Public Health Impact of Coal-fired Power Plants: A Critical Systematic Review of the Epidemiological Literature

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**Abstract**

BACKGROUND: Coal-based energy production is the most utilized method of electricity production worldwide and releases the highest concentration of gaseous, particulate, and metallic pollutants compared to any other form of electricity production. Toxicological research has shown that coal combustion by-products are carcinogens, endocrine disruptors, and cardiorespiratory toxins.

OBJECTIVES: The vast majority of the epidemiological literature primarily assesses the contribution of indoor coal combustion on health and neglects the question of the public health impact from coal-fired power plants. This article is the first systematic review of the epidemiological literature on the impact emissions from coal-based power production has on morbidity and mortality worldwide.

METHODS: A systematic review of the epidemiological literature was conducted on PubMed, Web of Science, and Toxline platforms from 1998–2018. Information on study design, population, exposure assessment, outcome measurement, adjustment for confounding, consideration of bias and measure of effect was recorded in a systematic manner.

RESULTS: 2,152 articles were retrieved based on search criteria. Word search of abstract and article text filtered the results to 156 articles which were reviewed by two expert reviewers. Of those 34 articles were included after screening.

DISCUSSIONS: There is substantial epidemiological evidence indicating emissions from coal-based power production negatively impacts the health of neighboring populations. Our review illustrates significant impact on respiratory, cancer and birth outcomes from gaseous, particulate, and metal emissions from coal-fired power plants. There is considerable inconsistency among the literature on exposure assessment models

Keywords: air pollution, coal energy, power plants, particulates

# Introduction

Coal is a combustible sedimentary organic rock comprised of dense hydrocarbons. Combustion of coal produces an exothermic reaction releasing particulate, gaseous and metallic pollutants into the environment. The health effects of coal are primarily known from the extensive research on indoor air pollution secondary to combustion of coal for cooking and heating throughout the developing world. An International Agency for Research on Cancer (IARC) systematic review has determined that indoor emissions from combustion of coal as a group 1 carcinogen (IARC 2012a). While indoor coal combustion continues to be a primary source of indoor air pollution for much of the developing world (WHO 2018), coal-based energy production is a major contributor to ambient air pollution worldwide. Despite this, the majority of epidemiological and toxicological research deals with indoor exposures and does not adequately research the potential public health risk associated with coal-based energy production. This is the first systematic review of the literature to date on the impact emissions from coal-based power production has on morbidity and mortality worldwide. We also present a critical assessment of the current literature, identify methodological limitations, impact on policy, and suggest directions for future research.

***The use of coal in power generation***

Coal-fired power plants produce electricity through the rotation of a turbine by the steam produced when coal combustion occurs under high-pressure. As a result, coal-fired power plants release 84 of the 187 compounds listed as “hazardous air pollutants” by the U.S. Environmental Protection Agency (2018). Coal is the largest fuel source for electricity production worldwide; according to the International Energy Agency, “coal is the biggest single source of energy for electricity production and its share is growing” (International Energy Agency 2010). This is primarily due to the prevalence of coal-fired power plants, expense of transitioning to cleaner sources of energy production, and the relatively low cost per ton of coal.

The public health impact of coal-based energy production is a particularly timely issue as the general trend of decreasing dependence on fossil fuel-based energy, specifically coal, has been called into question in the United States, the global leader in energy consumption (Friedman and Plumer 2017). While there has been a steady decline of coal-based energy production in the US over the past 30 years, political pressures since the inauguration of President Trump in 2017 have reopened development and investment of coal-based energy and coal mining (Dlouhy et al. 2018). Worldwide, over 1,600 coal-fired power plants are either under construction or planned in 62 countries (Tabuchi 2017). In most parts of the world coal-fired power plants is the primary source of power generation with capacity on the rise (Mokhtar et al. 2014). China's dependence on coal more than doubled between 2002 and 2012, with as much as 75% of energy production from coal-fired power plants. The United States is second to China in worldwide coal consumption (International Energy Agency 2016). Coal combustion accounted for 39% of US energy production in 2013 and has increased to 44.6% in 2017 (U.S. Environmental Protection Agency 2018).

***Ambient emissions from coal-fired power plants***

Energy production is a significant source of gaseous and particulate emissions throughout the world, accounting for 70%, 16% and 12% of total SOx, NOx, and PM2.5 emissions respectively (Caiazzo et al. 2013). Coal-fired power plants constitute a large majority of all emissions related to energy production. In the United States coal-fired power plants account for 60% of all sulfur dioxide, 50% mercury, 60% arsenic, and 13% nitrogen oxide emissions. Additionally, coal emissions from power plants is the number one anthropogenic source of greenhouse gases worldwide. In the United States approximately 81% of all greenhouse gas emissions is due to coal-fired power plant emissions, primarily carbon dioxide and to a lesser extent nitrous oxides (U.S. Environmental Protection Agency 2018).

Below is a brief overview of the primary gaseous, particulate, and metal emissions from coal-fired power plants.

Particulate Matter

Particulate Matter size, concentration and chemical composition is primarily determined by the emission source (Pope and Dockery 2006). Up to 35% of particulate matter in some areas of the United States is due to coal-fired power plants (Gilmour et al. 2007). Increased ambient concentration of PM has been associated with increased all-cause mortality, cardiovascular mortality, pulmonary mortality, lung cancer mortality, incidence of stroke, heart attacks, increased total hospital admissions, congestive heart failure admissions, COPD and pneumonia related admissions, and decreased pulmonary function among children (Anderson et al. 2012).

*SO2 and NOx*

Coal-fired power plants are one of the largest contributors to sulfur and nitrogen oxide air pollution. Sulfur dioxide and sulfur trioxide (SO3) transform to sulfuric acid (H2SO4) as flue gas is cooled during the emission process. SO2 emissions is the dominant precursor of sulfuric acid (Daniel 2006). Sulfuric acid condensation is dependent on temperature, water vapor and sulfur content of coal (Srivastava et al. 2004). Sulfuric acid emissions from power plants have been associated with pathological changes in ciliary clearance and bronchoconstriction in asthmatics (Pietropaoli et al. 2004). Increase in ambient SO2 concentration has been associated with increased hospital admissions for COPD, MI, and stroke related hospital admissions (Shah et al. 2015), as well as total and cardiorespiratory mortality (Wang et al. 2018). Increase in ambient NOx concentration has been associated with increased total respiratory and cardiovascular mortality (Faustini et al. 2014); COPD and MI mortality (Zallaghi et al. 2014); and incidence of lung cancer (Hamra et al. 2015)

Polyaromatic Hydrocarbons (PAH)

Polyaromatic hydrocarbons are organic pollutants which result from incomplete combustion of fossil fuels (Mastral and Callen 2000). Coal-fired power plants are a significant source of PAHs (Wang et al. 2010). PAH exposure among children is associated with developmental delay, behavioral problems and decreased IQ (Perera et al. 2008, 2012; Edwards et al. 2010). In addition to neurotoxic properties, PAH exposure induces oxidative stress associated with increased atherosclerosis build up and incidence of cardiovascular disease (Ridker 2009). PAHs covalently bond with DNA causing PAH-DNA adducts, a key mechanism in the development of lung, breast, and bladder cancer (Rengarajan et al. 2015).

Metals

Mercury is a neurotoxin that has been associated with decreased IQ and increased incidence of cognitive delay among infants. Point sources of coal combustion including coal-fired power plants is the largest source of anthropogenic mercury emission worldwide (U.N. Environment Programme 2013). Vegetables and grain crops located in the proximity of coal-fired power plants have elevated mercury concentrations with concentrations negatively correlated with distance from coal-fired power plants (Li et al. 2017). Decreased productivity resulting from impaired cognition and decrease intelligence is estimated to have an economic cost of $8.7 billion annually in 2000 dollars. It is estimated that $1.3 billion (range: $51 million–$2.0 billion) of this cost is due to coal-fired power plant mercury emissions (Trasande et al. 2006a). Of the 1,566 excess cases of mental retardation annually among US children due to environmental mercury exposure, an estimated 231 cases are due to coal-fired power plant mercury emissions (Trasande et al. 2006b). In addition to mercury, a number of trace metals are released during coal combustion including As, Be, Cd, Co, Cr, F, Ni, Pb, Sb, Se, and U (Finkelman 1999). Source apportionment studies have attributed coal-fired plant emissions as the primary source of Ni, Se and V components of PM2.5 (Connell et al. 2006).

*Fluorine*

Fluorine is the lightest of the halogens and one of the most electronegative and reactive elements. Fluorine has been found at exceedingly high concentrations in soil neighboring coal mines (Ando et al. 1998). Health effects related to excess fluorine exposure includes osteosclerosis, severe bone deformation and mottling of tooth enamel. The majority of fluorine exposure from coal combustion is secondary to ingestion of foods cooked on coal stoves and not from coal-fired power plants.

Ionizing Radiation

In addition to the gaseous and particulate exposures listed above, coal-fired power plants also produce measurable ionizing radiation including radon, uranium, and radium. Ionizing radiation induced chromosomal aberrations, apoptosis, mutagenesis, and DNA damage and is listed as an IARC Group 1 Carcinogen (IARC 2012b). The extent of radiation emission from coal-fired power plants is largely dependent on the quality of coal used in the combustion process, specifically the content of uranium and thorium. The ash remnants following coal combustion is known as fly ash and has a 10-fold increase in radioactivity when compared to pre-combustion whole coal. Mishra (2004) measured external gamma radiation dose in the area surrounding a power plant was 50 nSv/h, while workers in the coal handling area were exposed to 103 nSv/h of gamma radiation. Concentrations at the “ash pond” where the post-combustion fly ash is contained was nearly 200 nSv/h. The environmental and occupational health risk from ionizing radiation produced from coal-fired power plants is heavily dependent on the method used to contain and store fly ash. Many countries have enforced legislation regulating what happens to coal-fired power plant waste such as fly ash following energy production.

**Methods**

## Search strategy and inclusion criteria

A systematic review of the epidemiological literature was conducted on PubMed, Web of Science and Toxline (unique from PubMed) platforms utilizing an identification, screening, eligibility, inclusion algorithm. Multiple search terms where used on all three platforms. Search restrictions included English language, years of publication 1998–2018 and human participants.

**[Table 1 near here]**



In order to widen the scope and to minimize the likelihood of missing relevant literature, an additional term “power plant” was used in addition to “coal-fired.” In total there were 2,152 unique search retrievals for each search term for all three databases. In addition to articles identified through the search platforms, citation review for the above listed terms added another 83 articles. Following the initial identification process, duplicate articles from different search terms were removed prior to starting the screening process. Figure 1 outlines a flow chart of the selection process adapted from the Preferred reporting items for systematic review and meta-analyses (PRISMA) group statement (Moher et al. 2015).

**[Figure 1 near here]**

**Eligibility process**

The abstract of the 879 articles that were included in the different search results listed above were reviewed and narrowed the pool of relevant articles based on a narrow set of criteria:

Original scientific articles (not review articles).

* Predatory journals were excluded based on guidelines published by Laine and Winker (2017)
* Coal-fired power plant was one of the primary sources of exposures assessed
* At least one primary dependent variable is a health outcome in children
* Only morbidity and mortality studies were included. Economic analysis and exposure assessment studies without a health outcome were not included

Following the screening for eligibility, 95 articles remained. Articles were included in the final critical review if statistical methods were based on accepted parameters of p < 0.05 and power of > 80% to minimize risk of type I or type II error; a systematic effort was made to control for potential confounders; and both bias and misclassification were assessed with attempts made to minimize its effects. A total of 34 were included in the final systematic review. A review protocol and article abstract instrument was developed and applied to all articles included in the final systematic review. Measures of effect (odds ratio, relative risk, hazard ratio, etc.) were tabulated and summarized in a structured format.

**Results of Systematic Review of the Epidemiological Literature**

Below we summarize the primary findings and limitations of the epidemiological literature of the health impact from coal-fired power plant emissions. Table 1 outlines the study population, exposure and outcome assessment methods, and primary effect measurement for all studies reviewed.

***Respiratory***

Children living in three communities near a major coal-fired power plant in Hadera, Israel had a significant rise in asthma and respiratory related conditions as well as reduced PFT in the decade after the plant become operational (Goren et al. 1997). Exposure was assigned by measuring defined “air pollution ‘events’ in which the half-hourly averages for SO2 and NOx were above an arbitrary threshold” as measured by 12 regional air pollution monitors. Asthma prevalence increased from approximately 8% prior to the plants opening to 13% a decade later. Wheezing and shortness of breath increased from approximately 11% of children to 16%. After controlling for potential confounders, a significant association was found for asthma (OR: 1.79 [95% CI: 1.16, 2.74]) and wheezing/shortness of breath (OR: 1.59 [95% CI: 1.11, 2.28]). The primary limitation in this study was the lack of available exposure data and crude method for assessing exposure. The “event method” of exposure assessment described in the article is prone to exposure misclassification.

Further cohort studies assessed respiratory outcomes among children living near the same coal-fired power plant in Hadera, Israel. Yogev-Baggio et al. (2010) estimated personal exposure to NOx and SO2 among 1181 children based on home address and compared it with respiratory health status and pulmonary function tests (Yogev-Baggio et al. 2010). The authors estimated exposure to "air pollution events" above an arbitrary concentration cut off as estimated from regional monitoring stations, identifying low, medium, and high exposure areas. Pulmonary function tests were lowest in the “high pollution” areas. Children in "high pollution" areas with report of daily respiratory symptoms had larger decreases in pulmonary function than children without respiratory symptoms. This suggests that children with chronic respiratory disease may be more susceptible to respiratory effects associated with living in areas with higher emissions from power plants. Dubnov et al. (2007) analyzed the same data for 1492 children residing near the Hadera, Israel power plant and found a significantly negative association between increasing exposure to NOx and SO2 with decreasing FEV1 and FVC.

A cross-sectional study of 2,244 adults living in the vicinity of the same power plant in Hadera, Israel compared respiratory symptoms and disease as reported on the European Community Health Survey with personal estimates of NOx and SO2 exposure originating from the coal-fired power plant using a novel "source" approach. The authors (Amster et al. 2014) found that chronic cough (OR 1.58; 1.19, 2.11) and chronic phlegm (OR 1.45; 1.06, 1.98) were significantly associated with 1 ppb increase in NOx attributed to power plant emissions. The authors did not find significant associations for SO2 power plant emissions and associations with asthma and COPD were not statistically significant. The benefit of the Amster et al. (2014) study is that it estimated power plant specific exposures based on emissions modeling and was able to see an increase risk of respiratory morbidity associated with those emissions.

Peled and colleagues (2005), conducted a time-series analysis comparing daily PM10 and PM2.5 concentrations with hospitalization and emergency room (ER) visits for children (0–3 years) residing near a major coal-fired power plant Ashkelon, Israel. Of the 3,600 ER visits and 1,134 hospital admissions, the highest rate of hospitalization was found in the city closest to the power plant with a significant positive correlation between PM2.5 concentration and both respiratory ER visits (p = 0.02) and hospitalization (p = 0.03). A survey of daily respiratory symptoms and peek expiratory flow (PEF) was conducted for 285 asthmatic children living near the same coal-fired power plant in Ashkelon, Israel. Maximum daily PM2.5 concentrations were inversely associated with PEF for asthmatic children living in Ashdod, just north of the power plant (coefficient = -2.74; p < 0.001). While the study did show elevated air pollution concentrations were associated with increased respiratory symptoms and decreased pulmonary function, the lack of exposure modeling estimating personal exposure limits the ability to attribute the observed adverse health effects to power plant emissions.

A study of 196 children from 26 villages in Thailand surrounding a coal-fired power plant assessed the association between daily SO2 concentrations and the incidence of respiratory symptoms (Aekplakorn et al. 2003) in a time-series analysis. No significant association was seen between daily concentrations and respiratory symptoms in either asthmatic or non-asthmatic children in the cohort. One of the primary limitations of the study was the lack of personal exposure estimates and relying on only three ambient air monitors across a large area with diverse exposure pathways. The authors included PM2.5 and PM10 concentration in two-pollutant models, however there was significant error in exposure data which may have biased the results toward the null.

Health and socio-economic data from 40,000 Indian households was compared in regions with coal-fired power plants and those without from 2005 to 2012 (Gupta and Spears 2017). The authors found a relative increase in self-report cough symptoms among residents in regions where coal-fired power production increased; there was no association seen with change in frequency of fever or diarrhea. The primary limitation of the study is that the authors did not utilize actual emissions or exposure monitoring data, rather their primary metric of exposures was to assess addition of new power plants to the grid in an ecological fashion. The authors looked as power plant construction and not actual operation, consequently it is unclear if actual emissions increased during the study period.

Karavus et al. (2002) compared respiratory symptoms and pulmonary function among “exposed” residents living within 5 km of a coal-fired power plant in Kutahya, Turkey with residents living in villages with similar demographic makeup greater than 30 km from the power plant. Residents in the “exposed” group reported significantly higher prevalence of chest tightness (46.2% vs. 28.0%) and chronic cough (29.2% vs. 20.4%). Spirometric means (FEV1 and FEF 25%–75%) were significantly lower among residents in the villages within 5 km of the power plant. The difference in spirometric values was not seen among smokers. The study was limited both in outcomes assessment, relying on retrospective subjective symptom report, and exposure assessment, relying on distance from power plant and lacking any model to estimate exposure from actual emissions.

***Cancer***

Collarile et al. (2017) investigated the risk of lung and bladder cancers among residents near a mixed coal-oil-fired power plant in Italy from 1995–2009. Power plant specific exposures to benzene, PM10, NO2 and SO2 were estimated based on point source emission reports. Yearly age-standardized incidence rates of both lung and bladder cancers were compared according to exposure tertile of each pollutant. Increasing exposure to benzene was significantly associated with increased risk of lung cancer among woman greater than 75 years of age (IRR: 1.86 [95% CI: 1.15, 3.00] intermediate tertile vs. lowest tertile; IRR: 2.00 [95% CI: 1.23, 3.25] highest vs. lowest tertile). Both NO2 and SO2 also showed significant increase in lung cancer risk (NO2- IRR: 1.70 [95% CI: 1.07, 2.71] intermediate vs. lowest tertile; IRR: 1.72 [95% CI: 1.07, 2.77] highest vs. lowest tertile; SO2- IRR: 1.55 [95% CI: 0.96, 2.49] intermediate vs. lowest tertile; IRR: 1.71, [95% CI: 1.07, 2.73] highest vs. lowest tertile). A linear relationship was also seen for bladder cancer among women in the greater than 75 age group for benzene (IRR: 2.39 [95% CI: 1.29, 4.44] intermediate vs. lowest tertile; IRR: 1.94 [CI 95% CI: 1.01, 3.74] highest vs. lowest tertile), and NO2 (IRR: 1.97 [95% CI: 1.07, 3.63] intermediate vs. lowest tertile; IRR: 1.94, [95% CI: 1.03, 3.65] for highest vs. lowest tertile). There was no significant associate seen for men or women under age 75 for either lung or bladder cancer.

The primary limit with the study is the accuracy of exposure assessment based on power plant emission reports. Additionally, a number of potential confounders such as smoking patterns and social economic status (SES) were not taken into account. Residents with higher power plant exposures may have greater exposure to other industrial sources of pollution or have higher exposure to other cancer risk factors such as smoking or decreased SES. The differential results between men and women is one of the studies novel findings, however a full exploration of effect modification by gender, the physiological causes and implications for policy, is lacking.

Tang et al. (2013) measured a number of heavy metals in 112 surface soil samples collected near a major coal-fired power plant in Huainan City, China. The authors found samples downwind from the power plant had significantly higher concentrations of heavy metals when compared to upwind. Based on the soil metal concentration, the authors estimated that hazard ratios and cancer risk from exposure to arsenic, cadmium, chrome, and nickel was significantly higher for residents living downwind from the power plant. The article provides a unique approach to modeling health risk based on direct soil measurements. They conclude that the higher average hazard quotient for communities downwind from the power plant could result in adverse health effects.

Researchers in India (Kumar et al. 2014) analyzed the concentration of PAHs in residential soils in an area surrounding a major coal-fired power plant. The measured concentrations and soil ingestion pathway were utilized to estimate lifetime average daily dose. Incremental lifetime cancer risk due to PAHs through soil ingestion was 3.1 × 10−7 for adults and 1.5 × 10−7 for children. The authors premised that PAHs in soil samples originated from petrogenic and mixed pyrogenic activities such as coal combustion based on composition profiles and molecular ratios of PAHs in soils. The primary limitation of this study is solely relying on ingestion pathway of soil and not including agricultural or water ingestion as well as inhalation and dermal exposure pathways.

Pesch et al. (2002) conducted a population-based case-control study to determine if increased incidence of non-melanoma skin carcinoma is associated with arsenic emissions from a coal-fired power plant in Slovakia. The exposure assessment was fairly complex, incorporating residential exposure based on atmospheric dispersion modeling of emissions, dietary consumption of high-arsenic foods, and occupational exposure based on industry specific exposure estimates and job exposure matrix. Participants residing in the same district as the power plant had significantly higher incidence of non-melanoma skin carcinoma than the national incidence (SIR 1.64: 95 CI 1.24, 2.17). After controlling for gender, age and occupational exposure, risk estimates in the highest exposure category versus the lowest (90th vs. 30th percentile) were 1.90 (95% CI: 1.39, 2.60). The researchers use of dispersion modeling based on actual emissions and estimates of competing dietary and occupational sources of arsenic exposure enabled them to control for potential co-exposures. The population was geographically stable with over half of the study population having occupied their current homes for over 30 years, limiting exposure misclassification by residence location.

Mokhtar et al. (2014) carried out a health risk assessment for air pollution from a coal-fired power plant in Malaysia based on AERMOD plume exposure model for SO2, arsenic, mercury, and chromium. The researchers found an excess lifetime cancer risk with short-term dispersion of arsenic (1.46 × 10-5) and chromium (1.2 × 10-4) emissions. The study relied on health risk assessment using estimates for hazard quotients and lifetime cancer risk.

***Neurodevelopment***

A number of studies looked at the neurodevelopmental impact of PAH exposure from coal-fired power plant emission from the Tongliang power plant in China. China is an ideal location for the study of coal related health effects as 75% of power generation in China is reliant on coal, and coal generated power is a major source of PAHs and metals in many regions of China. Coal generated power production also accounts for nearly 50% of total SO2 emissions, 27% of NOx and 11% of PM10 emissions in China (Hao et al. 2007).

The Tongliang power plant provided a unique natural experiment as it operated seasonally until it was completely closed in 2004. Tang et al. (2006) found that prior to the power plants closure in 2004 maternal PAH-DNA adduct levels above the median adduct level was associated with decreased birth head circumference (p = 0.057) and significantly (p < 0.05) reduced children’s weight at 18 months, 24 months, and 30 months. The study however did not directly estimate the contribution of coal-fired power plant PAH emissions. Following the power plant closure, Tang et al. (2008) prospectively followed the same cohort of 133 maternal-infant pairs who lived within 2.5 km of the power plant prior to its closure. The researchers measured PAH-DNA adducts, lead and mercury in umbilical cord blood and subsequently assessed neurodevelopment at age 2 using the Gesell Developmental Schedules. The authors reported an inverse association between cord PAH-DNA adduct and motor, language, and total average developmental quotients (DQ). Cord lead was inversely associated with social and average DQ while a 0.1 increase in cord adduct associated with increased developmental delay (OR = 1.91; 95% CI, 1.22 to 2.97).

In a follow-up study Perera et al. (2008) assessed the neurodevelopmental benefits of reducing prenatal exposure to PAH following the closure of the Tongliang coal-fired power plant. The study included two identical prospective cohort studies both preceding (see Tang et al. 2006) and following the closure of a major coal-fired power plant in Tongliang, Chongqing, China. The authors reported that there were no longer significant associations between PAH-DNA adduct and developmental delay in the second post-closure cohort. The study concluded that “an intervention to eliminate emissions from a polluting coal-burning power plant was effective in improving developmental outcomes among children living” near a power plant. The authors reported that lower levels of PAH-DNA adduct, higher concentrations of mature BDNF protein and higher DQ scores were seen in the post power plant closure cohort when compared to the pre-closure cohort from 3 years earlier. Adduct concentrations were inversely associated with BDNF with motor, adaptive and average DQ. The exposed pre-closure cohort showed a statistically significant association between B[a]P-DNA adducts and decreased head circumference (Tang et al. 2014). This association disappeared in the post-closure cohort. Birth weight and height also increased in the post-closure cohort and were correlated with decreased PAH-DNA concentration (Tang et al. 2013).

The strengths of the studies of Perera and Tang are the prospective cohort design, use of molecular markers as a metric of physiologically significant internal dose, and adequate control for a number of potential confounders. The authors took advantage of a natural experiment to assess the reversal of effect on child neurodevelopment which followed the plant’s closure. The main limitation in these studies however is that the authors did not model power plant specific emissions. However, the assumption the majority of the PAH exposure was due to the power plant is reasonable since there was a 1.5–3.5 times increase in ambient PAH concentration during the plant’s seasonal operation. However, some proportion of the reported detrimental neurodevelopmental affect from PAH exposure was potentially due to other industrial or transportation sources.

***Mortality studies***

Li et al. (2017) assessed exposure to two metals, copper, and zinc, from coal combustion between 1995–2014 in China. Exposure was estimated by integrating coal combustion data with relevant emission factors across four different sectors. Power plant emissions were found to account for approximately 30% of ambient copper and zinc in China. The authors reported that regression models determined a positive correlation between metal emissions and mortality; 0.001% and 0.0002% increase in mortality with each ton increase in copper and zinc, respectively. The study’s primary limitation is that the authors do not detail the source of mortality data or other covariates included in their regression model. It is also not clear if the authors incorporated baseline mortality trends or controlled for socio-economic confounders in their analysis.

Soil heavy metal concentrations were measured in the area surrounding a major coal-fired power plant in West Bengal, India and compared to background levels (George et al. 2015). Arsenic, mercury, cadmium, chromium, and cobalt concentrations in affected areas were 2.7, 2.3, 1.7, 1.6, 1.3 times greater than in control areas. Exposure risk assessment was based on estimated cumulative daily intake from various exposure pathways. The cumulative hazard index for all measured metals was less than 1.0, suggesting exposure is within threshold limits and does not pose a significant health risk.

Li and Gibson (2014) assessed the decline in SO2 and PM2.5 emissions from coal-fired power plants following the enactment of a 2002 legislation which required pollutant emission reduction in North Carolina, USA. The authors utilized a Bayesian Maximum Entropy method for air pollution modeling and then modeled cardiopulmonary and lung cancer mortality attributable to change in PM2.5 concentrations. A 20.3% decrease of SO2 and 8.7% decrease in PM2.5 emissions per year was found in the decade following the legislation enactment. Their risk models estimated that air quality improvements resulted in a 63% decrease of PM2.5 attributable overall death (1500–1800 deaths), and 61% decrease of cardiopulmonary related PM2.5 attributable deaths (910–1000 deaths). The primary limitations of the study are the assumptions incorporated into the health outcome model, specifically the choice of concentration-response (C-R) functions. The authors point out that there is uncertainty throughout the epidemiological and toxicology literature regarding the dose-response relationship between PM2.5 exposure and cause-specific mortality. The authors utilized C-R functions specific to PM2.5 sulfate, however the specific composition of particulate matter from coal-fired power plant emissions is different than other sources of PM with a steeper C-R function. Despite the limitations, the research suggests that coal-fired power plant emission reduction legislation is effective in reducing both gaseous and particulate emissions and PM attributable mortality.

Chen et al. (2017) estimated ambient exposure and internal dose to heavy metals and PAHs for 252 subjects who lived near a coal-fired power plant in Taiwan. Urine concentration of four oxidative stress biomarkers was also measured. Participants were categorized into “high” and “low” exposure groups based on distance of residence to the power plant. Estimates of vanadium, and three PAHs (pyrene, fluoranthene, and dibenzo[a,h]anthracene) concentrations were significantly higher at the point of residence for “high exposure” subjects when compared to “low exposure” subjects. Measured urine concentrations of 1-OHP, vanadium, nickel, copper, arsenic, strontium, cadmium, mercury, and thallium, as well as all four urine oxidative stress biomarkers were increased for “high exposure” subjects when compared to “low exposure” subjects. The primary limitation of this study was the attribution of “low” and “high” exposure groups based solely on distance of residence from the power plant. The “high exposure” group lived in the three townships closest to the power plant, however the designation is arbitrary and not based on a plum analysis or environmental pathway modeling. This introduces considerable exposure misclassification that most likely biases the authors’ results.

Caiazzo et al. (2013) estimated the mortality associated with emissions from combustion of fossil fuels across different sectors in the United States. Based on the modeled exposure estimates from the 2005 EPA National Emissions Inventory, the authors estimated the mortality attributable risk from exposure to PM2.5 and ozone across different sectors. They estimated that power generation throughout the United States causes ~52,000 (90% CI: 23,000, 94,000) PM2.5-related and ~2000 (90% CI: 300, 4,000) ozone-related premature deaths per year. The authors estimates are based entirely on EPA emissions inventory and model assumptions of pollutant specific attributable risk. The study also does not assess the specific contribution of coal-fired power plants. The article does however highlight the extent to which policy measures could be undertaken in order to mitigate the impact of specific emissions from different sectors.

Ruiz-Rudolph et al. (2016), compared cancer, respiratory, and cardiovascular mortality and hospitalization rates with presence of power plant in Chile. Their study did not distinguish between coal and oil powered power plants and did not use GIS based estimates of ambient concentrations. Instead they compared rates of disease among different geographical areas with and without power stations in an ecological fashion. They reported a statistically significant increase risk of total mortality for both men (OR: 1.42 [95% CI: 1.16, 1.73]) and women (OR: 1.19 [95% CI: 1.02, 1.37]) as well as cancer mortality in men (OR: 1.94 [95% CI: 1.36, 2.74]) and women (OR: 1.25 [95% CI: 1.04, 1.50]). Hospitalization rates were significantly increased, as well as cardiovascular (men OR: 1.72 [95% CI: 1.12, 2.67]; women OR: 1.71 [95% CI: 1.16, 2.59]), respiratory (men OR: 1.72 [95% CI: 1.05, 2.80]; women OR: 2.07 [95% CI: 1.33, 3.19]), cancer (men OR: 1.78 [95% CI: 1.23, 2.59]; women OR: 1.78 [95% CI: 1.23, 2.59]) and pneumonia (men OR: 1.71 [95% CI: 1.05, 2.75]; women OR: 1.92 [95% CI: 1.16, 3.22]) in areas with power plants. The main limitation of this study is that it is ecological and is lacking actual exposure measurements or personal exposure estimates. Additionally, there is no source apportionment to differentiate between coal and oil plant emissions.

Gohlke et al. (2011) assessed the relationship between coal consumption in electricity production with life expectancy and infant mortality between 1965 to 2005 in 41 countries. The authors estimated total burden of disease attributable to environmental risk factors based on the WHO 2002 World Health Report and then compared it against per capita coal and electricity consumption using autoregressive time-series model. A GAINS model (Amann et al. 2008) was used to estimate coal-fired power plant emission, personal exposure to particulate matter and effect on life expectancy. The authors reported an average of 3.5 years life lost from coal-fired power plant emissions of particulate matter in China and 0.5 for the European Union.

***Fluorosis***

The phenomenon known as “coal-fired-pollution-induced fluorosis” is potentially due to increase burning of fluoride rich coal and the subsequent deposition in soil as well as locally grown produce (Zhang and Cao 1996) causing dental fluorosis. In order to further investigate this hypothesis, Wang et al. (2013) measured the concentration of 11 metals in soil near a coal-fired power plant in Chongqing, China. Additionally, whole blood concentration of five metals and urinary fluoride levels among fluorosis cases and controls were measured. Ni, I, F, Hg concentrations and soil pH values were positively correlated with fluorosis prevalence. Cu, Zn, Mg, and Fe levels of the children with fluorosis were lower and urine fluoride levels was higher when compared to children without fluorosis. This suggests that children living near coal-fired power plants in China have higher risk of fluorosis due to increased internal dose of fluoride while lacking exposure to some anti-fluoride elements.

***Birth outcomes***

Ha et al. (2015) assessed the association between distance of residence to coal-fired power plants and adverse birth outcomes for 423,719 births from 2004 to 2005 in Florida, USA. The authors also assessed prenatal exposure to PM2.5 based on US EPA Hierarchical Bayesian Prediction Model utilizing air monitor and national emission inventory data. Prenatal exposure to PM2.5 was significantly higher for children in close proximity to coal power plants (10.7 µg/m3) when compared to natural gas (9.5 µg/m3) and nuclear power plants (7.7 µg/m3). Infants born within 20 km to more than one coal-fired power plant had significantly higher odds of low birth weight (OR: 1.12 [95% CI: 1.03, 1.22]), preterm delivery (OR: 1.20 [95% CI: 1.14, 1.25]), and very preterm delivery (OR: 1.23 [95% CI: 1.10, 1.36]). Significant associations were not seen with residential proximity to non-coal-fired power plants. This study looked only at residential proximity and does not estimate actual personal exposure to emissions. Although the study did not account for potential residential mobility during pregnancy, it did meaningfully control for socio-economic confounders.

Yang et al. (2017) assessed birth weight among all children born within 30 miles of a major US coal-fired power plant in Pennsylvania between 1990 to 2006. They reported that mothers living within four counties designated as “downwind” from the power plant during the last month of pregnancy were 0.4% to 6.5% more likely to have a lower birth weight infant, while very low birth weight (VLBW) was 0.19% to 17.12% more likely. The study is subject to exposure misclassification due to the relatively broad exposure assessment based on wind direction and proximity to the power plant.

Mohorovic (2003) took advantage of a natural experiment when a large coal-fired power plant in Labin, Croatia, temporarily went offline for 6 months. The researcher tested methemoglobin levels as a biomarker of oxidative stress among pregnant woman three times during power plant operation and three times during off season. Ground-level SO2 concentrations were also compared during the two study periods. A positive correlation was found between methemoglobin concentration and daily SO2 concentration while the power plant was operational. There was a gradual decline in methemoglobin concentration during the period when the power plant was un-operational (r = -0.60, p = 0.05); after the plant restarted operation the average methemoglobin concentration increased (r = 0.73, p = 0.01).

A follow-up study (Mohorovic et al. 2010) of the same cohort of pregnant women reported that frequencies of stillbirth and miscarriage was 60% lower during the “control” period when the power plant was un-operational (p = 0.03). The researchers hypothesize that emissions from the coal-fired power plant causes hemoglobin oxidation resulting in elevated methemoglobin throughout the pregnancy, thereby resulting in fetal hypoxia and ultimately sudden fetal death.

***Source apportionment studies***

A number of studies modeled source apportionment of power plant exposures and estimated excess burden of disease associated with power plant emissions. Levy et al. (2009) estimated the costs associated with mortality from exposure to PM2.5 for 407 coal-fired power plants in the United States. The authors relied on publicly available databases for PM2.5, SO2 and NOx emissions data and utilized a source-receptor matrix to estimate increase in ambient concentrations. Mortality data from CDC WONDER database was collected using the average mortality rate from 1999 to 2003. Cost of mortality associated with coal-fired power plant specific emissions was estimated based on statistical life approach. The authors estimated damages across plants ranged from $30,000 to $500,000 per ton of PM2.5, $6,000 to $50,000 per ton of SO2, $500 to $15,000 per ton of NOx, and $0.02 to $1.57 per kilowatt-hour of electricity generated. Estimates were based on a concentration-response function from a single epidemiological study; this introduced a significant source of uncertainty in their model. This research, while demonstrating the potential significant cost of coal-fired power plant related mortality, also showed the substantial variability in estimating health-related damages, primarily due to the wide range of emission control equipment, coal sulfur content, and combustion efficiency across coal-fired power plants.

Lueken et al. (2016) estimated the potential human health savings associated with a hypothetical transition of all US coal-fired power plants to natural gas. Exposure assessment utilized the US Department of Energy Information Agency’s emission data; health assessment was based on Air Pollution Emissions Experiments and Policy model and Estimating Air pollution Social Impact Using Regression model. The researchers estimated that reduction in SO2 emissions of 90% and NOX emissions by more than 60% would result in a reduction of total national annual health damages by $20–$50 billion annually.

Flue gas desulfurization (FGD) is a process by which fly ash containing particulates and SO2 is removed through an absorbent material such as limestone (Srivastava and Jozewicz 2001). Since 1997 FGD equipment has been gradually installed at all 13 units of the Mae Moh coal-fired power plant in northern Thailand. By utilizing an impact pathway approach, Thanh and Lefevre (2001) studied the potential health benefits of reducing PM10 and SO2 emissions following the FGD installation. Installation of FGD units at the Mae Moh power plant resulted in a 98% reduction of SO2 and 60% reduction of PM emissions. The authors estimated that reduction of emissions in just one of the 300-MW coal-fired unites resulted in an annual decrease of 16 cases of acute mortality, 12 cases of respiratory and cardiac hospital admissions, and approximately 354,000 fewer days with acute respiratory symptoms annually. In monetary terms the beneﬁt was equivalent to US $18.2 million (1995 prices) annually. When extended across all 13 units the total direct health savings would be $159.25 million annually. The study only assessed decreased morbidity and mortality associated with decreased PM and SO2 emissions and did not estimate PAH and metal emission decrease and did not quantify cancer related health effects.

**Discussion**

There is substantial epidemiological evidence indicating emissions from coal-based power production negatively impacts the health of neighboring populations. Our review illustrates significant impact on respiratory, cancer and birth outcomes from gaseous, particulate, and metal emissions from coal-fired power plants. Despite the weight of the current epidemiological research presented, there is clearly a need for more and better research.

Overall there is considerable inconsistency among the literature on exposure assessment models. As such the results between studies are difficult to compare. The assumptions and parameters used in exposure models have significant impact on the studies outcomes. Amster et al. (2014) used two different exposure models in their analysis and reported very different outcomes in regression analysis for all health outcomes. The error introduced by exposure misclassification varied significantly among the studies reviewed. Consequently, internal validity varied greatly among the studies, primarily due to inconstancy in exposure assessment. Consistency in exposure assessment will greatly improve the state of the literature by increasing internal and external validity of the literature.

Future research should focus on improving exposure assessment models with an emphasis of source apportionment and GIS methods to isolate power plant specific emissions. The majority of studies reviewed did not assess power plant specific emissions. There are a number of co-exposures that coincide with emissions from power production including transportation and industrial sources of exposures. By not estimating power plant specific emissions, researchers are limited in their ability to assess impact of coal-based power generation on health. This also limits the ability to use the epidemiological literature in cost-benefit analyses when considering the cost of plant closure or transitioning to a cleaner form of electricity production. The studies which do however include emission-based estimates of personal exposure were able to show the contribution coal specific pollution has on morbidity and mortality worldwide.

The literature reviewed does not address the long-term health effects of greenhouse gas emissions. With the rising awareness and increasing body of evidence linking climate change to the public’s health, it is imperative to understand the contribution of greenhouse gas emission from coal-based power production on health.

The vast majority of studies reviewed either did not control for SES or did so inadequately. Communities located closest to coal-fired power plants are more likely to have lower SES. The presents a significant source of confounding as SES is highly associated with all of the outcomes investigated. SES also may be a significant effect modifier as poverty and decreased nutrition increases the detrimental effect environmental pollution has on health.

Multiple pollutant models were utilized in a few studies, however the vast majority of studies focused on single pollutant measurements and models. Considering the large number of pollutants attributed to coal production, the interaction toxicants have with one another is important to assess to fully understand the impact coal emissions has on public health.

Intervention studies such as those conducted following the closure of the Tongliang power plant in China and the conversion from coal-based to natural gas power production in Israel, provide a nice natural experiment to study the impact of reduced emissions following the elimination of coal combustion. These studies have a number of benefits. By using the same exposed base population residual confounding is limited. The cohort provides its own control for comparison pre and post intervention. This of course assumes no major migration or demographic change in the intervening period. This is only beneficial for more acute health outcomes as chronic conditions such as cancer rates are not likely to change significantly immediately following power plant closure or conversion. An additional benefit of intervention studies and natural experiments is that they help to strengthen a causal relationship between coal emissions and increased mortality and morbidity based on reversal of effect component of Hill’s (1965) criteria. Intervention studies are also useful in determining benefit of plant closure and conversion for cost-benefit analyses as demonstrated in the studies of Levy et al. (2009) and Lueken et al. (2016), the impact of which plays heavily on policy considerations.

**Conclusion**

In conclusion, there is a large and growing body of epidemiological evidence associating emissions from coal-fired power plants with increased morbidity and mortality worldwide. Significant improvement in child health, overall mortality and decreased health-related expenses is associated with closure of coal-fired power plants. There is however a need for improvement of exposure modeling and incorporating confounding by SES to produce high-quality literature assessing the impact of coal-fired power plant emissions on health. Future research should emphasize the impact of greenhouse gas emissions and global warming, focus on natural experiments following power plant closures, and emphasize the economic benefit and policy implications of ending dependence on coal-based energy production.

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### Disclosure of Interest

The authors report no conflict of interest.

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**Table 1.** Search terms and query results.

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Term** | **PubMed** | **Web Science** | **Toxline1** | **Unique results** |
| Coal-fired AND morbidity | 4 | 12 | 1 | 12 |
| Coal-fired AND mortality | 23 | 91 | 13 | 98 |
| Coal-fired AND health | 212 | 406 | 70 | 436 |
| Power plant AND morbidity | 28 | 34 | 19 | 43 |
| Power plant AND mortality | 108 | 265 | 65 | 287 |
| Power plant AND health | 928 | 1,190 | 519 | 1,276 |
| **Total** | 1,303 | 1,998 | 687 | **2,152** |

1 unique Toxline search without PubMed crossref

**Figure caption**

**Figure 1.** Flow chart of study selection, screening, and eligibility