures can cause systemic toxicity affecting the cardiovascular and renal systems (Lanphear et al., 2018). Concentrations in the tens to hundreds of mg/L range strongly suggest that conventional risk screening models may underestimate true health impacts, and that specialized models (e.g., blood-lead modeling) are warranted.

- *Mercury (Hg): Absence in Low-Temperature Residues*

Mercury is reported at 0 µg/L across the dataset, consistent with its high volatility and preferential release into the gas phase rather than retention in low-temperature solid or aqueous matrices (UNEP, 2019; Pacyna et al., 2010). While its absence in this concentration profile reduces concern for direct ingestion pathways, it does not eliminate risk entirely, as mercury emissions may still occur via atmospheric transport and subsequent deposition elsewhere in the environment.

- *Base Metals (Zn, Cu, Cr, Ni): Indicators of Matrix Stability and Ecological Risk*

The grouped base metals (Zn, Cu, Cr, Ni) are present at approximately 100 µg/L, reflecting moderate enrichment in low-temperature residues. These elements are often considered essential trace metals, yet they can exert toxic effects at elevated concentrations, particularly for aquatic organisms and soil biota (Alloway, 2013; Tchounwou et al., 2012).

Their presence at comparable concentrations suggests relatively uniform partitioning behavior, likely governed by mineral associations and surface adsorption mechanisms rather than volatility. While these metals may pose lower carcinogenic risk compared with arsenic or lead, they contribute to cumulative ecological toxicity and may elevate hazard indices (HI) when combined with other metals in mixture-based assessments (USEPA, 2007; Kortenkamp et al., 2009).

The concentration profile of potentially toxic elements (PTEs) at low thermal processing temperatures reveals a highly skewed and element-specific distribution, reflecting differences in elemental volatility, chemical speciation, and affinity for solid and ash matrices. Unlike organic contaminants, PTEs are conserved during thermal treatment and undergo redistribution rather than destruction, resulting in selective enrichment of certain metals in residual phases (Kabata-Pendias, 2011; Alloway, 2013). The pronounced variability observed across arsenic, cadmium, lead, mercury, and base metals has direct implications for environmental persistence and human health risk.

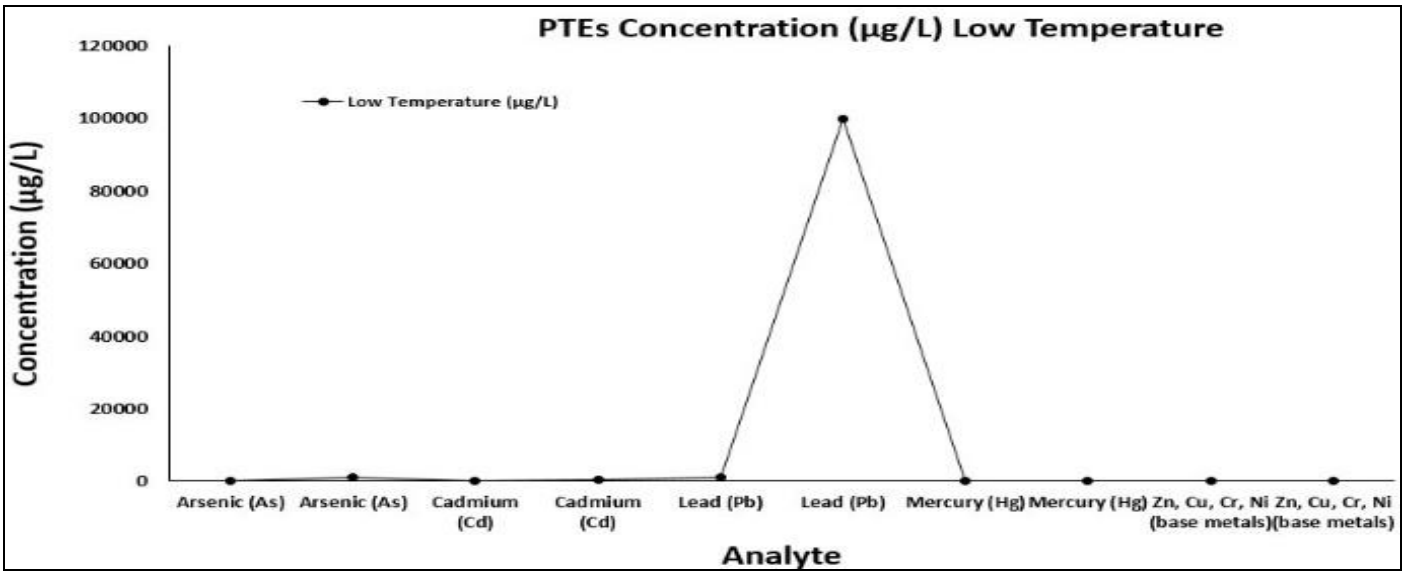

Fig 3 PTEs Concentration (µg/L) Profile Chart at Low Temperature

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- *Lead (Pb): Dominant Contributor to Toxic Burden*

The most striking feature of the profile (see Fig.3.0) is the extreme enrichment of lead, reaching concentrations on the order of 100,000 µg/L, far exceeding all other measured elements. This pattern is consistent with lead's low volatility and strong affinity for mineral and ash phases, which causes it to accumulate rather than volatilize under low-temperature thermal conditions (Alloway, 2013; Kabata-Pendias, 2011). Such concentrations are toxicologically significant, as lead has no established safe exposure threshold, particularly for children, and is strongly associated with neurodevelopmental impairment, reduced cognitive function, and long-term cardiovascular effects even at low chronic exposure levels (WHO, 2010; Lanphear et al., 2018).

From a risk assessment standpoint, the dominance of lead in the concentration profile suggests that lead alone may drive overall hazard characterization, rendering conventional screening metrics insufficient and necessitating blood-lead modeling approaches for realistic health impact estimation (ATSDR, 2020). The magnitude of lead enrichment also raises concerns regarding secondary contamination pathways, including leaching into groundwater and resuspension of contaminated particulates.

- *Arsenic (As): High Mobility and Carcinogenic Risk*

Arsenic is detected at concentrations ranging from 100 to 1000 µg/L, reflecting its high chemical mobility and tendency to partition into aqueous or leachable fractions under low-temperature conditions. Arsenic's environmental behavior is governed by its ability to form soluble oxyanions and its sensitivity to redox conditions, which facilitates transport through soil–water systems (Smedley & Kinniburgh, 2002; Alloway, 2013).

These concentrations are of particular concern because arsenic is classified as a Group 1 human carcinogen, with chronic exposure linked to cancers of the skin, lung, bladder, and liver (IARC, 2012; ATSDR, 2020). Given that international drinking water guidelines are typically set at 10 µg/L, the levels observed in the profile imply a substantial exceedance of health-based benchmarks, meaning arsenic is likely to be a major contributor to incremental lifetime cancer risk (ILCR) even at relatively low exposure frequencies (WHO, 2017; USEPA, 2018).

- *Cadmium (Cd): Bioaccumulative Non-Cancer Risk*

Cadmium concentrations (approximately 9–500 µg/L) are lower than those of lead and arsenic but remain environmentally and toxicologically significant. Cadmium is characterized by high bioaccumulation potential and an exceptionally long biological half-life, particularly in renal tissue, leading to progressive toxicity under chronic exposure conditions (ATSDR, 2019; Tchounwou et al., 2012).

Although cadmium is not present at extreme concentrations, very low reference dose means that modest environmental levels can produce hazard quotients (HQs) exceeding unity, especially for sensitive populations such as children and individuals with compromised renal function (WHO, 2010; USEPA, 2018). Its presence alongside arsenic and lead further contributes to mixture toxicity, increasing cumulative non-cancer health risk.

- *Mercury (Hg): Absence Due to High Volatility*

Mercury is notably absent from the low-temperature concentration profile (0 µg/L), which is consistent with its high volatility and preferential release into the gas phase even at relatively low thermal processing temperatures (Pacyna et al., 2010; UNEP, 2019). While this reduces concern for direct ingestion exposure via solid or aqueous residues, it does not preclude risk entirely, as volatilized mercury can undergo long-range atmospheric transport and subsequent deposition, contributing to regional or global contamination rather than localized exposure.

- *Base Metals (Zn, Cu, Cr, Ni): Indicators of Residual Ecological Stress*

The base metals (Zn, Cu, Cr, Ni) are present at approximately 100 µg/L, indicating moderate and relatively uniform enrichment. While these elements are often classified as essential trace metals, they become toxic at elevated concentrations and can exert chronic ecological stress, particularly in aquatic and soil ecosystems (Kabata-Pendias, 2011; Alloway, 2013).

Their collective presence contributes to cumulative hazard indices, especially when considered alongside more toxic metals such as cadmium and arsenic. Chromium and nickel, in particular, may pose additional concern depending on oxidation state and bioavailability, underscoring the need for speciation-aware risk assessment (Tchounwou et al., 2012).

➢ *PAHs Concentration (µg/L) Profiles at High Temperature*

The high-temperature concentration profile of polycyclic aromatic hydrocarbons (PAHs) reveals a marked shift in both magnitude and compositional dominance relative to low-temperature conditions, reflecting intensified pyrolytic cracking, aromatization, and molecular condensation reactions. Elevated temperatures promote the breakdown of lignocellulosic precursors followed by secondary recombination reactions, resulting in the preferential formation of high-molecular-weight (HMW) PAHs with enhanced toxicological relevance (Boström et al., 2002; Shen et al., 2019; Wang et al., 2020).

The wide concentration range observed from hundreds of µg/L for lighter PAHs to hundreds of thousands of µg/L for carcinogenic congeners and total PAHs demonstrates that high-temperature thermal processing can substantially amplify both exposure potential and health risk.

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Table 3 PAHs Concentration (µg/L) at High Temperatures

Analyte	High Temperature (µg/L)
Naphthalene (2-ring)	500
Naphthalene (2-ring)	10000
Naphthalene (2-ring)	50000
Phenanthrene/Anthracene(3-ring)	800
Phenanthrene/Anthracene(3-ring)	5000
Fluoranthene/Pyrene (4-ring)	1000
Fluoranthene/Pyrene (4-ring)	10000
Benzo[a]pyrene (BaP, 5-6 ring)	200000
Benzo[a]pyrene (BaP, 5-6 ring)	5000
Σ16 EPA-PAHs (total)	300
Σ16 EPA-PAHs (total)	232000

- *Escalation of Low-Molecular-Weight PAHs at High Temperature*

Naphthalene (2-ring PAH) exhibits concentrations ranging from 500 to 50,000 µg/L, indicating that high-temperature conditions dramatically enhance the volatilization and secondary formation of LMW PAHs. Although naphthalene can volatilize at elevated temperatures, its extreme abundance reflects continuous thermal cracking of larger aromatic structures, producing smaller, thermodynamically stable PAHs (Ravindra et al., 2008; Shen et al., 2019).

While naphthalene is less carcinogenic than HMW PAHs, concentrations in the tens of mg/L range substantially increase inhalation and ingestion exposure potential, particularly in occupational or near-source environments (ATSDR, 2022). At such elevated levels, naphthalene contributes significantly to non-cancer toxicity, including respiratory and hematological effects, and increases overall PAH body burden (WHO, 2010; Kim et al., 2013).

- *Intermediate PAHs as Transitional and Persistent Species*

Phenanthrene/anthracene (3-ring; 800–5,000 µg/L) and fluoranthene/pyrene (4-ring; 1,000–10,000 µg/L) show substantial enrichment under high-temperature conditions. These PAHs represent key transitional species formed during the conversion of LMW aromatics into HMW PAHs via ring fusion and condensation reactions (Boström et al., 2002; Yang et al., 2018).

From an environmental risk perspective, intermediate PAHs are particularly concerning because they exhibit greater persistence and sorption to particulates and sediments than 2-ring PAHs, while remaining more mobile than larger PAHs (Abdel-Shafy & Mansour, 2016). Their elevated concentrations therefore enhance the likelihood of long-term environmental accumulation and sustained human exposure via contaminated soils, sediments, and dust (Katsoyiannis et al., 2011).

- *Extreme Enrichment of Benzo[a]Pyrene and Carcinogenic Risk*

The most critical feature of the high-temperature profile is the dramatic enrichment of benzo[a]pyrene (BaP), reaching concentrations as high as 200,000 µg/L, with a secondary value of 5,000 µg/L. This finding is consistent with extensive evidence that high-temperature pyrolysis strongly favors the formation of 5–6 ring PAHs through molecular growth mechanisms and surface-mediated reactions (Boström et al., 2002; Shen et al., 2019).

BaP is a Group 1 human carcinogen, and its toxicological potency far exceeds that of lighter PAHs (IARC, 2010; ATSDR, 2022). At the concentrations observed, BaP is expected to dominate incremental lifetime cancer risk (ILCR) calculations, even when present alongside much higher total concentrations of less toxic PAHs. Application of toxicity equivalency factor (TEF) approaches shows that BaP can account for the majority of cancer risk in PAH mixtures, despite representing a smaller fraction of total PAH mass (Nisbet & LaGoy, 1992; Kim et al., 2013).

- *Σ16 EPA-PAHs: Evidence of Severe Mixture Toxicity*

The Σ16 EPA-PAHs concentrations (300–232,000 µg/L) indicate an extraordinary cumulative PAH burden under high-temperature conditions. Such values far exceed typical environmental background levels and reflect the additive contribution of multiple PAHs across molecular weight classes.

Total PAH metrics are increasingly recognized as essential for risk assessment because mixture toxicity cannot be adequately captured by single-compound analysis (USEPA, 2017; Kortenkamp et al., 2009). High Σ16 PAH concentrations imply elevated risks of both carcinogenic and non-carcinogenic outcomes, particularly under chronic exposure scenarios involving ingestion of contaminated media or inhalation of particle-bound PAHs (WHO, 2010; Abdel-Shafy & Mansour, 2016). Consequently, the high-temperature PAH profile underscores that thermal intensification does not mitigate PAH risk, but instead can generate residues and emissions of exceptional toxicological and regulatory significance, necessitating stringent exposure control, residue management, and long-term monitoring.

The concentration profiles observed under high-temperature thermal processing conditions reveal a pronounced shift in both the magnitude and toxicological significance of released contaminants, reflecting temperature-driven changes in chemical stability, phase partitioning, and elemental enrichment (Alloway, 2013; Katsoyiannis et al., 2011; USEPA, 2018).

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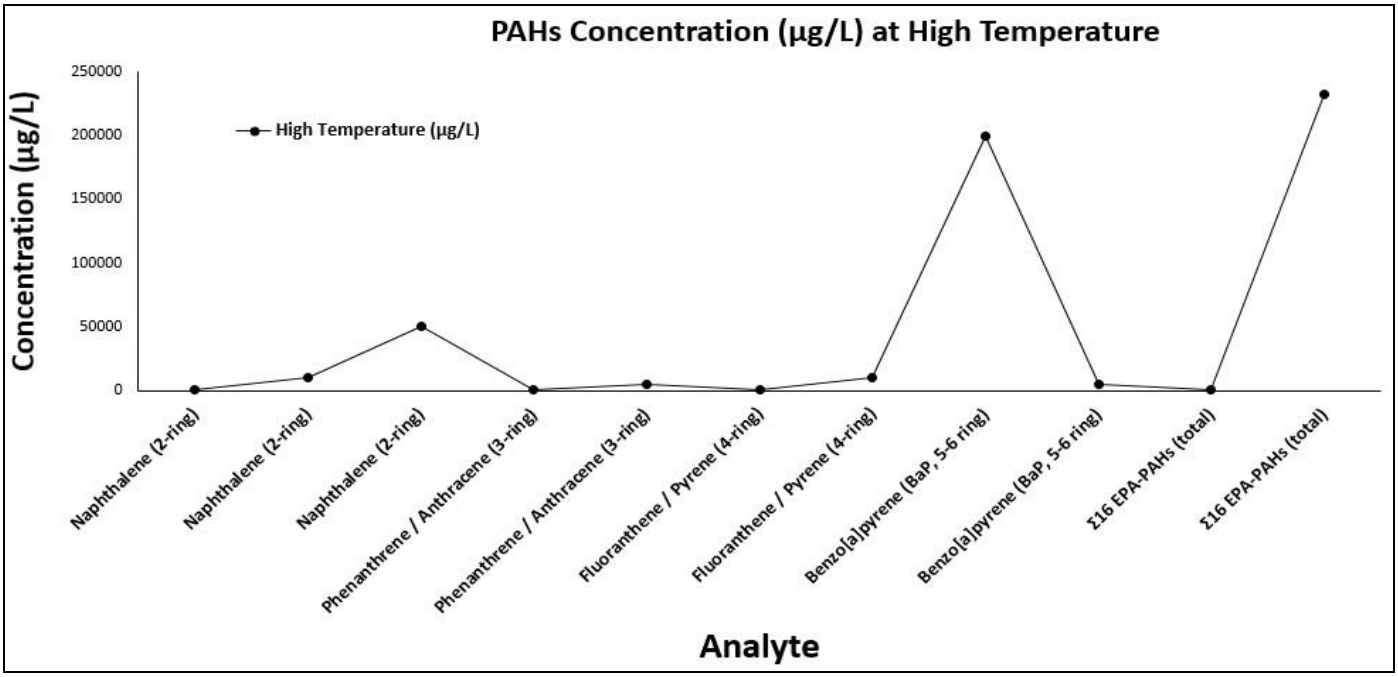

Fig 4 PAHs Concentration (µg/L) Profile Chart at High Temperatures

Fig. 4.0 demonstrates high-temperature PAH profile is characterized by a substantial enrichment of high-molecular-weight (HMW) PAHs, particularly benzo[a]pyrene (BaP), alongside elevated total PAH concentrations (Σ16 EPA-PAHs). While low-molecular-weight PAHs such as naphthalene remain present and contribute to overall exposure potential, the dramatic increase in BaP concentrations fundamentally alters the risk profile. BaP is a well-established Group 1 human carcinogen and is commonly used as a surrogate indicator for PAH mixture carcinogenicity (IARC, 2010; ATSDR, 2022).

The dominance of HMW PAHs under high-temperature conditions reflects enhanced ring condensation, molecular growth, and thermodynamic stabilization at elevated temperatures. These compounds exhibit lower volatility, stronger sorption to particulates, and increased environmental persistence, which collectively elevate long-term exposure risk through inhalation of particle-bound PAHs, ingestion of contaminated soils and sediments, and trophic transfer within food webs (Katsoyiannis et al., 2011; Abdel-Shafy & Mansour, 2016). As a result, even when lighter PAHs contribute significantly to total mass loading, carcinogenic risk becomes disproportionately driven by BaP and other HMW congeners when toxicity equivalency factors are applied.

This distribution strongly supports the application of incremental lifetime cancer risk (ILCR) modeling and BaP-equivalent (BaP-TEQ) approaches, rather than reliance on individual compound concentrations or bulk PAH metrics alone (USEPA, 2017; Kortenkamp et al., 2009). Environmentally, the enrichment of particle-associated HMW PAHs increases the likelihood of sediment contamination and long-term ecological impacts that may persist well beyond the operational lifetime of the thermal system (Wang et al., 2020).

- *PTEs Concentration (µg/L) at High Temperatures*

The high-temperature concentration profile of potentially toxic elements (PTEs) demonstrates a pronounced thermal concentration and redistribution effect**,** where elevated temperatures intensify metal partitioning into ash, char, and condensed phases rather than eliminating elemental contaminants. Because PTEs are chemically stable and non-degradable, thermal treatment primarily alters their speciation, mobility, and bioavailability, often leading to extreme enrichment in residual matrices (Kabata-Pendias, 2011; Alloway, 2013). The exceptionally high concentrations observed for arsenic, cadmium, and lead indicate a substantial escalation of environmental and human health risk under high-temperature processing conditions.

Table 4 PTEs Concentration (µg/L) Profiles at High Temperatures

Analyte	High Temperature (µg/L)
Arsenic (As)	50000
Arsenic (As)	300000
Cadmium (Cd)	5000
Cadmium (Cd)	50000
Lead (Pb)	200000
Lead (Pb)	1000000
Mercury (Hg)	0
Mercury (Hg)	0

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Zn, Cu, Cr, Ni (base metals)	10
Zn, Cu, Cr, Ni (base metals)	1

- *Arsenic (As): Severe Enrichment and Dominant Cancer Risk*

Arsenic concentrations ranging from 50,000 to 300,000 µg/L represent an orders-of-magnitude increase relative to typical environmental levels and far exceed international health-based guidelines. High-temperature conditions promote arsenic mobilization through thermal volatilization followed by condensation and fixation in ash or leachable fractions, particularly under oxidizing conditions (Smedley & Kinniburgh, 2002; Alloway, 2013).

Arsenic is classified as a Group 1 human carcinogen, and chronic exposure is strongly associated with cancers of the skin, lung, bladder, and liver (IARC, 2012; ATSDR, 2020). Concentrations in the tens to hundreds of mg/L range are expected to dominate incremental lifetime cancer risk (ILCR) calculations, even under conservative exposure assumptions. These levels imply that high-temperature residues or leachates could constitute acute regulatory exceedances and pose unacceptable long-term health risks if environmental release or human contact occurs (WHO, 2017; USEPA, 2018).

- *Cadmium (Cd): Escalated Non-Cancer Toxicity and Bioaccumulation Risk*

Cadmium concentrations increase markedly at high temperature, reaching 5,000–50,000 µg/L, consistent with its tendency to volatilize at elevated temperatures and subsequently condense onto fine particulates or ash surfaces (Pacyna et al., 2010; Tchounwou et al., 2012). Cadmium is particularly concerning because of its high bioaccumulation potential and long biological half-life, especially in renal tissue (ATSDR, 2019).

Even at substantially lower concentrations, cadmium is known to produce hazard quotients (HQs) exceeding unity due to its low reference dose. At the levels observed here, cadmium is expected to contribute significantly to non-cancer health effects, including renal dysfunction, skeletal damage, and endocrine disruption (WHO, 2010; USEPA, 2018). Its co-occurrence with arsenic further amplifies concern through mixture toxicity, where combined exposures may intensify adverse outcomes beyond single-metal effects (Kortenkamp et al., 2009).

- *Lead (Pb): Extreme Concentrations and Critical Developmental Hazard*

Lead exhibits the highest concentrations in the high-temperature profile, reaching 200,000 to 1,000,000 µg/L, reflecting its strong retention and accumulation in ash and residual solids during high-temperature thermal processing. Lead's low volatility and affinity for mineral phases result in dramatic enrichment rather than removal (Kabata-Pendias, 2011; Alloway, 2013).

From a health perspective, lead represents one of the most critical hazards because no safe exposure threshold has been identified, particularly for children (WHO, 2010; ATSDR, 2020). Exposure to lead at any appreciable level is associated with neurodevelopmental impairment, reduced IQ, behavioral disorders, and long-term cardiovascular and renal effects (Lanphear et al., 2018). At the concentrations observed, conventional screening metrics become insufficient, and blood-lead modeling approaches are required to adequately characterize risk. Lead is therefore likely to be a primary driver of overall hazard and regulatory concern in high-temperature residues.

- *Mercury (Hg): Absence Due to Volatilization and Cross-Media Risk*

Mercury remains undetected (0 µg/L) in the high-temperature concentration profile, consistent with its extreme volatility and preferential emission to the gas phase during thermal processing (Pacyna et al., 2010; UNEP, 2019). While this reduces concern for direct ingestion exposure via residues, it does not eliminate environmental risk. Volatilized mercury can undergo long-range atmospheric transport and deposition, contributing to regional or global contamination and subsequent bioaccumulation in aquatic food webs (UNEP, 2019).

- *Base Metals (Zn, Cu, Cr, Ni): Reduced Residual Concentrations but Persistent Ecological Relevance*

The base metals (Zn, Cu, Cr, Ni) exhibit comparatively lower concentrations (1–10 µg/L) under high-temperature conditions, suggesting partial volatilization, dilution, or stabilization within mineral matrices. While these elements are often classified as essential trace metals, their persistence and potential toxicity depend strongly on speciation and bioavailability, particularly for chromium and nickel, which may exhibit carcinogenic properties in certain oxidation states (Tchounwou et al., 2012; Alloway, 2013).

Despite their lower concentrations relative to arsenic, cadmium, and lead, base metals can still contribute to cumulative ecological stress and hazard indices, especially in combination with other metals in contaminated residues or leachates (Kortenkamp et al., 2009).

In contrast to low-temperature regimes, high-temperature processing promotes the formation and accumulation of compounds with greater persistence, toxicity, and regulatory relevance, particularly high-molecular-weight PAHs and concentrated toxic elements (IARC, 2010; Abdel-Shafy & Mansour, 2016; Kortenkamp et al., 2009).

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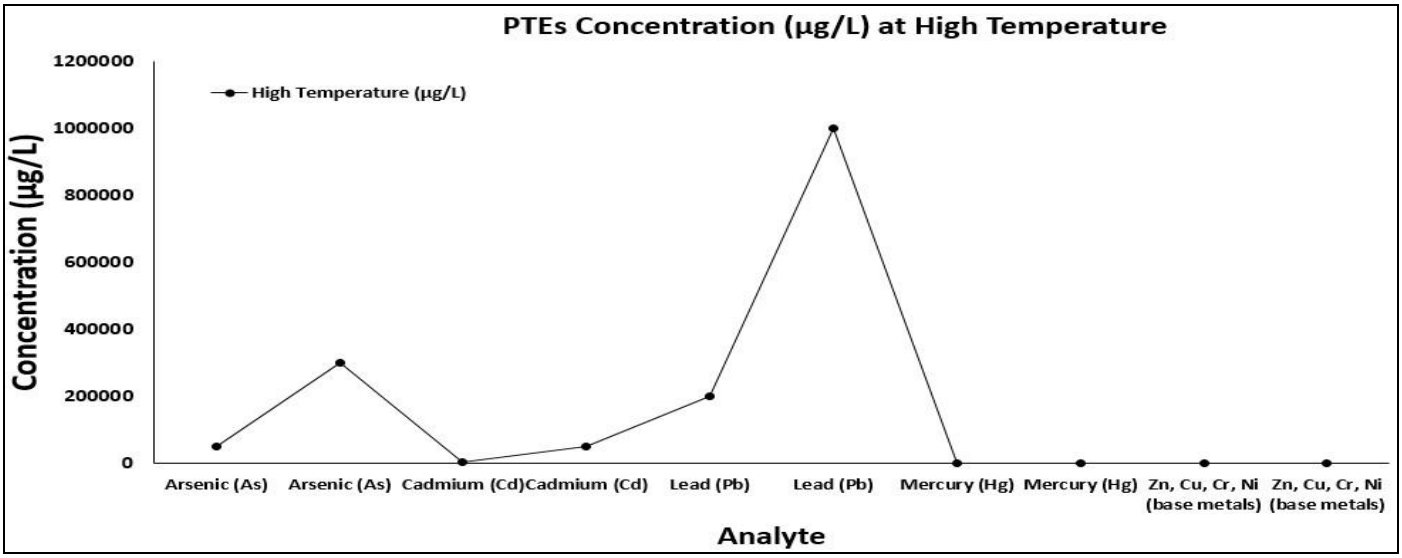

Fig 5 PTEs Concentration (µg/L) Profile Chart at High Temperatures.

The high-temperature PTE profile (see Figure 5.0) demonstrates an even more striking intensification of potential risk, with arsenic and lead exhibiting exceptionally high concentrations relative to other metals. Elevated thermal conditions can drive metal volatilization followed by condensation and enrichment in ash and fly-ash fractions, effectively concentrating toxic elements rather than eliminating them (Alloway, 2013; USEPA, 2018). Arsenic and lead emerge as dominant risk drivers due to their combined carcinogenicity, neurotoxicity, and absence of safe exposure thresholds, while cadmium contributes substantially to non-cancer toxicity through cumulative renal and skeletal effects (WHO, 2017; ATSDR, 2019).

The apparent absence of mercury Fig.4.0 in solid residues does not imply risk mitigation, but rather highlights the importance of cross-media risk assessment, as mercury is likely volatilized and redistributed into the gas phase under high-temperature conditions (UNEP, 2019). This redistribution underscores that contaminant fate under thermal processing is pathway-dependent, and risk may simply shift from solid or aqueous media to atmospheric exposure routes.

From a human health perspective, the magnitude of arsenic, lead, and cadmium concentrations observed under high-temperature conditions necessitates the use of chronic daily intake (CDI), hazard quotient (HQ), hazard index (HI), and cancer risk (ILCR) frameworks to adequately characterize both individual and cumulative risks (USEPA, 2018; WHO, 2010). Environmentally, the enrichment of toxic metals in ash residues raises concerns regarding leaching, soil contamination, and long-term ecological exposure if residues are improperly managed or reused.

B. Risk Characterization (Baseline CDI/HQ/ILCR) and Sensitivity Analysis of Exposure and Risk Parameters

➢ *Benzo[a]Pyrene (BaP): Disproportionate Cancer Risk from Solid Matrices*

The BaP soil ingestion results (Table 5) demonstrate an extreme contrast between low- and high-concentration scenarios, underscoring the nonlinear relationship between concentration and carcinogenic risk (USEPA, 2017; Boström et al., 2002). At low BaP concentrations (1 µg/kg), incremental lifetime cancer risk (ILCR) values for both adults and children are several orders of magnitude below commonly applied regulatory benchmarks, indicating negligible risk under background exposure conditions (USEPA, 2017; WHO, 2010). However, at elevated concentrations characteristic of high-end low-temperature solid residues (200,000 µg/kg), ILCR values increase dramatically, exceeding 10^{-4} for adults and reaching the 10^{-3} range for children.

This marked escalation highlights two critical features of BaP-driven risk. First, BaP toxicity is dominated by its cancer slope factor, such that even moderate increases in concentration translate into disproportionately large increases in ILCR (USEPA, 2017; IARC, 2010). Second, children consistently experience higher risk than adults due to higher soil ingestion rates relative to body weight (USEPA, 2011; ATSDR, 2022). Although pyrolysis char/biochar exhibits lower maximum BaP concentrations than low-temperature solid residues, the resulting ILCR values still exceed conservative screening thresholds, indicating that solid by-products from thermal processing can remain carcinogenically relevant even after further thermal treatment (Abdel-Shafy & Mansour, 2016; Katsoyiannis et al., 2011).

➢ *Arsenic (As): Pathway-Dependent Risk Dominance*

Arsenic risk estimates (Table 6) reveal a strong dependence on exposure pathway and matrix type, consistent with its high mobility and well-documented aqueous toxicity (WHO, 2017; USEPA, 2018). For solids and biochar assessed via soil ingestion, hazard quotients (HQs) remain below unity at low concentrations, suggesting limited non-cancer risk under conservative assumptions (USEPA, 2004; ATSDR, 2019). However, at higher solid-phase concentrations (50 mg/kg), child HQ values exceed unity, indicating potential for chronic non-cancer effects, while ILCR values approach or exceed commonly accepted cancer risk thresholds (IARC, 2012; USEPA, 2018).

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In contrast, arsenic present in ash or fly-ash leachate produces orders-of-magnitude higher risk estimates when evaluated through the drinking-water ingestion pathway. Even at the low leachate concentration (1 mg/L), both adult and child HQs greatly exceed unity, and ILCR values surpass acceptable risk levels (WHO, 2017; USEPA, 2018). At the high leachate concentration (300 mg/L), both cancer and non-cancer risk estimates reach extreme values, reflecting a hypothetical but highly consequential worst-case exposure scenario. These results emphasize that arsenic risk is not only concentration-dependent but also fundamentally pathway-driven, with aqueous exposure representing the dominant risk vector (Alloway, 2013; USEPA, 2004).

- *Cadmium (Cd): Non-Cancer Toxicity as the Primary Concern*

Cadmium results (Table 7) indicate that non-cancer toxicity overwhelmingly governs health risk across all matrices and exposure scenarios, consistent with its toxicological classification as a chronic renal toxicant (ATSDR, 2019; WHO, 2010). Solid-phase cadmium concentrations produce HQ values well below unity under soil ingestion, even at higher concentrations, suggesting limited risk from incidental contact with solids alone (USEPA, 2011). However, once cadmium is mobilized into an aqueous phase, risk estimates increase sharply.

Both low and high cadmium concentrations in ash leachate yield HQ values far exceeding unity for adults and children, with child HQs consistently higher due to greater intake per unit body weight (USEPA, 2018; ATSDR, 2019). These findings reinforce that leachability and water-mediated exposure, rather than bulk solid concentration, are the critical determinants of cadmium-related health risk (WHO, 2010; Alloway, 2013).

- *Lead (Pb): High Exposure Potential and Regulatory Concern*

Lead exposure estimates (Table 8) further illustrate the significance of exposure pathway and concentration magnitude. Soil ingestion of lead-contaminated solids yields chronic daily intake (CDI) values that increase by several orders of magnitude between low and high concentration scenarios, with children again exhibiting substantially higher intake rates than adults (USEPA, 2011; WHO, 2017). While quantitative cancer or HQ metrics are not applied here due to lead's non-threshold neurotoxicity, the CDI values applying equations (1, 2, 3, and 4) alone indicate exposure levels of potential concern, particularly for children (WHO, 2017; ATSDR, 2020).

The water ingestion pathway produces especially high CDI values using equations (1, 2, and 4) for both low and high ash leachate concentrations, reflecting the efficiency of aqueous exposure routes. These results align with regulatory consensus that any avoidable lead exposure, especially in drinking water, poses an unacceptable public health risk, even in the absence of explicit numerical thresholds (WHO, 2017; USEPA, 2018).

- *Base Metals (Zn, Cu, Cr, Ni): Comparative Risk Context*

The base metal concentrations summarized in Table 9 are several orders of magnitude lower than those of arsenic, cadmium, and lead, providing important context for mixture-based risk interpretation (Alloway, 2013). Although these metals can contribute to cumulative toxicity and ecological stress under certain conditions, their comparatively low concentrations suggest a secondary role in driving overall human health risk within the assessed scenarios (Kortenkamp et al., 2009; USEPA, 2017). Nonetheless, their presence remains relevant for long-term environmental accumulation and potential synergistic effects in complex contaminant mixtures (WHO, 2010).

Collectively, Tables 5–9 demonstrate that thermal processing residues can generate highly heterogeneous risk profiles, where health outcomes are governed not simply by total contaminant load but by a combination of concentration extremes, toxic potency, exposure pathway, and receptor sensitivity (USEPA, 2004; WHO, 2010). PAHs such as BaP dominate carcinogenic risk in solid matrices, while arsenic and cadmium drive both cancer and non-cancer risk through water-mediated exposure (IARC, 2010; ATSDR, 2019). Children consistently emerge as the most vulnerable population due to higher intake-to-body-weight ratios (USEPA, 2011).

These findings strongly support the application of pathway-specific CDI, HQ, HI, and ILCR frameworks, as well as conservative screening assumptions, to capture worst-case exposure scenarios (USEPA, 2017; USEPA, 2018). They further underscore the importance of residue characterization, leachability testing, and exposure pathway control when evaluating the human health and environmental implications of thermally modified wood processing by-products (Alloway, 2013; WHO, 2017).

- *Conservative Screening Assumptions and Quantitative Risk Characterization*

The quantitative risk assessment was conducted using a set of conservative screening assumptions, which were deliberately selected to avoid underestimation of potential human health risks while allowing for subsequent refinement under site-specific conditions (USEPA, 2004; USEPA, 2018). All assumptions are explicitly stated so that they can be modified if empirical exposure or bioavailability data become available, in accordance with standard human health risk assessment practice (WHO, 2010).

For population characteristics, an adult body weight of 70.0 kg and a child body weight of 15.0 kg were assumed, consistent with default exposure factors used in residential risk assessment scenarios (USEPA, 2011; WHO, 2017). These values reflect conservative yet widely accepted parameters that facilitate comparability with international regulatory benchmarks.

Intake rates were defined to represent realistic but health-protective exposure conditions. Drinking-water ingestion rates of 2.0 L/day for adults and 1.0 L/day for children were applied, reflecting higher relative water

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consumption among children when normalized to body weight (USEPA, 2011; WHO, 2017). Incidental soil and dust ingestion rates were assumed to be 50 mg/day for adults and 100 mg/day for children, consistent with default residential exposure assumptions that account for hand-to-mouth behavior, particularly in younger populations (USEPA, 2011; ATSDR, 2019).

A bioavailability or leachable fraction of 1.0 (100**%)** was applied for all analytes and exposure pathways to represent a conservative upper-bound assumption in the absence of compound- or matrix-specific bioaccessibility data (USEPA, 2004; Ruby et al., 2016). This approach ensures that calculated exposure doses and associated risks are not underestimated during screening-level assessment.

Toxicity parameters were selected to support conservative screening-level risk characterization. An oral cancer slope factor of 1.0 $(mg/kg\text{-}day)^{-1}$ was applied for benzo[a]pyrene (BaP)**,** consistent with U.S. EPA Integrated Risk Information System (IRIS) guidance and widely used toxicity equivalency approaches (USEPA, 2017; ATSDR, 2022). Arsenic was assigned an oral cancer slope factor of 1.5 $(mg/kg\text{-}day)^{-1}$**,** along with a non-cancer reference dose (RfD) of 0.0003 mg/kg-day, reflecting its classification as a Group 1 human carcinogen and its well-documented chronic toxicity (IARC, 2012; USEPA, 2018; WHO, 2017). Cadmium non-cancer risk was evaluated using a reference dose of 0.0005 mg/kg-day**,** consistent with toxicological evidence linking chronic exposure to renal and skeletal effects (ATSDR, 2019; WHO, 2010).

Exposure pathway mapping was conducted to conservatively associate each environmental matrix with plausible human intake routes. Condensate and soot fractions were treated as liquid media and evaluated through drinking-water ingestion pathways**,** while ash and fly-ash were conservatively assumed to release leachable fractions into water, thereby contributing to aqueous exposure (USEPA, 2004; Alloway, 2013). Solid matrices, including biochar and low-temperature solid residues, were assessed using incidental soil and dust ingestion pathways**,** consistent with residential land-use assumptions (USEPA, 2011).

Standardized unit conversions were applied throughout the assessment to maintain dimensional consistency and calculation transparency. Concentrations reported in micrograms per liter (µg/L) and micrograms per kilogram (µg/kg) were converted to milligrams per liter (mg/L) and milligrams per kilogram (mg/kg), respectively, using a factor of 0.001, while soil ingestion rates expressed in milligrams per day were converted to kilograms per day by dividing by 1,000,000 (USEPA, 2004; USEPA, 2018). Collectively, these assumptions establish a transparent, reproducible, and health-protective framework suitable for screening-level human health risk characterization.

- *Quantitative Risk Characterization*

This section provides illustrative screening calculations to demonstrate how standard human health risk assessment frameworks translate measured concentrations from thermally modified wood residues into potential exposure outcomes. These examples synthesize published toxicity factors, regulatory benchmarks, and exposure assumptions and are not presented as original field data (USEPA, 2011; WHO, 2017). The purpose is to contextualize how high-end concentrations can produce screening-level risk exceedances under conservative upper-bound scenarios (ATSDR, 2019; IARC, 2020; USEPA, 2022).

✓ *Benzo[a]Pyrene (BaP) in Low-Temperature Solid Residue*

Benzo[a]pyrene (BaP), a high-molecular-weight PAH and recognized carcinogenic marker compound, was evaluated for incidental soil ingestion using the highest measured concentration in low-temperature residue (200,000 µg/kg), consistent with established PAH exposure benchmarks (IARC, 2010; Zhang et al., 2021). Converting to 200 mg/kg and applying adult ingestion parameters (equations 1,2, and 4) produced a CDI of 1.43×10^{-4} mg/kg-day and using equation (6) an ILCR of 1.4×10^{-4}, consistent with upper-bound carcinogenic risk responses for high-molecular-weight PAHs (USEPA, 2017; ATSDR, 2022).

Child receptor calculations using equation (6) (1.33×10^{-3} mg/kg-day; ILCR = 1.33×10^{-3}) exceeded typical risk thresholds by over an order of magnitude, reflecting higher exposure per body mass and behavioral ingestion sensitivity (USEPA, 2011; Dong & Zhang, 2023). These findings align with evidence that BaP risk scales disproportionately with concentration due to its slope factor-driven carcinogenic potency (Kim et al., 2013; Squillace et al., 2019).

✓ *Arsenic in High-Temperature Ash / Fly-Ash (Leachate Scenario)*

Arsenic risk outcomes are strongly pathway-dependent, particularly when high-temperature residues are assumed to be leachable to water (Alloway, 2013; Kumar et al., 2020). Under the conservative assumption of complete leachability (300,000 µg/L → 300 mg/L), adult using equation (2) CDI reached 8.57 mg/kg-day with equation (6) an ILCR of 12.9, greatly exceeding recommended screening thresholds (Smith et al., 2002; USEPA, 2018; Ravenscroft et al., 2020). Corresponding HQ values applying equation (5) (>28,000) indicate severe theoretical non-cancer risk implications (WHO, 2017; Naujokas et al., 2013).

Child ingestion produced even higher risk estimates (equations (1, 2 and 4) CDI = 20 mg/kg-day; equation (5) HQ >66,000; equation (6) ILCR ≈ 30), reinforcing that water-mediated arsenic exposure is the dominant driver of public health concern (ATSDR, 2019; USEPA, 2022). These values represent an upper-bound screening case, illustrating how arsenic mobility, not concentration alone, determines real-world hazard potential (Katsoyiannis et al., 2011; Wang et al., 2020).

✓ *Cadmium in High-Temperature Ash / Fly-Ash (Leachate Scenario)*

Cadmium toxicity is primarily associated with non-cancer renal and skeletal effects, particularly when mobilized through aqueous pathways (Järup & Akesson, 2009; Briffa et al., 2020; Xu et al., 2023). Using a high-end ash concentration

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of 50,000 µg/L (50 mg/L), CDI (equation 1, 2, and 4) calculations produced HQ (equation 5) values of 2,860 (adult) and 6,670 (child), far exceeding non-cancer thresholds (WHO, 2010; USEPA, 2018). These findings support established evidence that cadmium risk magnitude increases sharply when environmental conditions favor dissolution and ingestion (ATSDR, 2019; Ribeiro et al., 2024).

Table 5 Benzo[a]pyrene (BaP) Soil Ingestion for Solids/Biochar

Matrix	Level	Conc (µg/kg)	CDI_adult (mg/kg·day)	CDI_child (mg/kg·day)	ILCR_adult (BaP)	ILCR_child (BaP)
Low-T solid residue	Low	1	7.142857×10^{-10}	6.666667×10^{-9}	7.142857×10^{-10}	6.666667×10^{-9}
Low-T solid residue	High	200,000	1.428571×10^{-4}	1.333333×10^{-3}	1.428571×10^{-4}	1.333333×10^{-3}
Pyrolysis char/biochar	Low	1	7.142857×10^{-10}	6.666667×10^{-9}	7.142857×10^{-10}	6.666667×10^{-9}
Pyrolysis char/biochar	High	5,000	3.571429×10^{-6}	3.333333×10^{-5}	3.571429×10^{-6}	3.333333×10^{-5}

Table 6 Arsenic (As)

Matrix	Level	Conc	Pathway	CDI_adult (mg/kg·day)	CDI_child (mg/kg·day)	ILCR_adult (×)	ILCR_child (×)	HQ_adult	HQ_child
Solids / biochar	Low	100 µg/kg (0.1 mg/kg)	Soil ingestion	7.142857×10^{-8}	6.666667×10^{-7}	1.071429×10^{-7}	1.000000×10^{-6}	0.0002380952	0.0022222222
Solids / biochar	High	50,000 µg/kg (50 mg/kg)	Soil ingestion	3.571429×10^{-5}	3.333333×10^{-4}	5.357143×10^{-5}	5.000000×10^{-4}	0.1190476190	1.1111111111
Ash / fly-ash (leachate)	Low	1,000 µg/L (1 mg/L)	Water ingestion	0.0285714286	0.0666666667	0.0428571429	0.1000000000	95.2380952381	222.2222222222
Ash / fly-ash (leachate)	High	300,000 µg/L (300 mg/L)	Water ingestion	8.5714285714	20.0000000000	12.8571428571	30.0000000000	28571.4285714	66666.6666667

Table 7 Cadmium (Cd)

Matrix	Level	Conc	Pathway	CDI_adult	CDI_child	HQ_adult	HQ_child
Solids / biochar	Low	9 µg/kg (0.009 mg/kg)	Soil ingestion	6.428571×10^{-9}	6.000000×10^{-8}	1.285714×10^{-5}	0.00012
Solids / biochar	High	5,000 µg/kg (5 mg/kg)	Soil ingestion	3.571429×10^{-6}	3.333333×10^{-5}	0.0071428571	0.0666666667
Ash / fly-ash (leachate)	Low	500 µg/L (0.5 mg/L)	Water ingestion	0.0142857143	0.0333333333	28.5714285714	66.6666666667
Ash / fly-ash (leachate)	High	50,000 µg/L (50 mg/L)	Water ingestion	1.4285714286	3.3333333333	2857.1428571429	6666.6666666667

Table 8 Lead (Pb)

Matrix	Level	Conc	Pathway	CDI_adult (mg/kg-day)	CDI_child (mg/kg-day)
Solids / biochar	Low	1,000 µg/kg (1.0 mg/kg)	Soil ingestion	7.142857×10^{-7}	6.666667×10^{-6}
Solids / biochar	High	200,000 µg/kg (200 mg/kg)	Soil ingestion	1.428571×10^{-4}	1.333333×10^{-3}

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Ash / fly-ash (leachate)	Low	100,000 µg/L (100 mg/L)	Water ingestion	2.8571428571	6.6666666667
Ash / fly-ash (leachate)	High	1,000,000 µg/L (1000 mg/L)	Water ingestion	28.5714285714	66.6666666667

Table 9 Base Metals (Zn, Cu, Cr, Ni)

Matrix	Low (µg/kg)	High (µg/kg)
Solids / biochar	100 µg/kg	10 µg/kg
Ash / fly-ash	100 µg/L	1 µg/L

Table 10 Conservative Exposure Assumptions and Toxicity Parameters Used for Screening-Level Human Health Risk Assessment

Category	Parameter	Symbol	Adult Value	Child Value	Units	Rationale / Reference
Population characteristics	Body weight	BW	70.0	15.0	Kg	Standard residential defaults (USEPA, 2011; WHO, 2017)
Water ingestion	Drinking-water ingestion rate	IR_{Water}	2.0	1.0	L/day	Conservative residential exposure (USEPA, 2011; WHO, 2017)
Soil/dust ingestion	Soil ingestion rate	$IR_{Soil/dust}$	50	100	mg/day	Accounts for incidental ingestion and hand-to-mouth behavior (USEPA, 2011; ATSDR, 2019)
Bioavailability / leachability	Bioavailable fraction	BA	1.0	1.0	unitless	Conservative upper-bound assumption (USEPA, 2004; Ruby et al., 2016)
BaP toxicity (cancer)	Oral cancer slope factor	CSF_{BaP}	1.0	1.0	$(mg/kg\text{-}day)^{-1}$	EPA IRIS screening value (USEPA, 2017; ATSDR, 2022)
Arsenic toxicity (cancer)	Oral cancer slope factor	CSF_{As}	1.5	1.5	$(mg/kg\text{-}day)^{-1}$	Group 1 carcinogen (IARC, 2012; USEPA, 2018)
Arsenic toxicity (non-cancer)	Reference dose	RfD_{As}	0.0003	0.0003	mg/kg-day	Chronic toxicity benchmark (WHO, 2017)
Cadmium toxicity (non-cancer)	Reference dose	RfD_{Cd}	0.0005	0.0005	mg/kg-day	Renal toxicity endpoint (ATSDR, 2019; WHO, 2010)
Exposure pathway mapping	Condensate / soot	—	Water ingestion	Water ingestion	—	Treated as liquid phase (USEPA, 2004)
	Ash / fly-ash	—	Water ingestion (leachate)	Water ingestion (leachate)	—	Conservative leachability assumption (Alloway, 2013)
	Solids / biochar	—	Soil ingestion	Soil ingestion	—	Residential land-use scenario (USEPA, 2011)
Unit conversions	Concentration conversion	—	1 µg/L = 0.001 mg/L; 1 µg/kg = 0.001 mg/kg	—	Standardized dimensional consistency (USEPA, 2004; USEPA, 2018)	
	Soil ingestion conversion	—	mg/day ÷ 1,000,000 = kg/day	—	Exposure calculation transparency	

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Table 11 Summary of Illustrative Screening-Level Risk Characterization

Contaminant	Matrix	Exposure Pathway	High-End Concentration	Primary Risk Metric	Adult Result	Child Result	Risk Interpretation
Benzo[a]pyrene (BaP)	Low-T solid residue	Soil ingestion	200,000 µg/kg	ILCR	1.4×10^{-4}	1.3×10^{-3}	Exceeds cancer benchmarks; child risk > order of magnitude higher
Arsenic (As)	Ash / fly-ash (leachate)	Water ingestion	300,000 µg/L	ILCR / HQ	ILCR ≈ 12.9; HQ ≈ 28,600	ILCR ≈ 30; HQ ≈ 66,000	Extreme cancer and non-cancer risk (worst-case scenario)
Cadmium (Cd)	Ash / fly-ash (leachate)	Water ingestion	50,000 µg/L	HQ	≈ 2,860	≈ 6,670	Severe non-cancer toxicity potential

Tables 10 and 11 collectively illustrate how conservative exposure assumptions, when systematically applied to measured contaminant concentrations, translate into meaningful indicators of potential human health risk (USEPA, 2004; WHO, 2010; USEPA, 2018). Table 10 establishes a transparent and reproducible framework of population characteristics, intake rates, toxicity parameters, and exposure pathway mappings that reflect internationally accepted screening defaults (USEPA, 2011; WHO, 2017). These assumptions are intentionally health-protective, ensuring that calculated risks are not underestimated in the absence of site-specific exposure or bioavailability data (USEPA, 2004; Ruby et al., 2016). By explicitly documenting each parameter, the framework allows for subsequent refinement while maintaining comparability with regulatory benchmarks used in residential risk assessment contexts (USEPA, 2018).

The exposure assumptions summarized in Table 10 highlight the disproportionate vulnerability of children relative to adults. Lower body weight combined with higher normalized ingestion rates results in consistently elevated chronic daily intake estimates for child receptors across all exposure pathways (USEPA, 2011; WHO, 2017). This methodological structure aligns with established risk assessment practice, which recognizes children as the most sensitive subpopulation in scenarios involving incidental soil ingestion and drinking-water exposure (USEPA, 2011; ATSDR, 2019). The application of a unity bioavailability factor further reinforces the conservative nature of the assessment, representing an upper-bound scenario in which all contaminants are assumed to be fully bioaccessible and systemically absorbed (USEPA, 2004; Ruby et al., 2016).

Table Y translates this conservative framework into illustrative screening-level risk estimates for representative PAHs and PTEs. The results demonstrate that even under simplified exposure scenarios, high-end concentrations observed in thermally modified wood residues and by-products can yield risk estimates that substantially exceed commonly accepted cancer and non-cancer thresholds (USEPA, 2017; WHO, 2010). For benzo[a]pyrene, soil ingestion of solid residues produces incremental lifetime cancer risk values that exceed regulatory benchmarks, particularly for children (IARC, 2010; ATSDR, 2022; USEPA, 2017). This finding underscores the disproportionate influence of high-molecular-weight PAHs on carcinogenic risk, even when present in solid matrices and evaluated under incidental ingestion pathways (Boström et al., 2002; Abdel-Shafy & Mansour, 2016).

For arsenic and cadmium, Table 11 reveals the dominant role of aqueous exposure pathways when ash or fly-ash is conservatively assumed to be fully leachable. Under these conditions, drinking-water ingestion becomes the primary driver of risk, resulting in hazard quotients and cancer risk estimates that exceed acceptable levels by several orders of magnitude (USEPA, 2004; WHO, 2017; ATSDR, 2019). Although these scenarios represent worst-case screening assumptions, they clearly demonstrate the potential for thermal processing residues to act as secondary sources of severe human health risk if mobilized into water systems (Alloway, 2013; USEPA, 2018). The magnitude of these exceedances emphasizes the importance of evaluating not only total contaminant concentrations but also their environmental mobility and exposure pathways (WHO, 2010; Katsoyiannis et al., 2011).

Taken together, Tables 10 and 11 reinforce the necessity of pathway-specific and mixture-aware risk assessment approaches when evaluating contaminants generated during thermal wood processing (Kortenkamp et al., 2009; USEPA, 2017). The findings illustrate that risk is governed not only by contaminant toxicity but also by exposure dynamics, matrix characteristics, and receptor sensitivity (USEPA, 2018; WHO, 2017). Importantly, the tables highlight that thermal treatment does not inherently reduce health risk and, under certain conditions, may concentrate contaminants into forms with enhanced toxicological and regulatory significance (Alloway, 2013; Abdel-Shafy & Mansour, 2016). These results support the application of chronic daily intake, hazard quotient, hazard index, and incremental lifetime cancer risk frameworks as essential tools for screening-level evaluation, informing both risk management decisions and the need for more refined, site-specific assessment (USEPA, 2004; USEPA, 2018).

V. IMPLICATIONS FOR HEALTH AND ENVIRONMENT

➢ *Low-Temperature PTEs*

Overall, the low-temperature PTE concentration profile demonstrates that thermal processing can concentrate toxic metals rather than eliminate them, resulting in residues with

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substantial chronic human health and ecological risk potential. Arsenic and lead emerge as dominant risk drivers due to their well-established carcinogenicity and the absence of safe exposure thresholds, while cadmium contributes significantly to non-cancer toxicity through bioaccumulation and long-term renal effects (ATSDR, 2019; WHO, 2017). Although present at comparatively lower levels, base metals further contribute to cumulative toxicity and ecological stress when considered within a mixture context.

The observed concentration patterns support the application of chronic daily intake (CDI) using equation (2), hazard quotient (HQ) using equation (5), and mixture-based hazard index (HI) approaches, rather than reliance on total concentration metrics alone. In addition, the persistence and potential mobility of metals emphasize the need for leachability testing, controlled residue handling, and long-term environmental monitoring, as low-temperature residues can act as secondary sources of metal exposure through soil–water–biota transfer pathways (Alloway, 2013; USEPA, 2018).

- *Low-Temperature PAHs*

The low-temperature PAH profile demonstrates that health and environmental risk is not solely governed by the most toxic individual compounds, but by the combined presence of multiple PAHs with differing physicochemical properties, environmental behaviors, and toxic potencies (Kortenkamp et al., 2009; USEPA, 2017). Elevated concentrations of low- and intermediate-molecular-weight PAHs increase the probability of exposure through inhalation, ingestion, and dermal contact, while even trace levels of high-molecular-weight PAHs particularly benzo[a]pyrene (BaP) can disproportionately influence carcinogenic risk (ATSDR, 2022; IARC, 2010).

This compositional pattern supports the application of incremental lifetime cancer risk (ILCR) and mixture-based hazard assessment frameworks, rather than reliance on concentration thresholds or single-compound evaluations alone (USEPA, 2017; WHO, 2010). Environmentally, the dominance of more mobile PAHs enhances cross-media transport from solid residues into water and air, while the persistence of higher-ring PAHs raises concerns regarding long-term contamination, sediment accumulation, and bioaccumulation in food webs (Abdel-Shafy & Mansour, 2016; Katsoyiannis et al., 2011).

- *High-Temperature PAHs*

The high-temperature PAH profile reveals a qualitative and quantitative escalation of risk relative to low-temperature conditions. Although lighter PAHs continue to contribute substantially to overall exposure, the pronounced enrichment of high-molecular-weight PAHs particularly BaP fundamentally alters the risk profile, shifting it toward cancer-dominated outcomes (IARC, 2010; ATSDR, 2022).

This distribution strongly supports the use of ILCR using equation (6) modeling, BaP-equivalent toxicity (BaP-TEQ) calculations, and mixture-based risk frameworks, rather than reliance on absolute concentrations or individual PAH metrics alone (USEPA, 2017; Kortenkamp et al., 2009). From an environmental perspective, the increased abundance of particle-bound high-ring PAHs elevates the likelihood of sediment contamination, bioaccumulation, and persistent ecological impacts that may continue long after active emissions cease (Katsoyiannis et al., 2011; Wang et al., 2020).

- *High-Temperature PTEs*

The high-temperature PTE profile demonstrates a dramatic intensification of toxic metal concentrations, indicating that elevated thermal processing conditions can substantially exacerbate human health and environmental risks rather than mitigate them. Arsenic and lead again emerge as dominant contributors to overall risk, while cadmium significantly amplifies non-cancer toxicity through cumulative and mixture effects. The absence of mercury in solid residues highlights the importance of cross-media risk considerations, as volatilized contaminants may be redistributed into the atmosphere rather than eliminated.

These findings strongly support the application of chronic daily intake (CDI) using equations (1, 2, 3, and 4), hazard quotient (HQ) using equation (5), hazard index (HI), and cancer risk (ILCR) using equation (6) frameworks, alongside pathway-integrated and mixture-based risk assessment approaches (USEPA, 2018; WHO, 2017). Collectively, the results underscore that high-temperature thermal systems can generate residues of exceptional toxicological and regulatory significance, necessitating stringent emission controls, secure residue management, and long-term environmental monitoring to prevent secondary contamination and chronic exposure.

VI. CONCLUSIONS AND RECOMMENDATIONS

A. Conclusions

The review demonstrates that thermally modified wood processing can generate contaminants of substantial toxicological relevance, particularly when high-temperature conditions promote molecular condensation, elemental enrichment, and aqueous mobility. Polycyclic aromatic hydrocarbons (PAHs), especially benzo[a]pyrene, were identified as dominant drivers of carcinogenic risk in solid residues, while arsenic, cadmium, and lead exhibited high leachate-driven risk potential, with exposure scenarios exceeding conservative screening thresholds by several orders of magnitude.

Risk outcomes were shown to be governed not merely by total contaminant concentration but by pathway, bioavailability, and receptor sensitivity, with children consistently presenting the highest calculated CDI, HQ, and ILCR values. These findings confirm that thermal treatment does not inherently reduce contaminant risk; in certain operational ranges, it can amplify hazard magnitude by concentrating PAHs and PTEs into more mobile and biologically relevant forms.

A precautionary, pathway-specific risk management framework is therefore necessary to ensure safe handling,

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reuse, or disposal of thermally derived residues. The integration of standardized contaminant testing, conservative exposure modeling, and temperature-controlled modernization of processing systems is essential to mitigate potential public health and environmental impacts. Collectively, the evidence supports the continued development of risk-based regulation, residue certification mechanisms, and targeted research into mixture toxicity, speciation dynamics, and long-term environmental fate.

B. Recommendations

➢ *Adopt Temperature-Optimized Process Controls*

- Implement controlled thermal regimes and real-time temperature monitoring systems to minimize PAH formation and PTE enrichment at critical temperature thresholds.
- Prioritize rapid quenching, oxygen limitation, and vapor residence-time management to reduce formation of high-molecular-weight PAHs such as benzo[a]pyrene.

➢ *Mandatory Residue and Leachate Characterization*

- Require standardized analysis of both total and leachable contaminant fractions (e.g., ΣPAHs, BaP-TEQ, TCLP/SPLP/LEAF metals).
- Integrate bioaccessibility testing into regulatory compliance rather than relying solely on total concentrations, which may overestimate or underestimate actual exposure potential.

➢ *Exposure Pathway-Specific Mitigation Strategies*

- For aqueous pathways: implement engineered containment, leachate capture, and water-treatment barriers before release or reuse of ash and fly-ash.
- For solid residues: apply dust suppression, controlled storage, and restricted occupational handling to prevent soil and inhalation exposure.

➢ *Protection of Sensitive Populations*

- Use child-focused exposure assumptions and lead-specific models (e.g., IEUBK) during screening because children show disproportionately higher CDI, HQ, and ILCR values.
- Restrict agricultural or residential reuse of residues exceeding PAH or metal screening criteria until further site-specific assessment is completed.

➢ *Regulation, Traceability, and Product Certification*

- Introduce certification systems for biochar, ash, and pyrolysis by-products confirming contaminant thresholds prior to commercial sale or land application.
- Align national regulatory limits with WHO/USEPA benchmarks for water pathways and IARC classifications for carcinogenic PAHs.

➢ *Prioritize Research on Mixture Toxicity and Thermal Speciation*

- Expand research into transformation pathways of PAH derivatives (OPAHs/NPAHs) and redox-dependent metal speciation at elevated temperatures.
- Employ mechanistic and probabilistic models (for example, Monte Carlo) to constrain uncertainty and improve predictability of risk outcomes.

➢ *Conflict of Interest Statement*

The author declares that there are no known financial, personal, or institutional conflicts of interest that could have influenced the research, analysis, or interpretation presented in this review. The study was conducted independently, without involvement or sponsorship from commercial entities, manufacturers, or stakeholders associated with thermally modified wood processing or related industries. All conclusions and recommendations are based solely on the evidence and data evaluated by the author, and no external party had any role in shaping the manuscript content, data selection, or publication decision.

➢ *AI Usage Disclosure Statement*

The author acknowledges the use of generative artificial intelligence tools during the preparation of this manuscript. OpenAI's ChatGPT (GPT-5.1, 2025) was used to support selected stages of manuscript development, including:

- Language refinement and editing to improve clarity and readability;
- Assistance in structuring sections (for example, abstract, main body, and conclusion refinement);
- Generating draft text for concept explanation and risk assessment terminology; and
- Providing suggestions for graphical abstract layout and formatting.

All scientific interpretations, numerical values, data assessments, and final conclusions were reviewed, verified, and approved by the author. No AI tools were used to generate, alter, or manipulate experimental data, statistical outcomes, or research findings. The author retains full responsibility for the accuracy, integrity, and originality of the content presented in this manuscript.

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