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Coal Fly Ash Aerosol: Risk Factor for Lung Cancer

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Authors' contributions

This work was a joint effort between the authors that is part of an ongoing collaboration aimed at providing scientific, medical, public health implications and evidence related to aerosolized coal fly ash including its use in the near-daily, near-global covert geoengineering activity. Author MW was primarily responsible for medical and public health considerations. Author JMH was primarily responsible for mineralogical and geophysical considerations. Both authors read and approved the final manuscript.

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ABSTRACT

Aim: Coal fly ash (CFA) is a major contributor to ambient air pollution in China and India, but it is trapped and sequestered in Western nations. Members of the public chronically exposed to aerosolized CFA are likely to have an increased incidence of respiratory disease, including lung cancer. Our objective is to review the multiple carcinogenic constituents of aerosolized coal fly ash in connection with their potentiality to cause lung cancer.
Methods: We review the interdisciplinary scientific and medical literature.
Results: CFA contains a variety of potentially carcinogenic substances including aluminosilicates, an iron oxide-containing magnetic fraction, several toxic trace elements, nanoparticles, and alpha-particle-emitting radionuclides. Silica, arsenic, cadmium, and hexavalent chromium are found in CFA and all have been associated with increased lung cancer risk. Radical generation catalyzed by transition metals associated with the particulate matter in CFA can result in a cascade of cell signaling, transcription factor activation, and mediator release. Ferric iron in the aluminum-silicate glass phase of CFA is a source of bioavailable iron. There is emerging evidence that reactive iron

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induces cancer stem cells and aggressive phenotypes in lung cancer. The potential pulmonary toxicity and carcinogenicity of aerosolized CFA is suggested by studies of asbestos, a fibrous silicate that also contains iron oxide. CFA contains an abundance of ultrafine particles and nanoparticles, including magnetite (Fe_3O_4). These tiny particles are toxic to lung cells, capable of producing oxidative stress, cytotoxicity, and genotoxicity. Radioactive elements are concentrated in CFA. CFA can settle deep in the lungs where its alpha-particle-emitting radionuclides pose significant risk factors for lung cancer.

Conclusion: Considering the well-known and manifold toxicities of CFA, the public should be made aware of the potential risks for lung cancer and severe respiratory disease posed by aerosolized CFA including its use in climate alteration activities.

Keywords: Aerosols; coal fly ash; climate intervention; geoengineering; particulate air pollution; oncology; magnetite; nanoparticles.

1. INTRODUCTION

When coal is burned by electric utilities, about 10% remains as ash. The heavy ash settles, while the light coal fly ash (CFA) condenses and accumulates in the flue gases. In India and China CFA is usually allowed to exit smokestacks, but in Western nations it is trapped and sequestered for public health reasons. The public is nevertheless being exposed to aerosolized CFA, not only through inefficient trapping, especially in the 0.1-1 μm range [1,2], and windblown CFA-rich dust from dumps [3], but also as workers in the CFA industry [4]. Epidemiological evidence indicates that aerosolized particulate pollution in the size range ≤ 2.5 μm is associated with numerous risks to health including, but not limited to, lung cancer [5]. Forensic evidence is consistent with jet-sprayed CFA being widely used for tropospheric climate alteration activities in North America and Europe [6,7]. Significant information is found throughout the scientific literature bearing on the health risks of CFA. The purpose of this Review is to bring together this information, specifically calling attention to the lung cancer risk of aerosolized CFA. The resulting implications pertain to human exposure in general including, for example, workers engaged in cleaning up CFA spills [8]. As a consequence of undisclosed tropospheric climate alteration activities, pilots and flight crews may be subjected to more intense exposure than general populations on the ground.

2. METHODS

The scientific literature is rich in information pertaining to the subject of this review. This information, however, is fragmented and scattered among many different journals. We review the interdisciplinary scientific and medical literature to bring forth and to connect logically the various and diverse information that bears on the potentiality of lung cancer caused by CFA.

3. RESULTS AND DISCUSSION

CFA, a major by-product of coal-burning by electric utilities, is formed by condensing and accumulating in the hot flue gases, usually as spheres typically ranging in size from 0.01 – 50 μm in diameter. Considered too toxic to be allowed to exit smokestacks, CFA in Western nations is collected by electrostatic precipitation and sequestered. CFA, one of the world's largest industrial waste-streams, forms in just the size range needed for aerosol spraying with limited processing.

The main elements in CFA are oxides of silicon, aluminum, iron, and calcium, with lesser amounts of magnesium, sulfur, sodium and potassium [9]. Primary components of CFA are aluminum silicates and an iron-bearing fraction that includes magnetite, Fe_3O_4 [10]. Among the trace elements in CFA are the following: arsenic, barium, beryllium, cadmium, chromium, lead, manganese, nickel, phosphorus, selenium, thallium, titanium and zinc [10]. The radioactive nuclides uranium, ^{235}U and ^{238}U, thorium, ^{232}Th, and potassium, ^{40}K, are present in CFA as well as their daughter products, which includes radioactive lead, ^{210}Pb, radium, ^{226}Ra, and radon, ^{222}Rn [11-13]. CFA also contains particles of unconsumed carbon some of which are identified as soot [14,15]. Small amounts of organic molecules found in CFA include the polycyclic hydrocarbons like benzopyrene which is known to be carcinogenic [16].

Aerosolized CFA for climate alteration constitutes one form of deliberate air pollution; there is now abundant evidence that ambient air pollution contributes to the growing global burden of

respiratory disease and lung cancer [17,18]. Long term, cumulative exposure to fine particulate pollution in the U.S. is associated with lung cancer and cardiopulmonary mortality [19]. A recent study documents a 31% increase in incident lung adenocarcinoma associated with increasing ambient $PM_{2.5}$ air pollution among nonsmokers [20]. Climate intervention projects utilizing CFA constitute a covert, insidious, and nearly global form of $PM_{2.5}$ air pollution. Chronic exposure to aerosolized CFA, emplaced in the atmosphere for climate intervention, may be an important, yet unrecognized, environmental risk factor for development of lung cancer.

Lung cancer is the leading cause of cancer deaths worldwide, and it is among the most common occupation-related cancers [21]. Silica, one of the main components of CFA [9], has long been known to cause silicosis and it may also predispose to lung cancer. Recent studies have shown an excess lung mortality in silica-exposed workers who do not have silicosis and have never smoked [22]. CFA contains known carcinogens such as arsenic, cadmium, and chromium, the latter of which is about 10% hexavalent [23]. Inorganic arsenic is unique in that it has been established to cause lung cancer with exposure through both ingestion and inhalation [24]. Although earlier investigations showing an association of cadmium with lung cancer were confounded by the presence of arsenic, a more recent study supports an independent risk of cadmium in lung cancer mortality [25]. Inhalation of hexavalent chromium is associated with increased lung cancer risk in several industries, most notably chromate production [26].

Iron is a ubiquitous component of CFA; all CFA samples examined in one study [27] were comprised mostly of amorphous aluminosilicate spheres with a lesser quantity of iron-rich spheres. Most of the iron-rich spheres contained two components: Iron oxide and amorphous aluminosilicate. Mössbauer spectroscopy indicates that ferric iron in the aluminosilicate glass phase of CFA is a source of bioavailable iron [28]. Differences in iron mobilization in pollution particles are correlated with minerology, chemical speciation, and morphology of the particles. In size-fractionated fly ash, the smallest particles produce higher amounts of mobilized iron from a given source [29].

Elemental iron is essential for cell growth and homeostasis, but through redox cycling it can be toxic to cells and tissue. This transition metal is carcinogenic due to its catalytic effect on the formation of hydroxyl radicals, suppression of host defense cells, and its promotion of cancer cell proliferation. Iron-catalyzed oxidative stress causes lipid peroxidation, protein modification, and DNA damage with consequent promotion of mutagenesis [30,31]. In both animals and man, primary neoplasms develop at body sites of excessive iron deposits. The invaded host attempts to withhold iron from cancer cells via sequestration of the metal in newly formed ferritin, the main storage form of iron in the body. Quantitative evaluation of body iron and of iron-storing proteins like ferritin have prognostic value in cancer patients, including those with lung cancer [32].

Both epidemiologic and laboratory studies have demonstrated that iron excess or imbalance is associated with the tumorigenesis of lung cancer and the growth of lung cancer cells. Pathways of iron uptake, storage, efflux, and regulation are all disturbed in cancer, suggesting that reprogramming of iron metabolism is a key feature of tumor cell survival [33]. Multiple cell culture, animal models, and epidemiological studies implicate iron in the development of non-small cell lung cancer [31]. A recent study provides compelling evidence that iron induces cancer stem cells and aggressive phenotypes in human lung cancer cells [34]. Iron is one of the most reactive ions in air pollution produced by CFA. Iron participates in the anti-apoptotic effect of particulate matter and since resisting cell death is a hallmark of cancer cell, this finding may relate to the development of lung cancer after atmospheric pollution exposure [35].

There is a growing concern about radioactive elements in coal products. These agents occur naturally in coal but during combustion they become concentrated in coal ash residues. Fly ash and bottom ash contain 5-10 times more natural radionuclides than feed coal, but they are most concentrated in CFA [12]. Uranium, thorium and potassium consist in whole or in part of radionuclides with extremely long half-lives. Although the human body contains much potassium, only 0.012% of that is the radionuclide ^{40}K, which decays both by electron capture and beta decay. Uranium and thorium, on the other hand, are not naturally indigenous in human tissue, and decay through a series of daughter products emitting 6-8 highly-damaging alpha

particles in the process to become non-radioactive lead. The activity concentrations of some radioactive nuclides in CFA are shown in Table 1.

Table 1. Activity concentrations of some radioactive nuclides in coal fly ash (CFA) in samples from Uttar Pradesh and West Bengal (India) and Kentucky (USA) determined by gamma radiation spectrometry. Units are Becquerel per kilogram which is equal to 16.66 times the number of disintegrations per minute per gram. After three ^{222}Rn half-lives (11.4 days) in which secular equilibrium is established, the activity concentrations of ^{222}Rn and ^{226}Ra become nearly equal [36]

CFA Nuclide	Activity concentration Bq/kg	Reference
^{226}Ra	118.6 ± 7.4	[37]
^{222}Rn	118.6 ± 7.4	[38]
^{210}Pb	241.7 ± 16.3	[38]
^{232}Th	147.3 ± 3.4	[37]
	112.9 ± 0.3	[11]
^{238}U	99.3 ± 1.3	[11]
^{40}K	352.0 ± 4.5	[37]
	308.9 ± 2.5	[11]

The most abundant radionuclide in CFA is thorium, present exclusively as ^{232}Th. Thorium produces higher radiation levels than uranium, ^{235}U and ^{238}U being the mainradionuclides. Lung cancer mortality is known to be higher than in controls for both thorium miners [39] and uranium miners [40], and is assumed to result from radon exposure: ^{222}Rn from the ^{238}U decay series. Radon, ^{222}Rn, exposure from ^{238}U in rocks may be the second most common cause of lung cancer and the first risk factor in nonsmokers [41].

The small grain size of CFA, extending into the nanoparticulate range, means that when inhaled these particles become trapped in terminal airways and alveoli where they remain for long periods of time. Fission track studies of CFA glassy particles demonstrate that uranium is distributed more-or-less uniformly on their surfaces [38]. Alpha particles emitted from those surfaces can damage lung tissue and as evidenced [42] cause lung cancer. Typically, alpha-emitting radon and lead radionuclides with comparatively long half-lives are mentioned as risk factors, for example, ^{222}Rn and ^{210}Pb [43]. But tiny aerosolized CFA particles which are inhaled and settle deep in the lungs potentially cause cancer both from short- and long-lived alpha particle emitters (Table 2).

Previous pulmonary toxicology studies of non-fractionated CFA reported that coarse particles were relatively inert with minimal respiratory effects in animal studies [45]. However, more recent studies of the effects of size fractionated CFA particles show significant pulmonary toxicity of ultrafine and CFA nanoparticles (CFA-NP's) [46]. The smaller the size of the particle, the greater its surface area is to its volume ratio, and the higher its chemical and biological reactivity [47]. Ultrafine particles and nanoparticles are small enough to enter the body transdermally [48,49]. Beyond shape and size, increasing attention is be paid to particle/fiber chemistry as a determinant of variables such as dissolution behavior, ion exchange, sorption properties and surface reactivity [50,51].

Table 2. Alpha particle-emitting nuclides present in coal fly ash (CFA). Percent isotopic abundances of parent nuclides are indicated. Data from [44]

Alpha particle emitting CFA nuclides	Uranium		Thorium
	^{238}U (99.2746%)	^{235}U (0.720%)	^{232}Th (100%)
Uranium	^{238}U ^{234}U	^{235}U	
Protactinium		^{231}Pa	
Thorium	^{230}Th	^{227}Th	^{232}Th ^{228}Th
Actinium		^{227}Ac	
Radium	^{226}Ra	^{223}Ra	^{224}Ra
Francium		^{223}Fr	
Radon	^{222}Rn	^{223}Rn	^{220}Rn
Astatine	^{218}At	^{219}At ^{215}At	
Polonium	^{218}Po ^{214}Po ^{210}Po	^{215}Po ^{211}Po	^{216}Po ^{212}Po
Bismuth	^{214}Bi ^{210}Bi	^{211}Bi	^{212}Bi
Lead	^{210}Pb		

Use of ultrafine grain sizes of aerosolized CFA particulates for climate alteration is advantageous for increasing residence time in the convecting troposphere, but this activity increases the respiratory risks. Ultrafine (0.1-1 μm) particles and nanometer-sized particles (<100 nm) are both found in CFA. The key to understanding the toxicity of nanoparticles is that their minute size, smaller than cells and cellular organelles, allows them to penetrate these biological structures, disrupting their normal function. Examples of toxic effects include tissue inflammation, and altered cellular redox balance toward oxidation, causing abnormal function or cell death [52]. CFA nanoparticles with surficial toxic heavy metals can act as cellular and DNA toxicant, capable of inducing inflammation, oxidative stress, DNA damage and cell death [47].

Exposures to particles and fibers are associated with many lung diseases including lung cancers, mesothelioma, chronic bronchitis, emphysema, pneumonitis, and pneumoconiosis. All particles and fibers have the capacity to present an oxidative stress to the lung [53], and among the characteristics shared by all of these particles introduced into the lung is the creation of a solid-liquid interface into the lower respiratory tract. Free radical production by fibers and particles in coordination with transition metals with two stable valence states can be observed at this solid-liquid interface [53-55]. For example, the same divalent character of iron that plays an important biologic role may also cause toxicity by sustaining oxidative conditions [56]. Radical generation catalyzed by metals associated with fibers and particles can result in a cascade of cell signaling, transcription factor activation, and mediator release [57-59]. Clinical manifestations of this process can present as inflammatory, fibrotic, and neoplastic disease.

Transmission electron microscopy investigations reveal an abundance of magnetite nanoparticles (NP's) among ultrafine CFA particles [60]. There are a growing number of reports of pulmonary toxicity from inhalation of magnetite, including nanoparticulate magnetite. Four different size fractions of magnetite on human alveolar epithelial cells showed adverse effects including cytotoxicity, genotoxicity, and increased production of reactive oxygen species [61]. Lung epithelial cells, treated with various concentrations of magnetic nanoparticles, showed that magnetite-treated cells induce oxidative stress, deplete antioxidant levels, and affect the apoptotic pathway [62]. Note the commonality: Iron oxide is a component of air pollution, CFA, and asbestos [56,63,64]; magnetite (Fe_3O_4) is even found in cigarette smoke and ash [65].

Titanium-rich nanoparticles (TiO_2 NP's) are also found in CFA. Whereas aluminosilicates are dominant in the micrometer size range in CFA, large numbers of iron and titanium particles in the ultrafine size range are present. TEM analysis of CFA reveals both titanium and iron oxide nanoparticles which exhibit highly crystalline characteristics [66]. Long term studies by method of intratracheal instillation confirm the carcinogenicity of submicron titanium oxide nanoparticles in rats [67]. There is evidence that titanium NP's can induce cytotoxicity, significant DNA damage, and apoptosis in human non-small cell lung cancer A549 cells [68]. Bioavailable nickel NP's are also found in CFA [69]. Metallic nickel and nickel oxide NP's are toxic to human lung epithelial cells [70].

Further implications of the pulmonary toxicity and potential carcinogenicity of aerosolized CFA are suggested by studies of asbestos, a fibrous silicate [71]. The presence of transition metals like iron in asbestos fibers and the ability of these fibers to attract iron from the surrounding environment may be key factors for asbestos toxicity and for the formation in the lung of the asbestos (ferruginous) bodies that characterize lung disease caused by asbestosis. Synchrotron-based scanning x-ray microscopy has demonstrated that long-lasting asbestos fibers and particulates cause a large mobilization of iron into the surrounding cells (mainly alveolar macrophages) and in tissue, which is partially a consequence of continuous iron adsorption onto the fibers and/or asbestos body degradation and metal release [56]. Iron (including magnetite) is an integral component of pathogenic amphibole (crocidolite, amosite) asbestos fibers and it occurs as a mineral contaminant of chrysotile (serpentine) asbestos [48, 72]. Studies suggest that chrysotile is not toxic by simply acting as a carrier of iron into the cell, but rather the redox activity of iron is potentiated when organized at the fiber's surface into specific crystallographic sites having coordination states able to generate free radicals [73].

Published scientific data demonstrates that CFA, a known environmental hazard [74], is consistent with the previously undisclosed material used in widespread, persistent atmospheric aerosol

Table 3. Alpha particle-emitting nuclides present in coal fly ash (CFA). Percent isotopic abundances of parent nuclides are indicated. Data from [44]

Carcinogenic agents with sufficient evidence in humans, common to or contained in CFA
Arsenic and inorganic arsenic compounds
Beryllium and beryllium compounds
Cadmium and cadmium compounds
Chromium(VI) compounds
Coal, indoor emissions from household combustion
Gamma-radiation
Iron and steel founding
Nickel compounds
Particulate matter in outdoor air pollution
Radon-222 and its decay products
Silica dust, crystalline
Soot

climate intervention [6,7,75-79]. The covert nature of those operations currently limits the ability to quantify human exposure to this deliberate form of air pollution or to separate it from other forms of air pollution caused by human activity. A person's level of exposure to air pollution depends on a variety of factors relating to the host, the environment, and their interaction. Newer bio-monitoring techniques should enable more accurate measurements of exposure to specific air pollutants [80], which may be useful to estimate dose-response, exposure assessment, and risk characterization from published data on the known toxic component-elements of CFA [74].

In this Review we have disclosed some of the potential public health hazards of aerosolized CFA, focusing on the special risks for lung cancer. In Western nations, where trapping and sequestering is practiced, there may be a false assumption that only those living or working in close proximity to CFA dumps potentially risk exposure. As the principal undisclosed particulate used for climate intervention operations is consistent with CFA [6,7], a widely available waste product that requires little processing; the exposure risk is neither localized nor limited in scale. Potentially hundreds of millions of people might be at risk even at low exposure levels; airline flight crews and frequent fliers may be more at risk. Like the lung cancer risk for cigarette smoking, the full consequences of this type of air pollution might be decades away.

For more than 40 years, the Monograph series of the International Agency for Research on Cancer (IARC) has classified human carcinogens. Researchers rearranged that data according to specific cancer sites for each relevant carcinogen, and further subdivided those into the known and suspected causes of cancer [81]. In Table 3 for lung cancer we abstract from that tabulation the relevant known carcinogens that are components of or common to CFA.

4. CONCLUSION

CFA contains a plethora of potentially carcinogenic agents likely to have cumulative additive and/or synergistic interactions with long-term exposure. The CFA industry can be diligent about minimizing the likelihood of CFA aerosolization for sake of workers and those living in the proximity of CFA dumps. Jet-spraying of CFA into the regions where clouds form represents a potential global and previously unrecognized long-term risk factor for respiratory disease and lung cancer, especially in vulnerable populations.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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