

Characteristics and Risks of Composite Pollutants Formed by Particulate Matter and Polycyclic Aromatic Hydrocarbons During Combustion of C1 Bituminous Coal in Xuanwei Area

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Abstract: Since the 1980s, Xuanwei, Yunnan Province, China, has remained a global hotspot for lung cancer incidence and mortality. Extensive multi-disciplinary research—spanning epidemiology, atmospheric science, and molecular biology—suggests that this health crisis is not driven by a solitary variable but is the consequence of synergistic interactions among multiple high-risk factors. While the domestic use of bituminous coal and resultant indoor air pollution are identified as primary drivers, the specific contributions of stove typologies and behavioral habits warrant further investigation. This paper systematically analyzes the etiology of high lung cancer rates in Xuanwei, focusing on the characterization and health risks of fine particulate matter (PM_{2.5}) and polycyclic aromatic hydrocarbons (PAHs) generated during coal combustion.

Keywords: Nano-silica; Polycyclic Aromatic Hydrocarbons; Fine Particulate Matter; Health Risks.

1. Introduction

Xuanwei, a county-level city in Qujing, Yunnan Province, is characterized by its abundant coal reserves and the prevalence of small-scale mining despite its underdeveloped heavy industry. The local populace, primarily engaged in agriculture, relies heavily on unprocessed lignite sourced from nearby kilns for domestic energy. The widespread use of traditional open fire stoves, coupled with inadequate ventilation and exhaust systems, results in severe indoor air quality degradation [1,2]. The particulate matter emitted during combustion is a complex matrix comprising silica, polycyclic aromatic hydrocarbons (PAHs), transition metals, and heavy metals. Notably, PAHs adsorbed onto the surface of nano-silica particles can penetrate deep into the pulmonary system due to their ultra-fine scale, triggering severe health risks. Socio-behavioral patterns further exacerbate this crisis: while local men frequently migrate for work, women typically spend approximately 17 hours per day indoors. This prolonged exposure explains the disproportionately high lung cancer morbidity and mortality rates among the female population. This paper reviews the current research landscape of indoor air pollution to elucidate the underlying mechanisms driving the lung cancer epidemic in Xuanwei.

2. Regional Overview of The Late Permian in Xuanwei Area

2.1. Impact of Coal Types and Open Stoves on Particulate Emissions

Previous research has shown that the coal seam in Xuanwei area, Yunnan belongs to the Late Permian coal seam, and the coal species is classified as bituminous coal. Nearby residents mostly use locally produced unprocessed bituminous coal as

fuel for life and heating. Research by Yang Kaiyun and others found that the content of particulate matter suspended in the air produced by burning lignite in Xuanwei area is much higher than that produced by anthracite coal. The content of organic matter in the particulate matter is 43%, and most of these substances are PAHs. The sex ratio (male: female) of lung cancer mortality varies greatly among regions. It is 13.5:1 in the Netherlands, 1.8:1 in the United States, 2.07:1 in China, 1.09:1 in Xuanwei Region of Yunnan, and 0.87:1 in Laibin Town of Xuanwei [3]. The peak age of lung cancer death among people burning bituminous coal is advanced, and the lung cancer death rate among people burning bituminous coal is 1.7 times that of people who burn non-bituminous coal. In rural areas of Xuanwei, "open fire ponds" are often used for heating and cooking. "Open fire ponds" refer to the use of fire ponds directly at home and lack ventilation. Research by NBurce et al. found that stove/fuel type is the most important determinant of kitchen pollutants [4]. Secondly, the maintenance of stoves can reduce the concentration of pollutants to a certain extent. The mineral components in Xuanwei area are mainly clay minerals and quartz, followed by calcite, pyrite and siderite [5]. The special geographical structure of the Late Permian determines that the element in the local lignite is mainly silicon. The silicon element mainly exists in the form of oolite in the Xuanwei area, which is a Si+Fe complex. The ratio of silicon to aluminum in Xuanwei coal samples ranges from 2.24 to 8.16, with an average value of 4.17, which is much higher than the average value in my country's coal (1.14) [6]. The content of some transition metal elements is also higher than in other regions. Some large quartz grains are formed by attaching small quartz grains together. Xuanwei coal is mainly composed of bituminous coal of the vitrinite group with high medium volatile content. The main minerals in the coal are quartz, oolitic, kaolinite and calcite; the degree of

crystallization of minerals varies among different coal samples. The silica content in locally produced lignite is three times the national average. A large number of studies have shown that the distribution of lung cancer incidence in Xuanwei area is consistent with the distribution of coal seams in the Late Permian region.

3. The Main Components and Characteristics of Indoor Pollutants in Xuanwei Area

3.1. Characteristics of Indoor Air Pollution

The Characterization and Health Risks of Indoor Pollutants: Household air pollutants (HAPs) primarily exist in gaseous and particulate phases [7]. Research by Guo et al. has established a significant positive correlation between the degree of HAP exposure and lung cancer incidence [8]. Specific measurements in Xuanwei households revealed that the mean diurnal concentration of PM 2.5 reached 166 ug/m³, substantially exceeding the national air quality threshold of 75ug/m³. Furthermore, the biological toxicity of PM 10 in high-incidence areas was significantly higher than in low-incidence regions ($P < 0.05$) [9], a finding consistent with the observations of KHLui et al [10]. Regarding risk assessment, Q Liu et al. reported that poor indoor ventilation elevates the odds ratio (OR) for lung cancer to 2.1 (95% CI: 1.2–3.8) for males and 3.6 (95% CI: 1.4–9.3) for females [11]. While Behera D et al. suggested that blended fuel usage is associated with a mitigated risk (OR = 3.04) [12], the monolithic fuel structure in rural Xuanwei facilitates persistent high emissions, driving cumulative carcinogenic risk [13]. Toxicological characterization by Zhang Rongchi et al. demonstrated that fine particles account for 87.4% of the total toxic equivalence (TEQ) of PAHs in inhalable matter, dwarfing the contributions of coarse (12%) and ultra-fine (0.4%) fractions [14]. Similarly, SHCho et al. found that carbon-black-containing ultra-fine particles exhibit greater toxicity than their larger counterparts [15]. Source apportionment by DWu et al. via GC-MS identified 18 PAHs, confirming that coal combustion contributes 42%–84% and 75%–82% of the PAH mass in PM 2.5 and PM 10, respectively [16]. Compared to wood or anthracite emissions, the smoky coal utilized in Xuanwei releases high concentrations of sub-micron particles enriched with mutagenic organic matter—specifically aromatic and polar fractions—leading to severe indoor pollution and elevated health risks.

3.2. Characteristics and Risks of Fine Particulate Matter Emissions

Silica, or quartz, is widely found in nature. However, it rarely occurs in nature in its simple form. Silica is usually not considered to be a carcinogen. However, occupations characterized by high exposure to crystalline silica have excessive lung cancer mortality. Tian [17] found that there are a large amount of kaolin (mainly composed of $\text{Al}_2\text{O}_3 \cdot 2\text{SiO}_2 \cdot 2\text{H}_2\text{O}$) in C1 coal seam, which is rich in nano-sized silica crystals smaller than 100nm. But in the form of complex silicate or silica, widely found in rocks, gravel and dust. Previous studies have shown that [18], most of the silicon dioxide in lignite in Xuanwei area is micron, (the particle size is mostly distributed between 1 ~ 2 μm). GJLi [19] measured the content of silicon dioxide in C1 bituminous

coal and its combustion products in the high incidence area of lung cancer in Xuanwei City, Yunnan Province. The results showed that the diameter of silicon dioxide particles in the bottom ash was mostly 120~500nm. Different shapes, including iron, aluminum, calcium, potassium and other elements, silica particles in soot are mainly nano-sized, diameter between 37~80nm, different shapes, some are fibrous, the surface is not smooth, containing iron, potassium, calcium, aluminum, sulfur. WLin et al used crystalline silica as a positive control in cultured human bronchoalveolar carcinoma-derived cells and studied the cytotoxicity of 15nm and 46nm silica nanoparticles. It was found that ROS in alveolar cells exposed to SiO_2 nanoparticles significantly increased, glutathione content decreased, while the increase in malondialdehyde and lactate dehydrogenase release indicated lipid peroxidation and cell membrane damage [20]. The study of FanJS and ZhouL found that the composition of indoor pollutants in Xuanwei lung cancer high incidence area was spherical particles > mineral particles > dust aggregates > unknown particles, and in low incidence areas from high to low was unknown particles > dust aggregates > mineral particles > spherical particles. More than 70% of indoor respirable particles contained O and Si elements. In the high incidence area, the volume of silica in particulate matter is larger [21 ~ (th), 22 ~ (th), 23]. SLu shows that fine particles are the main components of Xuanwei atmospheric particles. PIXE results show that crustal elements (Ca, Ti, Si, Fe) are mainly distributed in coarse particles, while trace metals (Cr, Mn, Ni, Cu, Zn, Pb) in fine particles are dominant. The correlation coefficients (R^2) between free radical intensity and fluorescence intensity produced by size decomposition particles are 0.535 and 0.507, respectively. The ambient air particles in Xuanwei atmosphere have the ability to produce free radicals, and fine and ultra-fine particles may cause harm to local residents [24]. Some metal ions can react with silanols on the surface of silica, changing the ability of the surface to produce free radicals, thus further affecting the toxicity of silica [25]. The lungs are the target organs of silica. Some experiments have shown that nanometer silica has greater health risks than micron silica. Nano-sized silica may be produced by cracking lignite during combustion. Experiments in mice have shown that the smaller the particulate matter, the greater the damage to the lungs [26]. The reason may be that small particulate matter will be inhaled and stay deep in the lungs. They are not easily excreted from the body, and free radicals carried on the surface of silica particles can cause irreversible damage to lung cells.

Li Guangjian et al. [27] performed the characterization and isolation of nano-scale silica generated from the combustion of Xuanwei bituminous coal. Their findings indicate that while silica in Hehe coal predominantly exists at the micron level, the silica produced by Xuanwei coal combustion is primarily nano-scale (ranging from 150nm to 200nm), a concentration significantly higher than that found in coal from other regions ($P < 0.05$). The aerodynamic diameter of these fine particles is a critical determinant of their deposition patterns within the human respiratory tract upon inhalation [28]. While the IARC has classified crystalline silica as a Group 1 carcinogen, nano-scale silica exhibits more distinct physicochemical properties than its bulk crystalline counterparts. Owing to its diminutive particle size, substantial specific surface area, and the prevalence of unsaturated bonds, nano-silica can alter cellular distribution and catalyze the generation of reactive oxygen species (ROS). Consequently,

nano-sized silica poses a more formidable health risk compared to micro-sized silica.

MouseChronic Exposure and Carcinogenic Mechanisms of Nano-silica: Murine models indicate that acute, high-dose exposure to nano-silica fails to induce pulmonary oncogenesis; consequently, the lung cancer epidemic in Xuanwei is likely triggered by protracted, low-dose environmental exposure [29]. Under such chronic conditions, the inflammatory response serves as the primary manifestation of silica-induced lung injury. While localized inflammation is typically non-tumorigenic, persistent inflammatory milieu can facilitate the destruction of adjacent healthy tissues, leading to genetic mutations, cell death, or the onset of comorbid pathologies. Zhao Guangqiang et al. [30] demonstrated that nano-silica predominantly accumulates within the cytoplasm of bronchial epithelial cells, inflicting damage on organelles such as the endoplasmic reticulum and mitochondria. Similarly, YZhou et al. [32], employing electron microscopy and centrifugal techniques, correlated the utilization of "C1" smoky coal and cumulative nano-quartz exposure with elevated lung cancer risk. As a unique constituent of coal-combustion-derived fine particulate matter, the health risks associated with nano-silica have become a focal point of contemporary scientific inquiry.

3.3. Risk Characteristics of Polycyclic Aromatic Hydrocarbons Produced by Coal Burning

Distribution Characteristics and Carcinogenic Mechanisms of PAHs: Polycyclic aromatic hydrocarbons (PAHs) are semi-volatile, toxic compounds composed of two or more fused benzene rings in linear, angular, or clustered configurations. Characterized by high chemical stability and long-range transport potential, PAHs are classified as persistent organic pollutants (POPs) [33]. In Xuanwei, the combustion of smoky coal releases massive quantities of PAHs. Benzo[a]pyrene (BaP), recognized by IARC as a Group 1 carcinogen, serves as the primary indicator for environmental health risk, with its Toxic Equivalence Factor (TEF) standardized at 1.0 [34]. Reports indicate that BaP levels in Xuanwei's high-incidence areas reach 0.133 $\mu\text{g}/\text{m}^3$, exceeding safety limits by 132-fold [35]. While dietary intake of PAHs exists, it is significantly lower than inhalation exposure from indoor air [36]. Residents chronically exposed to indoor smoky coal combustion exhibit an odds ratio (OR) for lung cancer of 7.7 [37, 38], with females inhaling higher annual concentrations than males due to domestic behavioral patterns [39]. Indoor BaP concentrations consistently exceed outdoor levels and national standards [43-45], though the installation of chimneys has been shown to mitigate these risks [46, 47]. Metabolically, PAHs undergo activation via cytochrome P450 enzymes and epoxide hydrolase to form anti-benzo[a]pyrene-7,8-diol-9,10-epoxide (BPDE) [48]. This reactive intermediate binds to DNA to form *BPDE-N²-dG* adducts, which predominantly accumulate in bronchial epithelial cells [49]. The concentration of these adducts correlates positively with lung cancer risk [50], and the mutation spectrum induced by BPDE mirrors the *p53* gene mutations observed in human lung tumors [51-53]. Furthermore, PAHs exhibit a high affinity for mitochondria, causing mitochondrial DNA (mtDNA) damage and frequent *p53* mutations [61]. Elevated levels of PAHs-DNA adducts in the lung tissues of Xuanwei women [60] and higher urinary PAH metabolites [56] further substantiate that the continuous

emission of PAHs into poorly ventilated indoor environments is a primary driver of the local lung cancer epidemic.

4. Association between 3C1 Bituminous Coal and Increased Polycyclic Aromatic Hydrocarbon Emissions.

Coal Formation Pathways of PAHs during Combustion and the Catalytic Role of Metals: Coal is a complex macromolecular composite characterized by aromatic clusters interconnected via aliphatic chains or structural oxygen bridges [62]. The generation of polycyclic aromatic hydrocarbons (PAHs) during coal combustion primarily follows three mechanistic pathways. First, the volatilization of raw coal: Lawrence et al. [63] observed that low-molecular-weight PAHs bound by weak hydrogen bonds are readily released even at ambient or moderate temperatures. Second, high-temperature pyrolytic synthesis: large aromatic compounds decompose into highly reactive radicals, which subsequently undergo radical recombination to form fused benzene rings [64, 65, 66], aligning with the model proposed by Bitter et al. [67, 68]. Third, the thermal cleavage of PAHs previously tethered by covalent chemical bonds. Research by Li Xiaodong et al. [69] demonstrated that PAHs yields from bituminous coal (6.231 $\mu\text{g}/\text{g}$) significantly exceed those from anthracite (2.9057 $\mu\text{g}/\text{g}$) and lean coal (0.2923 $\mu\text{g}/\text{g}$), consistently with findings by Yanwang et al. [70]. Specifically, in Xuanwei, the bituminous coal sourced from the Late Permian C1 coal seam exhibits an exceptionally high iron (Fe) content, averaging 3.5% [71]. The direct combustion of this unprocessed coal significantly amplifies PAH emissions. During combustion, active sites on the surfaces of metals or metal oxides act as catalytic centers. Metal ions, through electron transfer (redox processes), interact with diverse radical species in the flame, modulating radical concentrations and facilitating the synthesis of complex PAHs. The domestic use of such coal is statistically associated with the local lung cancer epidemic. Open-fire stoves facilitate the discharge of immense quantities of fine particulate matter into the indoor environment; these particles serve as high-affinity carriers for PAHs, substantially increasing the cumulative daily exposure among local women.

5. Characteristics of Interactions Between Polycyclic Aromatic Hydrocarbons and Fine Particles

In the early years Interactions between Particulate Matter and PAHs and Their Composite Toxicity: Early domestic research suggests that smaller sediment particle sizes, characterized by larger specific surface areas, lead to significant variations in the adsorption capacity for polycyclic aromatic hydrocarbons (PAHs) [72]. Conversely, Weber et al. argued that these adsorptive differences stem from the divergent physicochemical properties of the particles rather than size alone [73]. Zhao Chengmei et al. [74] further demonstrated that PAH adsorption on soot scales positively with carbon content, while Kun Yang et al. confirmed the high adsorptive affinity of nanomaterials for PAHs [75]. Utilizing indoor benzo[a]pyrene (BaP) concentrations, silica levels, and lung cancer mortality data, Vermeulend et al. proposed a synergistic risk hypothesis involving the interaction between silica and PAH exposure [76]. Downward et al. [77] analyzed multiple pollutants across various fuels

and stove types, finding that bituminous coal generates significantly higher loads of fine particulate matter and PAHs than anthracite, reinforcing the theory of a multi-factorial etiology for local lung cancer.

Research by PCocco indicates that among silica-exposed populations, multiple risk factors interact in complex ways to modulate lung cancer risk [78]. Valavanidis et al. posited that redox-active transition metals within particulate matter (PM) interact with adsorbed PAHs to generate reactive oxygen species (ROS) [79]. These ROS are recognized as the primary drivers of PM-induced cytotoxicity and carcinogenicity, leading to oxidative stress and subsequent damage to lung tissue and DNA. Similarly, Cavallo et al. found that organic compounds adsorbed on fine PM can interact with other components or physical factors through additive or synergistic pathways, thereby amplifying adverse health outcomes [80]. Due to their high specific surface area, fine particles act as substrates for metals or metal ions emitted during coal combustion, creating complex pollutants with toxicological profiles distinct from single-system contaminants. Given that oxidative damage is a major pathway for ultra-fine particle toxicity, and nanoparticle pollutants possess unique "cell-friendly" properties that facilitate cellular internalization [81], the mechanistic interplay between PAHs and fine PM represents a critical, yet under-researched, frontier in environmental toxicology.

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