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Understanding the Impact of Air Quality on the Incidence of Polycyclic Aromatic Hydrocarbons (PAHs)

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ABSTRACT

Polycyclic aromatic hydrocarbons (PAHs) are environmental contaminants predominantly linked to chronic respiratory diseases. Heightened epidemiological evidence demands a focused initiative to enhance knowledge about the impact of air quality on the incidence of certain PAHs in airborne particulates. Polycyclic aromatic hydrocarbons (PAHs) are linked to respiratory issues such as asthma, aggravation , chronic bronchitis, and emphysema. The study provides an overview of contemporary PAH exposure and its hazardous effects on the respiratory system. A literature search across four scientific databases identified 120 pertinent research publications that examine urine concentrations of diverse persistent PAHs and associated biomarkers. The study emphasized the hazards associated with PM2.5-PAH conjugates, which can induce mutagenesis, carcinogenesis, and teratogenesis; disrupt signaling pathways; and lead to oxidative stress, acute and/or chronic respiratory ailments, cognitive deficits, cardiovascular issues, and mortality. The research highlights the considerable toxicity of PAHs and their metabolites to the respiratory system, activating interrelated AhR/non-AhR signaling pathways that result in oxidative stress, immune system impairment, asthma/COPD, and cancer. The study mostly demonstrates favorable connections between PAHs and respiratory toxicity.

Keywords: Biomarkers, Oxidative Stress, Polycyclic Aromatic Hydrocarbons.

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INTRODUCTION

The present situation observes a swift expansion of industrialization and urbanization, resulting in a marked rise in numerous pollutants, including polycyclic aromatic hydrocarbons (PAHs) [1]. Polycyclic aromatic hydrocarbons (PAHs) are widespread, persistent hazardous xenobiotic compounds released from natural events like wildfires and volcanic eruptions, as well as human activities such as incomplete combustion of fossil fuels and industrial processes. Polycyclic aromatic hydrocarbons (PAHs) are a common category of environmental contaminants, usually appearing as complex mixtures of over 300 chemical molecules with numerous aromatic rings. These substances are significant due to their widespread presence and ability to generate carcinogenesis, genetic mutations, and toxicity. Polycyclic aromatic hydrocarbons (PAHs) consist predominantly of 2 to 6 fused aromatic rings arranged in various configurations. Nine compounds are classified as 'petrogenic PAHs' (alkylated or oxygenated PAHs resulting from oil spills), while sixteen priority PAHs are designated as 'pyrogenic PAHs' (originating from fossil fuels), which are prevalent in the atmosphere. Although PAH levels fluctuate in the environment, certain highly carcinogenic variants account for merely 3% of the emissions of the sixteen priority PAHs. High molecular weight polycyclic aromatic hydrocarbons present a considerable pulmonary exposure hazard, as mutagens and carcinogens exist in both gaseous and particle states.

The proliferation of PAHs due to air pollution is acknowledged to affect the health of humans and other creatures, either directly or indirectly. Recent epidemiological studies have demonstrated a significant association between PAH exposure and respiratory illnesses as well as mortality rates. Exposure to polycyclic aromatic hydrocarbons (PAHs) and their photochemical oxidation products may lead to the development of lung cancer. Moreover, innovations in extraction methods (solid-phase microextraction, solid-phase extraction, and liquid-liquid extraction), analytical techniques such as GC-MS/MS, HPLC-MS/MS, UPLC-MS/MS, and miniaturized portable devices (microfluidic technology, electrochemical sensors, and surface-enhanced Raman spectroscopy), along with molecular and immunological assays and computational modeling, have empowered researchers to measure PAH concentrations in diverse environmental matrices and biological samples with enhanced precision and sensitivity. Furthermore, recent molecular and mechanistic investigations have clarified the

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cellular pathways implicated in PAH-induced respiratory toxicity, facilitating the formulation of innovative therapeutic strategies. Recently, the increase in anthropogenic activities has significantly amplified the emission of PAHs into soil, water, and air, exceeding their established permissible limits. Recent examinations have determined that some chemicals may be carcinogenic. Polycyclic aromatic hydrocarbons (PAHs) are categorized into four groups: 'Group 1 Carcinogenic to humans,' 'Group 2A Probably carcinogenic to humans,' 'Group 2B Possibly carcinogenic to humans,' and 'Group 3 Not classifiable regarding carcinogenicity to humans.' Specific PAHs, including chrysene (CHR), benzo[a]pyrene, benzo[a]anthracene, and benzo[fluoranthene], are recognized as mutagens, teratogens, and carcinogens.

Prior investigations into polycyclic aromatic hydrocarbons (PAHs) and their respiratory toxicity have yielded substantial knowledge; nonetheless, notable gaps persist, particularly regarding integrated mechanistic insights into PAH-induced respiratory effects and accurate exposure assessment methodologies. Addressing these deficiencies will enhance the comprehension of PAHs' respiratory toxicity and elucidate the mechanisms, biomarkers, epidemiological correlations, risk assessment methodologies, and intervention options. The authors want to investigate recent developments in assessing the current state of respiratory disorders attributed to PAHs and delineating their role in respiratory diseases. Restricting the literature review to a defined timeframe yields more precise methodologies, resulting in enhanced accuracy in the detection of PAHs and their metabolite concentrations in biological systems. The past five years have elucidated the current state of respiratory disorders attributed to PAHs and have generated epidemiological evidence concerning the function of PAHs in respiratory problems, if applicable. A literature survey within a certain timeframe presents the latest findings, augmenting the relevance and currency of the study while allowing the review to include a thorough array of contemporary studies. This study may serve as a foundation for future research aimed at delivering more precise interpretations through pre-established synergistic signaling pathways for effective therapies and creative techniques.

RESEARCH ELABORATIONS

An observational study was performed on published articles from the past five years to examine the correlation between PAH exposure and the advancement of respiratory toxicity.

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The literature survey was conducted utilizing the Elsevier-Science Direct, PubMed, Google Scholar, and Springer databases to discover recently published articles in scientific publications from MDPI, Nature, SAGE, ScienceDirect, Springer, Wiley, Hindawi, and others from April 2019 to July 2024. The pertinent keywords employed in the literature included 'PAHs exposure,' 'PAHs respiratory health,' 'PAHs toxicity mechanism,' 'PAHs metabolism,' 'PAHs metabolites,' and 'PAHs exposure effect.' Moreover, established rules pertaining to the nomenclature and classification of PAHs were also incorporated. Eligibility Criteria Original, full-text research publications, review articles, and clinical studies published in English were deemed eligible for inclusion. The study was included if it represented the effects of PAH exposure among healthy participants and/or elucidated the mechanism of action of PAH exposure. Articles detailing the correlation between hazardous PAH exposure and lung inflammation, compromised pulmonary function, lung cancer, COPD, and acute lower respiratory infections (ALRI) were also incorporated. Criteria for Exclusion.

The study eliminated individuals from non-English linguistic backgrounds, irrelevant study groups, unrelated exposures, and unrelated outcomes, as well as patients with previously documented risk factors. The study of PAHs-related respiratory toxicity involved pooling and compiling research, with an initial search yielding 3,998 studies from four distinct databases. A total of 3,892 articles were retracted from these studies, comprising 328 duplicates, 888 irrelevant articles, 1,396 excluded based on title and abstract, 573 short communications, letters to editors, and proceedings, 336 background articles from non-indexed journals, 39 excluded due to lack of full text, and 332 excluded for non-relevant keywords. Ultimately, 120 studies were selected for this review, with full-text articles meticulously analyzed, concentrating on polycyclic aromatic hydrocarbons (PAHs) and their biomarkers, biological uptake availability, extraction and quantification methods, detection limits, and mechanisms of respiratory toxicity. This review examined various PAHs and their effects on the respiratory system, including the corresponding mechanisms of action within biological systems. The chemical structures of priority PAHs were generated using ACD ChemSketch, USA version 2020.2.1, while names, abbreviations, and CAS numbers were sourced from PubChem.

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Polycyclic aromatic hydrocarbons (PAHs) released into the atmosphere are classified as Group 2A and 2B human carcinogens by the International Agency for Research on Cancer (IARC). Identifying PAH metabolites in biological samples such as mucus, urine, blood, plasma, serum, sputum, seminal plasma, and milk is essential for evaluating human exposure and comprehending their possible health implications. Metabolites of PAHs are generated in the body by metabolic processes subsequent to PAH exposure, and their detection in biological samples acts as an indicator of exposure. The identification of PAH metabolites in environmental samples (water, air, and dust) aids in evaluating the presence and dispersion of pollutants in the environment. Recent advancements in the detection of polycyclic aromatic hydrocarbons (PAHs) and their metabolites in biological and environmental samples have incorporated more precise and selective analytical techniques, including gas chromatography-mass spectrometry (GC-MS) and liquid chromatography-mass spectrometry (LC-MS), utilizing sensitive instruments such as ultraviolet-fluorescence detector mass spectrometry, high-performance liquid chromatography-tandem mass spectrometry (HPLC-MS/MS) with negative electrospray ionization, isotope-dilution liquid chromatography with tandem mass spectrometry (ID-LC-MS/MS), and ultra-performance liquid chromatography Orbitrap mass spectrometry (UPLC Orbitrap MS) analysis. Biological monitoring by urine analysis for assessing metabolic health and exposure to chemicals, medicines, and pathogens is the recommended method due to its noninvasive sample collection, minimum matrix contamination, and extensive half-life range (5 hours to 17 days). High-performance liquid chromatography-ultraviolet-fluorescence detector mass spectroscopy (HPLC-UV-FLDMS) and gas chromatography-mass spectroscopy (GCMS) are commonly employed for the determination of urine PAH metabolites due to their superior sensitivity and selectivity. Samples undergo treatment with a β-glucuronidase/arylsulfatase enzyme combination for the separation of polycyclic aromatic hydrocarbons (PAHs), followed by extraction with solid-liquid and liquid-liquid extraction techniques. The extracted PAH samples are subsequently concentrated, reconstituted in appropriate solvents, and quantified.

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RESULTS AND DISCUSSIONS

Numerous research concentrated on evaluating exposure to polycyclic aromatic hydrocarbons (PAHs) and their metabolites. These studies collectively elucidate methodologies and health effects, including potential implications for maternal and fetal health, the transfer of PAHs from mothers to infants via breastfeeding, health consequences for workers in high-risk industries, and valuable insights into environmental factors contributing to respiratory health issues. They employed gas chromatography-mass spectrometry to quantify ten urinary carboxylic acid metabolites and utilized a graphene poly(L-GA)/SPGE for the detection of 1-hydroxypyrene (1-OHP), while also evaluating urinary PAHs and their metabolites in pregnant women using HPLC-MS/MS. Quantified five urine PAH metabolites utilizing gas chromatography-mass spectrometry (GC-MS). utilized an ID-LC-MS/MS technique to examine urine PAH metabolites within a single-pollutant framework. Performed biomonitoring of PAH metabolites in lactating individuals with HPLC-MS. Examined the correlation between PAH exposure, socio-demographic variables, and PM2.5 levels in pregnant women by evaluating urine PAH metabolites using GC-MS. Quantified urine PAH metabolites in high-risk occupational cohorts utilizing GC-MS. Assessed PAHs exposure and its correlation with health outcomes in the Shiraz province population, Iran, utilizing blood and urine samples to analyze urinary 1-OHP in children at elevated asthma risk via UPLC Orbitrap MS. Liu et al. (2023) investigated the impact of PAHs exposure on lung epithelium in coke plant workers, evaluating PAHs and nicotine biomarkers in urine alongside the lung epithelium damage biomarker (CC16) in plasma samples.

Polycyclic aromatic hydrocarbons (PAHs) associated with PM2.5 are regarded as harmful and are likely to exhibit mutagenic, carcinogenic, and teratogenic properties. In ambient air, multi-ring PAHs are more significantly adsorbed onto PM2.5 (66%–86%) compared to PM10, whereas low-ring PAHs are mostly collected on the PM10 surface (83%–86%). The primary source of PAH exposure by inhalation of fumes or ingestion of grilled food is the incomplete combustion of biomass during food grilling. The greatest concentration of PAHs was identified in charcoal-grilled food (382.02 μg/m^3), whereas the least concentration was noted in gas-grilled food (1.44 μg/m^3). The coal grilling process predominantly produced 4-ring PAHs, with coal ash exhibiting a high concentration of FLU and PYR PAHs. An appreciable elevation in NAP, ACE, FLA, PHE, PYR, and B[a]P was seen in urine samples

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collected during the hours of grilling. Air monitoring conducted over three years in Dalian, North China, identified 53 PAH chemicals in both gas and particle phases, with concentrations varying from 40 to 240 ng/m³, averaging 95 ± 40 ng/m³. This comprised 26 alkylated PAHs (mean 17 ± 7.6 ng/m³), 16 priority PAHs (mean 68 ± 33 ng/m³), and 4 high-molecular-weight PAHs (mean 1.3 ± 1.3 ng/m³). The population attributable fraction for cancer cases linked to ambient PAH exposure in an urban setting was recorded at 12.0‰, with 35.7 cases per million individuals attributed to excessive inhalation of PAHs over an average lifespan of 70 years. A total of 12 polycyclic aromatic hydrocarbons (PAHs) and their corresponding metabolites were examined in 170 ambient air samples collected from Chinese cities engaged in coal and oil production, and these were compared to their relative incremental lifetime cancer risk (ILCR). The initial ILCR for the heating season (April-October) was documented as 2.65×10^-9 in coal-producing cities and 4.60×10^-9 in oil-producing cities. In the non-heating season (November-March), the baseline ILCR was 1.17×10−8 for coal-producing cities and 3.34×10−8 for oil-producing cities. The bioaccessible proportion of PAHs and their metabolites, released in the gastrointestinal tract lumen during digestion, resulted in a reduction of the ILCR by over 80% throughout both times. Polycyclic aromatic hydrocarbons (PAHs) and their halogenated derivatives are prevalent in the atmosphere, especially within aerosolized particles smaller than PM2. 5. The correlation between PM2.5 and PAHs in the ambient air was determined to be the source of Inhalation Lifetime Cancer Risk (ILCR) in Bangladesh and Japan. The observed median ILCR was 10^-3, exceeding the tolerable risk threshold of 10^-4. In Bangladesh, the levels of PAHs and xenobiotic PAHs (XPAHs), along with their corresponding ILCRs, were 1-2 times more than those in Japan.

Epithelial-mesenchymal transition (EMT) involves nuclear factor erythroid 2–related factor 2 (NRF2), Kirsten ras oncogene (KRAS), and hypoxia-inducible factor 1α, along with mesenchymal phenotypic markers such as N-cadherin, fibronectin, and vimentin. Conversely, exposure to B[a]P resulted in the down-regulation of E-cadherin gene expression, leading to injury in the human alveolar epithelial cell line A549, accompanied by a reduction in fibrotic mass and G-protein-coupled receptor class C group 5 member A (Gprc5a). The effects of 2-Benz[j]aceanthrylene were noted in human hepatocellular carcinoma cells through the activation of DNA damage signaling, as indicated by the sensitive markers pChk1 and γH2A histone family member X (γH2AX). The correlation between high molecular weight polycyclic

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aromatic hydrocarbons (PAHs), fluorene, and DNA methylation linked to cancer was demonstrated by Xu et al. (2024). Exposure to fluorene was associated with reduced DNA methylation of F2RL3 and AHRR, which are epigenetic biomarkers for lung cancer. The genotoxicity of 2-Benz[j]aceanthrylene was observed to induce DNA damage through DNA adduct formation. The correlation between polycyclic aromatic hydrocarbons (PAHs) and hepatic physiology was noted, with female subjects exhibiting elevated serum alanine transaminase (ALT) levels after exposure to 2-FLU (OR = 2.33, CI (95%) 1.15, 4.72). Urinary 2-FLU exposure (38.6%) was associated with elevated ALT levels (OR = 2.19, CI 95% 1.12, 4.27, p = 0.004). Exposure to 2-Fluorene was correlated with leukocytosis (3.56%), hypertriglyceridemia (6.99%), and hypercholesterolemia (1.70%) compared to normal levels. Impaired pulmonary redox status, inflammation, immunosuppression, and pulmonary malignancy were noted in mice exhibiting elevated expression of nuclear factor-κB (NFkB), cyclooxygenase-2 (COX-2), inducible nitric oxide synthase (iNOS), tumor necrosis factor α (TNF-α), proliferating cell nuclear antigen (PCNA), and matrix remodeling enzymes. Exposure to these factors activated the aryl hydrocarbon receptor (AhR), resulting in allergic airway inflammation.

Exposure to polycyclic aromatic hydrocarbons (PAHs), both outdoors and inside, has been linked to a heightened incidence of health issues, including endocrine malignancies, developmental toxicity, cardiovascular and pulmonary toxicity, diminished plasma insulin-like growth factor-1, and impaired immunological function, among others. The wide range of deleterious effects highlights the serious toxicological issues linked to PAHs, generating increased interest in comprehending and mitigating their impact. Exposure to PAHs is associated with respiratory issues such as allergies, asthma, chronic obstructive pulmonary disease (COPD), emphysema, and lung cancer. Exposure to PAHs mostly occurs via dietary intake and/or inhalation. For non-smokers, the primary source of exposure is charred food, whereas for smokers, tobacco use constitutes a major source of exposure. Polycyclic aromatic hydrocarbons (PAHs) are absorbed in the human body through pulmonary, gastric, or cutaneous routes, each involving distinct pathways and metabolic processes. These processes include the formation of DNA adducts, DNA lesions, alterations in cell cycle regulation, activation of the AhR receptor resulting in toxicity, genotoxicity mediated by reactive oxygen species (ROS) and oxidative stress, modulation of protein expression, and disruption of lipid

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metabolism. The oxidation-conjugation of PAHs in phases I and II activates phase I (cytochrome P450 monooxygenases—CYP1A1/2 and 1B1) and phase II metabolic enzymes (aldo-keto reductases, epoxide hydrolases, glutathione S-transferases, NADP quinone oxidoreductases, and uridine 5'-diphospho-glucuronosyltransferase) through both AhR-dependent and AhR-independent mechanisms. The metabolism of PAHs through the AKR, CYP peroxidase, and CYP1A1/1B1/EH pathways generates reactive metabolites, including carcinogenic diol-epoxides, radical cations, and o-quinones, which lead to DNA adduct formation, resulting in mutations, altered gene expression, and oncogenesis. The mutation in the xenobiotic metabolic and p53 genes correlates with heightened vulnerability to lung cancer. Intratracheal exposure markedly influences hepatic fatty acid metabolism and modifies levels of glycerolipids, glycerophospholipids, triacylglycerol, phosphatidylinositol, and phosphatidylcholine, while reducing concentrations of lysophosphatidylcholine, phosphatidylethanolamine, lysophosphatidylethanolamine, free fatty acids, and eicosanoids.

CONCLUSIONS

Polycyclic aromatic hydrocarbons (PAHs) have been categorized according to their human toxicity, predominantly linked to inhalation, ingestion, and skin exposure. Recent scientific literature indicates the increasing impact of exposure to polycyclic aromatic hydrocarbons on respiratory health. The presence of PAHs adsorbed to particulate matter, particularly PM2.5, can elevate health hazards above those posed by PAHs alone. This phenomenon can be ascribed to the size-specific penetration efficacy of PM2.5, which is facilitated by increased inhalation and deposition, prolonged residence time, and synergistic health consequences, as it acts as a transport medium for PAHs, allowing it to reach alveolar cells more effectively than PM larger than 5 μm. This review concludes that exposure to PAHs induces health issues through AhR-mediated interconnected signaling pathways, leading to heightened oxidative stress, immune system impairment, asthma/COPD, and cancer. The mitigation of ambient air pollution can substantially enhance worldwide human health. This review established a thorough and rigorous methodology for assessing the respiratory toxicity of PAHs and their physiologically active metabolites. The results of this analysis may provide

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a foundational framework for the design of future research investigations and the formulation of policy implications about the toxicity of PAHs. Further research is required to elucidate, substantiate, and reinforce the significance of PAHs, especially in the context of multiple-pollutant exposure or in multidisciplinary investigations encompassing PAHs' respiratory toxicity. Engaging in these research activities can enhance comprehension of the role of PAHs in respiratory toxicity and guide evidence-based solutions for alleviating the related health hazards.

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