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Health Effects of Inhalation of Coal Combustion Products

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ABSTRACT

This report assesses the potential human health effects of inhaled coal combustion products (CCPs), which consist of fly ash, bottom ash, boiler slag, and flue gas desulfurization (FGD) products. The focus is on as-managed CCPs, with evaluation of the potential effects of exposure through fugitive emissions from storage facilities. Because the literature pertaining to bottom ash, boiler slag, and FGD solids is scarce, this review draws almost entirely from studies of fly ash as a surrogate particulate matter (PM) type. Pertinent *in vitro, in vivo*, and epidemiological studies that contribute to the understanding of human health effects are reviewed.

The report summarizes plausible mechanisms of action and notable effects resulting from CCP exposure and describes their significance with reference to the general human population. Most of the studies performed have used exposure concentrations or doses that are far greater than those anticipated during environmental or even occupational exposures. Therefore, although adverse effects have been observed in some experiments, it is difficult to compare the biological relevance of the experimental findings to that of human exposure. Some studies, including a recent study that assessed realistic exposures *in vivo*, found no significant adverse effects at high and low exposure concentrations. Therefore, it is plausible that fugitive dust emissions from CCP management facilities might similarly represent a low threat to human health through inhalation exposure, although data on concentrations of resuspended particles downwind of these facilities are currently scarce. A follow up to this report will consider the issue of exposure more comprehensively and will, therefore, present a more integrated estimation of the potential human health risk of as-managed CCPs.

Keywords
Boiler slag
Bottom ash
Coal fly ash
Epidemiology
FGD solids
Toxicology

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INTRODUCTION

1.1 Introduction

United States coal-fired power plants burnt more than 1 billion tons of coal annually and produced nearly 82 million tons of coal ash in 2009 (ACAA 2011; EPRI 2009). Of interest are the potential health effects due to exposure to stored (as-managed) coal combustion products (CCPs). Unfortunately, there are few data regarding the toxicity of this material, so the bulk of our knowledge of this issue comes from studies conducted using fly ash as a surrogate for ambient particulate matter (PM). It is well established that emissions from coal-fired power plants contribute to both ambient PM levels and gaseous pollutant levels such as SO_2 and NO_2, but the health effects of their exposure, particularly though inhalation, have yet to be fully understood. It is thought that particle size, particle acidity, transition metal content (e.g. Cu, Fe, V, Ni, and Z), and polyaromatic hydrocarbon (PAH) content could be plausible mechanisms by which PM could induce health effects (Amdur et al. 1986; Fernandez et al. 2002; Mauderly et al. 2011). Primary PM – which is often used interchangeably with “fly ash”, although this is not accurate as primary PM, by definition, is that which escapes particulate controls at the plant and is emitted into the atmosphere, while fly ash is the collected material - contributes to ambient PM in the US via stack emissions. Fly ash has been studied primarily in the context of this contribution to ambient PM; in this context it represents a significant potential for human inhalation exposure compared to other CCPs such as bottom ash, boiler slag, and flue gas desulfurization (FGD) products (Chen and Lippmann 2009; Costa DL and Dreher KL 1997; Ozkaynak H and Thurston GD 1987) which are not emitted to the atmosphere. However, the focus of this report is on potential exposures to fugitive emissions in the vicinity of CCP storage facilities.

While other coal combustion products (CCPs) such as bottom ash, FGD solids, and boiler slag may be generated during the coal combustion process, fly ash has been the most studied of the CCPs, primarily because it (or a closely related material) is released to the atmosphere and therefore contributes to population exposures to ambient PM. Also, its size range is relevant to human inhalation exposure, with the potential for deep lung deposition. There are many experimental studies investigating the effects of inhaled coal fly ash; however, as Mauderly et al. note, most laboratory settings suffer from conducting exposures which are several magnitudes higher than those that would occur in the natural environment (Mauderly et al. 2011). Furthermore, few exposure studies are able to adequately reproduce coal combustion emissions for use in generating inhalation exposures due to the complex physicochemical processes that occur during combustion. Ultimately, many studies use intratracheal instillation or oropharyngeal aspiration as a method of exposure (Mauderly 2003). That being said, many studies have investigated the effects of inhaled CCPs, particularly fly ash, using a wide variety of methods including *in vivo* exposures, *in vitro* exposures, and epidemiological studies. This review presents an update of the literature related to the effects of inhaled coal combustion products.

The objective of this literature review is to evaluate the potential health effects associated with inhalation of as-managed CCPs. Since most of the literature pertains to health effects of fine particles in power plant stack emissions, these will be used as a surrogate. These data will be used to provide a preliminary assessment of the potential for inhalation health effects from as-managed CCPs.

1.2 Coal Combustion Products (CCPs)

As coal is combusted, CCPs are generated in several forms which can be categorized into groups based on where in the combustion process they are generated. These broad categories include fly ash, FGD solids, bottom ash, and boiler slag. When organic matter is released from coal during combustion, the inorganic content falls to the bottom of the combustion chamber as bottom ash or falls to of the bottom of the boiler as boiler slag, or it is present in the flue gas as fly ash (EPRI 2010). All of the bottom ash and boiler slag, and nearly all of the fly ash are captured by power plant environmental control systems and either disposed of or used in a variety of construction or geotechnical applications. FGD products are produced when an alkaline material such as limestone or lime is added to the flue gas to react with the sulfur dioxide that is generated during the combustion of coal to produce calcium-sulfur products that can then be used in commercial processes or disposed of via landfilling or ponding (EPRI 2010).

Coal can be combusted in a variety of different ways including stoker fired combustion, fluidized bed combustion, pulverized coal in dry-bottom boilers (most common), slag-tap boilers, as well as in cyclone boilers, with each method producing different CCPs of varying physicochemical properties (Borm PJ 1997). There are four types of coal used for combustion purposes which all vary on their heating value, chemical composition, ash content, and geological origin: anthracite, bituminous, sub-bituminous, and lignite coals (Ahmaruzzaman M. 2010). It is well known that the type of combustion process used, the emission control equipment used, and the type of coal used can alter the emitted CCP characteristics, such as size and chemical composition of the particles generated (Borm PJ 1997). Thus, it is often difficult to compare the results of one study to another.

1.2.1 Fly Ash

Coal fly ash particles of combusted coal represent approximately 78% of the total captured coal residue depending on the type of coal and method of combustion used (EPRI 2009). A variety of particle control technologies exist including cold- or hot-side electrostatic precipitators, fabric filter bag houses, and wet particulate scrubbers and can remove upwards of 99% of fly ash in the flue gas stream (EPRI 2008).

Typically, coal fly ash is dominated by silica and aluminum with significant amounts of iron and calcium present, as well as a variety of trace elements. Fly ash itself is a fine spherical powdery particle, either solid or hollow in nature (Ahmaruzzaman M. 2010), and while most fly ash is disposed of in landfills, it is also widely used in concrete manufacturing processes or other industries (Alvarez-Ayuso et al. 2006).

1.2.2 Flue Gas Desulfurization (FGD) Solids

Flue gas desulfurization may operate using wet or dry techniques. Wet limestone FGD systems, often called scrubbers, are the most common due to their high desulfurization performance, reliability, and low energy consumption (Alvarez-Ayuso et al. 2006; Kikkawa et al. 2002). In the wet limestone FGD system with forced oxidation, SO_2 is scrubbed from the flue gas by absorption into a limestone slurry to form a gypsum slurry which can be dewatered and used in commercial applications such as wallboard manufacturing (Alvarez-Ayuso et al. 2006). Dry FGD systems, often called spray dryers, produce a dry product that does not require dewatering such as in the wet system (EPRI 2008). In 2009, an estimated 1.4 million tons of solids were produced by dry FGD systems and 11.7 million tons of calcium sulfite hemihydrate and 18 million tons of FGD gypsum were produced by wet FGD systems (ACAA 2011). The primary emissions of FGD systems are CO_2, due to the chemical reactions involved in removing sulfur. Other gas contaminants, specifically acid gases and mercury vapor, can also be captured in the wet FGD system or can be present in the gas effluent (Lee JY et al. 2009).

1.2.3 Bottom Ash and Boiler Slag

combustion which fall to the bottom of the furnace or boiler. Boiler slag is similar to bottom ash with the exception that collection occurs in a wet-bottom boiler which uses quenching water to cool molten particles generated from coal combustion. Similar in composition but coarser (0.2 mm to 50 mm) than fly ash, bottom ash is dominated primarily by aluminosilicate materials (EPRI 2009). The health effects of bottom ash and boiler slag are not well studied, likely due to their larger size and lower potential to be inhaled, combined with lower leaching characteristics when compared to fly ash.

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TOXICOLOGICAL EFFECTS

Historically, the toxicological evaluations of CCPs, primarily coal fly ash, have conducted *in vitro* and *in vivo* studies using materials collected from an electrostatic precipitator hopper. Thus, the collected exposure materials used in these experiments may have slight physicochemical differences when compared to down-wind primary particles. Furthermore, the concentrations at which the exposures occurred were typically far greater than that which the general population would encounter. Therefore, it is often difficult to make general conclusions about the human health effects associated with primary CCP particle exposure.

2.1 *In vitro* Studies

In vitro studies have primarily been used to quantitatively determine the effects or mechanisms of action of CCPS, primarily coal fly ash. In this report the studies have been broken down into reactive oxygen species (ROS) generation, cytotoxicity, mutagenicity and genotoxicity as these are the primary areas of researcher generated literature.

2.1.1 ROS Generation

The ability of CCPs, particularly fly ash, to generate ROS due to Fenton reactions or other mechanisms is an important part in understanding the particle's toxicology. Several studies have found that coal fly ash can generate ROS and that transition metals may be responsible for the observed adverse effects. Aust and Smith and colleagues found that *in vitro* exposure to coal fly ash resulted in an increase in ferritin, an indicator of excess iron, as well as an increase in interleukin-8 (IL-8), an inflammatory mediator which also appeared to have a size dependent response (Aust et al. 2002; Smith et al. 1998). In both cases, these researchers used the same three coal sources to investigate the potential ROS generation of fly ash; low-sulfur bituminous coal from the Utah Deer Creek mine, bituminous Illinois coal with a high iron concentration from the Consol mine, and a lignite coal from the Knife River mine in North Dakota. Similarly to previous findings, and using the same coal sources, Smith et al. (2000) found that coal fly ash could induce IL-8 in human lung epithelial cells; an effect which was even more pronounced when sub-micron particles were used (Smith et al. 2000). These authors demonstrated that iron present in the fly ash may be responsible for the observed effects through the generation of free radical species. In support of this, work by van Maanen and colleagues found that hydroxyl radical generation and associated oxidative damage to rat lung epithelial cells was related to iron as well as particle size (van Maanen et al. 1999). The samples used for their study were collected from a coal gasification facility in the Netherlands and the origin of the coal is unknown. Using coal fly ash samples from a western US power plant burning western coal and coal fly ash from the Exxon Research and Engineering Company, Prahalad and colleagues found that the samples could not significantly induce dG hydroxylation to 8-oxo-dG formation (Prahalad et al. 2000; Prahalad et al. 2001). Thus, when ROS generation or its ensuing damage occur, it appears that metals, particularly metal availability, play the most important role in toxicity().

2.1.2 Cytotoxicity

In a comparative study using samples from coal gasification, fluidized bed combustion, and conventional combustion coal fired plants, Garrett and colleagues found that coal fly ash samples were the most toxic to rabbit pulmonary alveolar macrophages (Garrett et al. 1981). Similarly, other researchers showed that exposure of rat lung epithelial cells or alveolar macrophages to coal fly ash can result in cytotoxicity as well as cause functional reductions in activity which may be attributed to size and/or bioavailable metal components (Aranyi C et al. 1979; Fisher GL and Wilson FD 1980; Kondo T et al. 1993; van Maanen et al. 1999).

2.1.3 Mutagenicity and Genotoxicity

Morris et al. (1989) determined that of the 7 samples tested for their mutagenicity potential using bacterial, *in vivo*, and *in vitro* methods, none proved positive (Morris DL et al. 1989). These samples were obtained from Battelle Columbus Laboratories and were collected downstream from the electrostatic precipitators. Similarly, Ahlberg et al. (1983) also found that the Polish coal (13% ash content and 0.8% sulfur content) stack samples collected used in their experiments were not mutagenic as well (Ahlberg et al. 1983). That being said, Chrisp and colleagues found that fly ash from combusted pulverized low-sulfur, high ash coal collected downstream of the electrostatic precipitator could produce mutations both with S9 activation and without requiring enzyme activation using the Ames assay (Chrisp et al. 1978). Since PAHs require bioactivation to become mutagenic, the mutagenicity potential of fly ash may not be solely attributed to PAHs alone (Fisher 1983). However, Griest et al. (1982) found that post-electrostatic precipitator fly ash samples required the presence of aromatic hydrocarbons and polar organic compounds in order to be mutagenic (Griest et al. 1982). Further, Chrisp and Fisher showed that the same fly ash samples of 3.2 µm were more mutagenic than the 2.2 µm fraction of fly ash tested suggesting that particle size may also play a role in mutagenicity, perhaps due to the adsorbed species on the particle (Fisher et al. 1979). The mutagenicity and cytotoxicity of coal fly ash has also been demonstrated by others using a laboratory scale plant (Mumford and Lewtas 1982). Work by van Maanen and colleagues suggest that coal fly ash may be genotoxic by causing oxidative DNA damage and ultimately cytotoxicity (van Maanen et al. 1999).

2.2 *In vivo* Studies

2.2.1 Whole Body and Nose-only Exposure

Several studies have reported that the inhalation of fly ash did not result in significant adverse effects (Fisher 1983; MacFarland HN et al. 1970; RAABE et al. 1982). For instance, a high concentration fly ash inhalation exposure (180 day 8hr/day exposure to 2 µm fly ash particles at a concentration of 4.2 mg/m^3) using cyanmolgus monkeys resulted in no significant adverse findings (MacFarland HN et al. 1970). Similarly, a chronic inhalation study using low-sulfur, high ash coal (particles <3 µm) did not induce any significant adverse health effects in rats (RAABE et al. 1982). Fisher (1983) notes that many *in vivo* studies use particle concentrations that are not biologically relevant (Fisher 1983) and therefore extrapolating toxicological effects from stack collected samples is difficult.

Recently, a more robust, large scale study known as the TERESA project was completed which assessed emissions from three coal-fired power plants in the US with the addition of photochemical ageing equipment to simulate downwind power plant emission scenarios. This study conducted extensive exposure and toxicological characterization on Sprauge-Dawley rats under a variety of scenarios and assessed several outcomes including breathing patters, pulmonary inflammation responses and cardiovascular response (Godleski et al. 2011b). Using a variety of different scenarios, the researchers ultimately found that little to no response was observed in response to the aerosol exposure, however, the more complex scenarios involving forced oxidation of emissions and secondary organic aerosol simulations produced more significant responses than the oxidized and non-oxidized emissions alone (Godleski et al. 2011a). Interestingly, the TERESA project did not identify specific metals or sulfate as a key toxicant in affecting respiratory response although breathing parameters were associated with some metals and gases (Al, Si, Pb, Mg, Ni, Na, NO, NO_x).

It is clear that the type of coal used in *in vivo* exposures affects the responses measured. For instance, Chen et al. (1990) found that Illinois no. 6 fly ash but not Montana lignite fly ash caused changes to guinea pig total lung capacity, vital capacity, and diffusing capacity for carbon monoxide (Chen LC et al. 1990) which may be attributed to the acidification of particles due to the formation of sulfuric acid. Differences between emissions from the three TERESA sites also exemplify how changes in coal type and emission controls can alter toxicological study outcomes and thus future studies should fully characterize these.

Chauhan and colleagues performed a whole body exposure to collected fly ash (<40 µm) in male Wistar rats using a Wright dust feeder for 6 hrs per day for 15 days at a concentration of 270 mg/m^3 (Chauhan et al. 1987). The authors found that fly ash significantly reduced WBC, RBC, and hemoglobin content following exposure, but returned to baseline values after a recovery period. The exposure also coincided with thickening of alveolar septa, and alveolar dilatation which were also reversible. Using re-suspended particles at a concentration of approximately 1 mg/m^3 collected from a pilot scale combustion unit, Fernandez and colleagues (2003) found that exposure caused slight decreases in lung permeability which were reversible in 12 days (Fernandez et al. 2003). Kircher et al. (1983) found that after a 500 or 1000 hr inhalation exposure to high concentrations of resuspended fly ash (20.8 mg/m^3 and 19.8 mg/m^3, respectively) from a fluidized bed coal combustor that there was little mutagenicity detected in the fly ash samples tested and that the biological reactions to the exposure were characteristic of chronic lung irritation (Kirchner et al. 1983).

While the immune effects of fly ash exposure are not as defined for other toxicological endpoints, Fujimaki et al. (1989) found that coal fly ash may act as an adjuvant to cause the release of IgE and IgG antibodies (Fujimaki et al. 1989). Dormans and colleagues found that inhalation of re-suspended coal fly ash particles from a small scale (3.8 MW) combustion scale in male wistar rats (0, 10, 30, or 100 mg/m^3) for 6 h/day for 5 days resulted in alterations in lymph node cell count (all doses), increase in serum IgA (30 and 100 mg/m^3), increased number of alveolar macrophages (100 mg/m^3) (Dormans et al. 1999).

2.2.2 Intratracheal Instillation and Aspiration Exposure

Using ultrafine (<0.2 µm), fine (<2.5 µm), and coarse (>2.5 µm) coal fly ash from combusted low-sulfur subbituminous coal and high-sulfur bituminous coal, Gilmour and colleagues intratracheally instilled CD1 mice with 25 µg or 100 µg in order to examine the pulmonary inflammatory response (Gilmour et al. 2004). The findings, observed in the bronchoalveolar fluid 18 hrs post-exposure, showed significant influx of neutrophils in the low-sulfur subbituminous ultrafine group compared to the saline group which coincided with significant increases in TNF-α, MIP-2, and IL-6. However, Ogugbuaja et al. (2001) found that rabbits instilled with coal fly ash at 50, 100, and 200 mg/kg body weight produced no significant differences in haematological parameters such as red and white blood cell count, hemoglobin levels, packed cell volume, and platelet counts (Ogugbuaja et al. 2001). Similarly, a 15 week intratracheal instillation of coal fly ash (4.5 mg) failed to produce any malignant tumors in golden hamsters (Persson et al. 1988). The varying results illustrate how not only dose, but also size and parent coal type may affect the study outcomes.

3

PM CONTRIBUTION AND EPIDEMIOLOGICAL AND OCCUPATIONAL STUDIES

Due to the lack of robust information regarding the contribution of primary PM by coal fired plants, most epidemiological studies do not focus solely on CCP associated health outcomes - although a few studies have attempted to make such estimations. Chow and Watson (2002) found that coal combustion plants contribute less than 2% of the primary PM_{10} and $PM_{2.5}$ mass when emissions are controlled, such as in the US, and that the largest primary PM_{10} and $PM_{2.5}$ contribution results from gasoline and diesel vehicle exhaust (Chow and Watson 2002). Their analysis further shows that the primary contributions to PM are negligible once the emission control equipment has been modernized. A more recent estimation suggest that that coal plants contribute less than 4% of all primary $PM_{2.5}$ (Marmur et al. 2006). The discrepancies between studies likely arises from the models used to calculate such estimations.

In addition to general population exposure to CCPs, particularly fly ash, Smith and colleagues (2006) note that power plant workers may be exposed to excessive levels of particles, especially during boiler maintenance, and that construction workers may be exposed to fly ash that is reused in cement or as fill material (Smith et al. 2006). A recent study found that PM_1 and $PM_{0.1}$ particles in a coal fired power plant were highest near the combustion sources where they were several times more than upwind concentration, and lowest in air-conditioned rooms where levels were the same as or lower than upwind concentrations (Hicks et al. 2011).

A recent life cycle risk assessment of bottom ash reuse found that laborers would experience the highest risk associated with dermal cadmium exposure for bottom ash use in paving and landfilling while ash treatment workers had the highest risk from chromium due to an inhalation exposure (Shih and Ma 2011). In addition to these metals, mercury may pose a health risk. Unlike coal fly ash which does not release much of the adsorbed mercury, Gustin and Ladwig reported that mercury captured during the FGD process can be released over time, thus representing a potential downstream hazard post-landfilling or during reuse (Gustin and Ladwig 2010; Heebink and Hassett 2002).

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DISCUSSION

It is has been shown that fly ash toxicity is inversely related to particle size and is often associated with increased trace metal and sulfur content, particularly in the 1-10 µm region, although in some studies show that submicron fly ash particles elicited a larger effect than their larger counterparts (Gilmour et al. 2004; Reddy et al. 2005; Smith et al. 1979). And while laboratory studies have typically shown that high concentrations of CCPs, particularly fly ash, can induce a variety of effects including a mild lung and blood inflammatory response, contradictory research has found that fly ash particles do not appear to have a greater potency than ambient particles to cause pulmonary effects (Smith et al. 2006). In general, research suggests that coal fly ash has relatively low toxicity compared to mined coal or quartz – especially when compared to a potential general human exposure (Borm PJ 1997).

While recent as well as previous reports have shown little to no adverse effects after inhalation exposure, others have shown that fly ash particles can activate alveolar macrophage and epithelial cells causing the release of inflammatory mediators, alter lung permeability, generate ROS and enzymes such as elastase, proteases, and collagenases as well as cytokines such as TNF-α and MIP-1, and growth factors such as TGF-β (Borm PJ 1997; Broeckaert et al. 1999; Fernandez et al. 2002; Fernandez et al. 2003). However, work by Broeckaet and colleagues have shown the coal fly ash can inhibit TNF-α release in alveolar macorophages (Broeckaert et al. 1997; Broeckaert et al. 1999). Work by Smith et al. (2006) showed that rats exposed to high concentrations of $PM_{2.5}$ (1.4 mg/m^3) and PM_1 (600 $µg/m^3$) of coal fly ash for 4 hrs/day for 3 days induced a mild neutrophilic inflammatory response in the lungs and blood (Smith et al. 2006). As previously noted, the concentration used in these studies were high and therefore extrapolating these experiments to plausible human exposures is difficult.

In some studies, coal fly ash has been shown to have mutagenic properties which may be due to PAH content, even though PAHs are present in fly ash at low concentrations. In one measured instance, it was estimated that a site worker would be exposed to approximately 5 pg/m^3 – well below acceptable concentrations for ambient air (Meij, R., te Winkel, H. 2001) and therefore likely a negligible exposure to the general human population. It is possible that site workers or those who reside near coal plants or where CCPs are landfilled may have the highest exposure to CCPs, particularly through inhalation although the concentrations they may be exposed to are not well studied. Using an animal model to assess lung clearance of coal fly ash, Negishi (1995) found that alveolar macrophages are efficient at removing the deposited particles but that particle laden cells typically remain in the alveoli due to loss of cell migration capability (Negishi T 1995). Matsuno and colleagues did not find fly ash in other organs and noted that clearance of fly ash deposited in the lungs was slow suggesting that translocation of fly ash does not occur (Matsuno K et al. 1986). Thus, while normal macrophage clearance mechanisms appear to remove fly ash from the lungs, an overburden would result in loss of clearance function. However, it should be stressed that the concentration of particles used in the animal exposures is well beyond levels found in the environment – even in an occupational exposure setting.

There is currently no workplace limit for coal fly ash however the ACGIH occupational limit is 10 mg/m^3 and 3 mg/m^3 for inhalable and respiratory size range particles, respectively. In terms of emissions, the National Ambient Air Quality Standard (NAAQS) has regulated PM$_{10}$ to 150 µg/m^3 over 24 hrs, and PM$_{2.5}$ to 15 µg/m^3 and 35 µg/m^3 annually and daily, respectively. In both cases, recent estimates of the contribution of coal powered plants in the US to PM emissions have been shown to well below these levels, especially for primary PM releases.

While there is abundant literature illustrating adverse effects associated with CCP inhalation exposure, particularly coal fly ash, most of these studies were conducted at high concentrations and are not biologically relevant when compared to a realistic lower dose human exposure. The TERESA project attempted to correct this issue by designing a robust toxicological study for stack emissions which factored in a variety of known flaws into their study design. Ultimately, their results failed to identify a specific chemical species that may explain some of the past studies results and noted that the observed effects from the inhalation exposures were generally non-significant in nature. Thus, given the relatively small estimation of PM contribution to overall ambient PM from coal-fired power plants stack emissions and recent findings assessing the effects of inhaled CCPs, it appears that these particle emissions may not present a significant threat to the general human population. Therefore, it is plausible that fugitive dust emissions from CCP management facilities may similarly represent a low threat to human health via inhalation exposure; although data on concentrations of resuspended particles downwind of these facilities are currently scarce. A follow-up to this report will consider the issue of exposure more comprehensively and will thus present a more integrated estimation of the potential human health risk of as-managed CCPs.

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