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Health Impacts of Air Pollutants from the Coal-Fired Power Sector

Runsheng Zheng

Faculty of Forestry and Environment, University Putra Malaysia, Selangor, Malaysia
213850@student.upm.edu.my

Abstract. Coal-fired power plants remain the primary baseload electricity source in many countries, yet the substantial particulate matter generated during combustion poses a long-term threat to human health. This paper synthesizes and analyzes recent advances in research on air pollution from the coal-fired power sector and its health impacts, with in-depth discussions centered on emission characteristics, exposure risks, biological mechanisms, and mitigation strategies. It first identifies the major pollutant types emitted from coal-fired plants and describes their spatiotemporal distribution in representative regions (e.g., Beijing–Tianjin–Hebei), integrating monitoring data and model analyses to reveal the influence of regional transport and seasonal meteorological conditions on pollution levels. From the perspective of occupational exposure, the paper highlights the long-term accumulation risks of dust, toxic organics, and heavy metals faced by frontline power-plant workers, drawing on on-site measurements and biomarker data to demonstrate their heightened health vulnerability. On the mechanistic level, it explains how air pollutants can trigger respiratory, cardiovascular, and neurological disorders through pathways involving inflammatory responses, oxidative stress, endothelial injury, and genetic and epigenetic alterations. The governance section summarizes the achievements of China's ultra-low-emission retrofits, identifies shortcomings in occupational protection and health-oriented standard systems, and proposes future directions, including enhanced exposure monitoring, long-term cohort studies, and interdisciplinary research. This study aims to provide scientific evidence for health risk assessment and policy optimization.

Keywords: Coal-Fired Power Industry, Occupational Exposure, Health Impacts, Governance Policies

1. Introduction

Although global and Chinese energy systems have gradually transitioned toward low-carbon development over recent decades, coal-fired power generation still accounts for a substantial share in many regions, and its impacts on air quality and public health remain significant. The World Health Organization (WHO), in its 2021 update of the Global Air Quality Guidelines, emphasized that fine particulate matter ($PM_{2.5}$) and various gaseous pollutants can exert significant adverse health effects even at concentrations far below the standards currently adopted in many countries, indicating that policy targets should be set based on health protection [1]. This study investigates the

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emission characteristics of major air pollutants from the coal-fired power sector and their spatiotemporal distributions in representative regions, while also analyzing exposure levels and associated health impacts among both the general population and power-plant workers, supported by quantitative data and regional case studies. Building on this foundation, the study further explores the biological mechanisms through which pollutants induce respiratory, cardiovascular, and systemic diseases, and evaluates the practical effectiveness and existing limitations of current control measures in China, to provide scientific evidence to improve pollution-prevention strategies, strengthen occupational health protection, and support the development of more health-oriented energy and environmental policies.

2. Characteristics and representative data of emissions from coal-fired power plants

2.1. Major emission types and their physicochemical forms

Coal combustion can generate inhalable particulates (PM_{10} and $PM_{2.5}$), gaseous SO_2, NO_x, carbon monoxide (CO), trace metals (Hg, Pb, As, etc.), and unburned organic compounds (including PAHs). Secondary atmospheric reactions can convert SO_2 and NO_x into sulfates and nitrates, significantly increasing secondary $PM_{2.5}$ loading. Particulates can be categorized into filterable and condensable phases, with the latter contributing non-negligibly to PM mass during cooling [1].

2.2. Constraining evidence and specific values for the Beijing–Tianjin–Hebei (BTH) region

Observational and emission-characterization studies for the Beijing–Tianjin–Hebei region indicate that, during 2013–2018, the annual mean $PM_{2.5}$ concentrations at several monitoring sites ranged from 59 to 77 $\mu g \cdot m^{-3}$ (based on 12 sites). Moreover, the $PM_{2.5}$ composition (e.g., elemental and organic carbon) varied markedly among sites, suggesting spatial heterogeneity in the contributions of industrial point sources (including coal-fired power plants), traffic, and fugitive dust to total exposure [2,3]. Attribution studies based on regional models indicate that reductions in local and regional emissions were among the primary drivers of the marked decline in $PM_{2.5}$ in the capital during 2013–2017, with local controls contributing the most; measures such as coal-fired boiler control and industrial restructuring played major roles. Such studies provide a quantitative basis for assessing the air quality and health benefits of emission reductions from coal-fired power plants [4].

2.3. Power-plant point sources and occupational exposure data (China sample)

As one of the major potential high-pollution emission sources, coal-fired power plants exert significant pressure on regional air quality, while high concentrations of dust and harmful particulates within the workplace pose a persistent and tangible threat to workers' health. According to CDC Weekly, based on occupational and environmental dust monitoring in coal-fired power plants of various scales from 2017 to 2019, silica dust and coal dust are the primary occupational hazards, and both personal exposure and ambient levels decline as generating-unit capacity increases, since larger units typically feature more advanced dust-removal and centralized-control systems. Comprehensive assessments indicate that power-plant workers face a moderate level of dust-related risk, although personnel in coal transportation, coal pulverization, and ash-handling positions experience markedly higher dust exposure. The report also provides distributions of monitoring data for workplace conditions and personal exposure in power plants [5].

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2.4. Special pollutants in power plants: occupational accumulation evidence for PAHs and heavy metals

Polycyclic aromatic hydrocarbons (PAHs) and heavy metals are two of the most representative persistent toxic pollutants emitted by coal-fired power plants. PAHs are lipophilic organic pollutants with multi-ring conjugated structures, typically originating from incomplete coal combustion; their metabolites can form adducts with DNA, inducing gene mutations, and are recognized as carcinogenic and mutagenic agents. Heavy metals (such as mercury, lead, and arsenic) pose long-term toxic risks due to their non-degradability and high bioaccumulation, causing chronic health effects by disrupting enzyme activity, inducing oxidative stress, and damaging the nervous and hematopoietic systems. Accordingly, workers in coal-fired power plants are particularly highly exposed and sensitive to these pollutants [6,7]. Biomonitoring studies of Chinese coal-fired power plant workers show sustained accumulation of parent PAHs and their halogenated derivatives (HPAHs) in serum, with pronounced differences by sex and job category. Specifically, male workers generally exhibit higher serum PAH levels than female workers, and exposure is particularly elevated in high-risk positions such as boiler operation. These findings suggest that long-term occupational exposure in coal-fired power plants leads to a systemic increase in workers' PAH body burden, which may be associated with elevated risks of cancers (e.g., lung cancer) and other chronic health effects such as cardiovascular disease and respiratory dysfunction. The study further provides detailed concentration ranges of serum PAHs and corresponding risk-assessment indicators (such as carcinogenic risk indices), offering direct scientific evidence for occupational exposure risk evaluation [6,7].

3. Biological pathogenic mechanisms of pollutants in the human body

3.1. Particulate matter ($PM_{2.5}$)

Fine particulate matter can penetrate the alveoli and activate local macrophages and epithelial cells, releasing pro-inflammatory cytokines (IL-6, TNF-α, IL-1β) and chemokines, thereby initiating localized inflammation and increasing alveolar permeability. Smaller particles and their adsorbed toxic components (heavy metals, PAHs) can traverse the alveolar epithelium into the circulation, elevating inflammatory markers, inducing oxidative stress (increased RONS), and promoting LDL oxidation, which in turn damages vascular endothelium, facilitates atherosclerotic plaque formation and destabilization, and increases thrombogenicity (platelet activation and upregulation of coagulation factors). The American Heart Association and multiple reviews have summarized these pathways as the primary biological mechanisms underlying PM-induced cardiovascular diseases, including acute myocardial infarction, stroke, and heart failure.

3.2. Gaseous pollutants (SO_2/NO_x/O_3): direct oxidation and synergistic formation of secondary particles

As two of the most important gaseous pollutants emitted by the coal-fired power sector, sulfur dioxide (SO_2) and nitrogen oxides (NO_x) play central roles in atmospheric chemistry and health-hazard pathways, serving as key precursors that affect regional air quality and public health.SO_2 and NO_x can directly irritate the respiratory mucosa, causing bronchospasm and worsening asthma; NO_x reacts with VOCs under sunlight to form ozone (O_3), a potent oxidant capable of damaging epithelial cells and amplifying inflammatory pathways. More importantly, as precursors of

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secondary particles, SO_2 and NO_x form sulfates and nitrates in the atmosphere, substantially increasing $PM_{2.5}$ mass and amplifying PM-mediated systemic effects [1].

3.3. PAHs, heavy metals, and genetic/epigenetic damage

PAHs are metabolized by the P450 enzyme system into reactive metabolites that form DNA adducts, causing DNA damage, mutation accumulation, and increased cancer risk. Heavy metals such as mercury and lead exhibit neurotoxicity—affecting neural transmission and synaptic plasticity—and can disrupt cellular function by displacing metalloenzyme cofactors or inducing oxidative injury. Evidence indicates that long-term occupational accumulation is associated with chromosomal damage, biomarker alterations, and increased incidence of certain cancers [6,7].

After entering the human body, PAHs are primarily metabolized by the cytochrome P450 enzyme system into electrophilic active metabolites, such as BPDE (benzo[a]pyrene diol epoxide), which can form stable DNA adducts, causing base mismatches, strand breaks, and replication errors, thereby inducing mutation accumulation and increasing cellular carcinogenic risk. Additionally, PAHs can activate the aryl hydrocarbon receptor (AhR) signaling pathway, leading to cell-cycle disruption and immunosuppression. Halogenated PAHs, due to their higher lipophilicity and persistence, accumulate more readily in adipose tissue and blood, posing potential disruptive effects on the nervous and endocrine systems.

Heavy metal pollutants such as mercury, lead, and arsenic also exhibit high biological toxicity. Mercury disrupts thiol-protein function and glutathione metabolism, causing mitochondrial dysfunction, excessive ROS generation, and neuronal injury. Lead competes with calcium to enter neurons, impairing synaptic transmission, affecting learning and memory, and suppressing hematopoiesis. Arsenic alters DNA repair enzyme activity and promotes methylation dysregulation, inducing genomic instability. These mechanisms indicate that long-term occupational exposure can cause chromosomal abnormalities, altered DNA methylation patterns, miRNA dysregulation, and epigenetic changes, ultimately manifesting as cancer, neurodegenerative disorders, cardiovascular injury, and immune impairment. Biomonitoring studies further confirm significant associations between PAH/heavy-metal accumulation in power-plant workers and abnormalities in multiple biomarkers, reinforcing their chronic health risks.

3.4. Autonomic nervous and acute triggering mechanisms

Short-term high-level exposure can stimulate the sympathetic and parasympathetic systems, inducing arrhythmias or blood-pressure fluctuations (e.g., $PM_{2.5}$-related dysrhythmia and acute coronary events), providing a physiological basis for pollution peaks triggering acute cardiovascular events. Beyond chronic cumulative effects, short-term high-concentration air-pollution exposure can rapidly trigger acute cardiovascular events via neuroregulatory pathways.$PM_{2.5}$ and gaseous pollutants can stimulate airway receptors and pulmonary vagal nerve endings, causing autonomic imbalance—sympathetic activation and parasympathetic suppression—leading to reduced heart-rate variability and elevated arrhythmia risk. Studies show that during pollution peaks, ECG monitoring reveals increased atrial or ventricular ectopy, higher arrhythmia incidence, and QT-interval variability, indicating impaired electrophysiological stability of the myocardium. Meanwhile, air pollution can trigger acute systemic inflammation and coagulation activation—elevated pro-inflammatory cytokines, enhanced platelet reactivity, and increased fibrinogen—leading to higher blood viscosity and elevated risk of acute thrombosis [6,7].

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4. Empirical research and case studies: quantitative evidence of exposure–effect relationships

4.1. Regional modeling and health burden estimation (evidence from China's pollution-control achievements)

Model-based studies covering multiple regions in China indicate that the clean air actions implemented after 2013 significantly reduced $PM_{2.5}$ levels, with regional emission reductions playing a dominant role in the improvement of Beijing's $PM_{2.5}$ (local emission cuts contributing ~65%), thereby lowering the disease burden associated with PM-related pollution [3,4]. Recent studies have also evaluated the short- and medium-term contributions of control strategies targeting coal-fired emissions—including ultra-low-emission retrofits—to air-quality improvement and health benefits, showing clear positive effects on reducing regional $PM_{2.5}$ levels and associated premature mortality [10].

4.2. Exposure–biomarker relationships and health outcomes among power plant workers

Occupational exposure monitoring and biomarker studies conducted among power plant workers in China—for example, measurements of serum PAHs/HPAHs—provide direct evidence of internal dose, indicating that workers in specific positions bear substantially higher internal chemical loads than control populations. In addition, occupational dust-monitoring reports show that the concentrations of inhalable dust in certain job categories can reach levels corresponding to "moderate" or higher occupational hazard ratings (based on data from the China CDC Weekly Reports). These quantitative findings support the implementation of stricter personal-protection measures and long-term follow-up policies for power plant workers [6,7].

As the population most directly bearing health risks in the coal-fired power sector, evidence on internal exposure and health effects among power plant workers is critical for understanding industry-specific hazards. Recent on-site monitoring and biomarker studies in China have provided a quantitative basis for assessing these risks. In a cohort study of workers from a coal-fired power plant, serum measurements of polycyclic aromatic hydrocarbons (PAHs) and their halogenated derivatives (HPAHs) showed that high-exposure workers—such as those in boiler operation, pulverized coal transport, and ash handling—had median ΣPAH concentrations of 2.1–3.6 ng/mL, markedly higher than the 0.7–1.0 ng/mL observed in office-based control groups; meanwhile, internal levels of HPAHs were approximately 2–3 times higher in high-risk workers. Concurrently, statistically significant elevations were observed in oxidative damage biomarkers (8-OHdG) and inflammatory markers (such as IL-6), indicating trends of DNA damage and chronic inflammatory activation [6,7].

Occupational dust monitoring conducted by the China CDC for coal-fired power plants during 2017–2019 showed that concentrations of inhalable dust in coal storage areas, coal-milling workshops, and fly-ash handling zones ranged from approximately 2.0 to 6.5 mg/m^3, markedly higher than those measured in centralized control rooms (0.05–0.2 mg/m^3). Some job positions exceeded the threshold for "moderate risk" in occupational hazard classification, and although exposure scores were generally lower in medium- to large-scale units, exceedance points were still present.

These quantitative findings indicate a clear gradient of occupational exposure and a discernible trend of internal pollutant accumulation among workers in coal-fired power plants, thereby substantiating the causal pathway linking long-term operational exposure to increased internal dose and biological damage. Accordingly, strengthening the management of personal protective

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equipment, implementing job rotation and regular biomonitoring, and establishing long-term health follow-up cohorts for workers constitute core strategies for reducing occupational health risks and represent key priorities for health governance within the power sector [6,7].

4.3. Performance of representative units and the effectiveness of ultra-low-emission retrofits

Comparative studies of ultra-low-emission (ULE) units and non-ULE units show that, following ULE retrofits, the emission intensities of PM, SO_2, and NO_x are substantially reduced, with removal efficiencies of desulfurization, dust control, and denitrification systems each exceeding 90%, alongside improved co-removal of trace metals [3,8]. However, because regional air pollution is still jointly influenced by multiple sources, point-source control alone cannot immediately eliminate severe pollution episodes, necessitating coordinated regional control and adjustments to the energy structure [4]. Monitoring data from a 600 MW unit before and after ULE retrofitting indicate that SO_2 concentrations decreased from 120–150 mg/m³ to <20 mg/m³, NO_x from 200–260 mg/m³ to <50 mg/m³, and particulate emissions from 20–35 mg/m³ to <5 mg/m³; additional studies report that mercury emission factors in fly ash declined by approximately 70–85% following ULE retrofits [10]. However, during winter $PM_{2.5}$ episodes in the Beijing–Tianjin–Hebei region, about 35–45% of pollution still originates from regional transport and multi-source contributions, indicating that ULE must be implemented in coordination with cross-regional control measures [4].

5. Evaluation and critical analysis of policies and governance

China's recent ultra-low-emission (ULE) retrofits and large-scale industrial emission-reduction initiatives have substantially lowered point-source emission intensities and align with rapid $PM_{2.5}$ improvements observed in several cities, as well as with notable estimated health benefits—such as reductions in premature deaths and hospitalizations [1]. Since the launch of the Air Pollution Prevention and Control Action Plan (APPCAP) in 2013, multiple peer-reviewed studies using observational and modeling evidence have quantified the policy's effectiveness, demonstrating that point-source emission reductions and coordinated regional control have significantly decreased $PM_{2.5}$ concentrations and generated considerable health and economic gains, however, further strengthening is required for ozone and certain gaseous pollutants [3,4].

Measured improvements in Beijing and the broader Beijing–Tianjin–Hebei region indicate that the city's annual mean $PM_{2.5}$ concentration decreased from approximately 89.5 μg/m³ in 2013 to 58 μg/m³ in 2017, with model-based source attribution analyses identifying emission-reduction measures—including coal consumption control, industrial emission cuts, and traffic restrictions—as the primary drivers of this improvement [3].

Targeted governance of coal-fired power plants has also been quantitatively assessed: for instance, pollutant-control processes and ULE retrofits (including desulfurization, denitrification, and particulate control) have been linked to substantial reductions in mercury emissions between 2011 and 2015, with corresponding decreases in IQ loss and premature mortality, demonstrating the direct contribution of power-plant emission reductions to specific pollutants and associated health outcomes [9].

Although ultra-low-emission retrofits have substantially reduced unit-level emission intensities in the coal power sector, repeated severe pollution episodes continue to arise from the combined influence of regional transport and unfavorable meteorological conditions, making point-source control alone insufficient for resolving the problem. Studies in the Beijing–Tianjin–Hebei region

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show that external transport can contribute 40–60% of $PM_{2.5}$, underscoring the necessity of cross-regional coordination and early-warning mechanisms [4].

At the same time, structural gaps remain in occupational-exposure governance—frontline workers continue to face moderate-risk levels of dust and toxic pollutants, with exposures in coal handling and maintenance positions markedly higher than those in managerial roles [5]. Furthermore, current standards still diverge from the WHO 2021 Air Quality Guidelines, suggesting that future regulatory frameworks should gradually align with health-based benchmarks to better protect high-risk populations [1]. Achieving a transition from "compliance-oriented emission control" to "health-oriented emission reduction" will be crucial for advancing air-risk management.

6. Research outlook and recommendations

In the future, an integrated high–spatiotemporal–resolution exposure monitoring system encompassing power plants, surrounding communities, and occupational settings should be established, combining fixed monitoring stations, online emission monitoring, and wearable personal-exposure devices to enable exposure source apportionment and refined health-risk assessments for sensitive populations [9].

At the same time, long-term occupational cohorts should be established among power plant workers, with routine biological sample collection to obtain PAH, metal, and epigenetic indicators, thereby strengthening analyses of internal dose–response relationships and reinforcing the role of biomonitoring in occupational hazard research [10]. Mechanistic studies should incorporate pollutant metabolism, DNA adduct formation, and methylation-pattern alterations into exposure-to-phenotype pathways to support causal inference [7].

In addition, health–economic analyses should be incorporated to compare the cost–health benefits of coal-based pathways (including ULE and CCUS) with renewable-energy transition scenarios, thereby providing scientific evidence for energy-policy decisions [11]. This multi-level strategy will help shift policy orientation from mere emission compliance toward maximizing health protection and broader societal benefits.

7. Conclusion

This paper underscores that the coal-fired power sector remains a major source affecting regional air quality and public health, posing ongoing exposure risks, particularly for frontline power plant workers and sensitive populations living nearby. Although ultra-low-emission technologies and policy interventions have led to substantial improvements, residual emissions, regional transport effects, and the complex interactions among multiple pollutants indicate that health risks have not been fully eliminated. To fundamentally reduce the health burden associated with coal-fired power generation, it is essential not only to strengthen regional joint prevention and control and optimize industrial structures, but also to enhance occupational health protection systems, establish health-oriented emission and air-quality standards, and accelerate the transition of the energy structure toward cleaner and more sustainable pathways. Future research should further accumulate biological evidence on long-term exposure and health effects, develop long-term health follow-up cohorts for power plant workers, and integrate health–economic evaluation into energy and environmental policy-making, thereby providing a stronger scientific foundation for achieving the coordinated goals of "reducing emissions, protecting health, and promoting transition.

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