The Centre for Environmental Rights (CER) commissioned report: The health effects associated with ambient pollution arising from the proposed Lephalale Coal and Power Project (LCPP) and critique of selected reports

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Contents

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Mandate

In inviting me to prepare this report, CER requested the following:

1. A summary of the health impacts from air emissions and ambient air quality as a result of the proposed Lephalale Coal Mine (LCM) as described in the project air quality impact assessment (AQIA) as well as the Centre for Research into Energy and Clean Air's (CREA) report.
2. A description of the expected impacts on human health due to emissions and ambient air quality levels reported in LCM's AQIA and CREA's report, commenting on increases, if any, in disease such as asthma, mortality rates, incidence of emergency room visits or hospital admissions, and any other relevant health outcomes.
3. An assessment of the AQIA's conclusions about impacts to human health.
4. Comments on what CREA's modelling and risk assessment means for people of various ages and health conditions in Limpopo;

Executive Summary

Coal-related activities of extraction, production and combustion release a range of pollutants into the environment. These include criteria pollutants, such as particulate matter (of 10, 2.5 and 1 $\mu g/m^3$ in diameter), oxides of nitrogen and sulphur dioxide. Metals, of which mercury and arsenic are of particular concern, and volatile organic compounds, including polycyclic aromatic hydrocarbons are additional pollutants of interest in coal exposure. During its extraction, haulage to dump/storage sites, crushing and grinding process and subsequent combustion for energy present health risks to workers and exposed communities.

The peer-reviewed scientific literature investigating health outcomes associated with these coal-related pollutants have presented convincing evidence for specific health outcomes, particularly respiratory and cardiovascular disorders, both acute and chronic, as well as birth outcomes due to antenatal exposures, cancer outcomes and increased mortality from variety of diseases. Concerns exist for other disorders such as neurocognitive disorders, and evidence is emerging for health outcomes such as autoimmune disorders and chronic kidney disease. To describe the latest scientific knowledge on health outcomes associated with coal-related pollutants, a review of the most recent literature is presented. In many instances, the evidence presented in meta-analysis conducted since 2020 strengthens the findings of individual epidemiological studies over the last few decades, presenting pooled estimates of risk that may have a more global application.

The communities at risk of exposure from the proposed Lephalale Coal Mine project and its associated power production in the surrounding areas of Limpopo are at substantial health risk based on their modelled levels of exposure. [For purposes of this report, the Lephalale Coal Mine and the associated plans for coal use, in either existing or new power plants, is collectively referred to as the Lephalale Coal and Power Production (LCPP) Project]. Unfortunately, the Air Quality Impact Assessment and the Community Health Impact Assessment contained in the Final Environmental Impact Assessment Report that was conducted for the latter project has several shortcomings. The key is the absence of providing any meaningful estimates of health risk or impacts on the affected communities, and the reliance on the South African National Air Quality Standards as a benchmark for health protection.

The report prepared by the Centre for Research on Energy and Clean Air (CREA) defines the weaknesses of the AQIA, and addresses these shortcomings. In so doing, the CREA Report provides modelled estimates of exposure for the key pollutants and mercury, using best available scientific methods. These estimates are substantially higher that than proposed by the AQIA. Using established and well validated concentration-response functions from the scientific literature, and the modelled estimates of exposure, CREA was able to quantify expected morbidity and mortality for these outcomes. These assessments present a serious concern for the health of the exposed communities. The economic losses associated with these health outcomes were calculated, raising concerns about cost-benefit relationships for this project.

The identification of this substantial burden of “unfunded liability” (US$33 600 million) is a greater concern. These costs are externalised by the project owners to other stakeholders, particularly government and the communities themselves. In most instances, over the period of the project, it is the affected community, particularly households experiencing ill-health, that carry these costs.

Personal Background and Experience

I am a medical doctor, graduating in 1987 from the University of Natal, and a specialist in occupational medicine. I obtained my Masters in Public Health (Occupational Medicine) and PhD in Industrial Health from the University of Michigan in 2002. I have been employed in joint academic/clinical service posts at the University of KwaZulu-Natal and KZN Department of Health since 1994. I am responsible for the only public sector specialist clinic in occupational and environmental medicine in KwaZulu-Natal.

Apart from my clinical responsibilities, I conduct research as an occupational and environmental epidemiologist. I conducted the first major epidemiological research among South African coalminers, which investigated their health effects as a result of coal dust exposure. I have been responsible as the lead Principal Investigator (PI) in several of the largest environmental epidemiology projects in South Africa. The first, the South Durban Health Study investigated the effects of air pollutants in the city of Durban on exposed communities. Since then I have been the PI on a birth cohort study, which selected pregnant women, followed them through pregnancy and birth, and subsequently followed the children. The objective of the study was to understand the effects of air pollution on the respiratory health of children, with exposures commencing in the antenatal period. The cohort is now in its 10^{th} year. I have also led major research projects in the Vaal Triangle and the Mpumalanga Highveld, with a particular focus on childhood respiratory health and air pollution.

In addition to the above, I have served on various technical committees and panels addressing the issue of air pollution, the most recent was the Technical Panel set up by the Department of Fisheries, Forestry and the Environment to review the Minimum Emission Standards, and consider issues related to the weakening of these standards, and the implication for health and the economy. I also serve as a medical advisor to the legal team in a class action suit brought on behalf of workers exposed to coal dust and who have experienced a range of coalmine dust related lung disease.

I have published over 120 peer-reviewed papers in occupational and environmental epidemiology, and have over 80 presentations of my research in national and international conferences.

Opencast/Surface Mining: Pollutant-related Processes and Activities

Opencast or surface mining, by its very nature, results in pollutant emissions into the environment, placing surrounding communities at risk for adverse outcomes associated with exposure. At the construction of a new opencast mine, the first task is the removal of the topsoil (overburden) to access the mineral deposit. The extent of the overburden is dependent on the depths of the coal layers. This influences the methods used to extract the coal. It includes the use of excavators, loaders, dumpers, conveyor belts etc. (Ghose and Majee, 2000). All of these processes result in the release of particulate matter. Coal extraction and processing includes excavation, size reduction of coal, waste material transportation, loading and unloading, stock piling, etc. (Ghose, 2007). According to the FEIAR Reports, the extraction method for LCM will be truck and shovel, power shovels or loaders to load coal onto haul trucks, and transport to the coal handling and processing/beneficiation plant (CHPP) for washing/ beneficiation. The latter is a facility at which the raw material is processed, including washing, crushing and grading into appropriately sized chunks for stockpiling and subsequent transportation (GESI, 2017). As a product, the combustion of coal is associated with the emission of additional pollutants. This is relevant to this report, as the proposed LCM operation includes a possible power plant (the LCPP), or the use of this local within Limpopo, a region already burdened with coal combustion from the existing power plants.

Emissions and Sources of Pollution from Opencast Mining and Combustion

Ambient pollution from opencast mines could be from point or fugitive sources. While point sources are from stationary exhaust stacks, line and mobile sources, fugitive sources are primarily the open, exposed areas and processes. These include exposed overburden soil and coal stockpiles exposed to dry and windy conditions, releasing suspended and transported particles (Ghose and Majee, 2000). Extractive processes, such as drilling, blasting and loading are central to dust production. Hauling truck dumping, cleaning, sifting and grinding further add to this. Up to 80% of total dust is generated through vehicle traffic on haul roads (Ghose, 2007). All of these activities are influenced by local factors including silt content, moisture, vehicle speed, size of loader, drop height, area of exposed surfaces, frequency of loading/unloading and precipitation days (Patra et al., 2016).

Although particulate matter (PM), especially PM_{10} and $PM_{2.5}$ are of primary concern with opencast mines, particularly during the extraction process, these are not the only pollutants that are associated with coal dust exposure. The coal itself, its rank and silica content has important health consequences for exposed workers, and associated with a variety of pneumoconiosis and chronic obstructive pulmonary disease (grouped as “coal mine dust lung disease”). During the burning of coal, as a source of energy, other associated pollutants are of concern from an environmental perspective. This includes sulphur dioxide, nitrogen oxides and a range of heavy metals. Geographic areas and coal seam affect the heavy metal concentration of coal. This becomes a hazard during the mining, grinding, dumping of waste and most critically during combustion of coal (GEM, 2022). Metals released with potential health consequence include lead, mercury, nickel, cadmium and arsenic (Roy et al., 2016). Coal also contains small amounts of radioactive substances, thorium, strontium and uranium, which is more important after combustion. The “fly ash” combustion product of coal reportedly contains uranium and thorium at levels higher than in non-combusted coal (Deonarine et al., 2023). The largest contributor (81%) to mercury emissions in South Africa is coal combustion (Pirrone et al., 2010). The content of heavy metals in SA coal is reported in USGS World Coal Quality Inventory, with rates as high as 4.8ppm for arsenic; 713 ppm for barium; 98ppm for chrome; 0.83ppm for mercury; 22ppm for lead; 2.7ppm for selenium; 4.66 ppm for uranium and 93.4 ppm for vanadium.

Another important group of pollutants emitted from coalfired power-plants are polycyclic aromatic hydrocarbons (PAH). PAH's 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 (Amster, 2021). PAH concentrations in the soils around three South African coalfired power plants (Matla, Lethabo, and Rooiwal) ranged from 9.73 to 61.24 µg g^{-1}. The US Agency for Toxic Substances and Disease Registry recognises a standard of 1.0 µg g^{-1} for significantly contaminated site (Okedeyi et al, 2013).

Although not a focus of this report, coal production impacts not just air and soil, but water sources. During the coal washing processes, coal sludge is generated, and may be disposed of in specially created areas near coal mines. Depending on the disposal strategies, of the presence of these metals and other toxins in the sludge, water sources, both surface and underground could possibly become contaminated (GEM, 2022). Acid mine drainage, a major problem associated with disused gold mines in Gauteng, as well as South African coal mines (Coetser and McGeorge, 2020)) is as a result of pyrite-containing rock being exposed to air and water after mining, forming sulphuric acid and iron containing waterflows (USGS, undated).

Health Impacts from air emissions and ambient air quality as a result of the LCPP

CER Mandate: *A summary of the health impacts from air emissions and ambient air quality as a result of the LCM as described in the project air quality impact assessment (AQIA) as well as CREA's report.*

The Air Quality Impact Assessment (AQIA) (GESI, 2017) focused its findings on particulate and other criteria pollutants (oxides of nitrogen and sulphur dioxide) only, while the CREA report importantly, included mercury emissions. This review of associated health impacts is based on these pollutants specifically, as per the CER mandate, but also considers other pollutants associated with coal production and combustion. This review focuses on the epidemiological findings presented in published peer-reviewed scientific literature.

[NOTE ON INTERPRETATION OF ESTIMATES OF RISK: The information presented below uses the data as presented in the scientific publications particularly to quantify the effect. However, estimates of risk can be simply interpreted as a % of risk. For example, a Hazard Ratio or Odds Ratio of 1.2 implies that there is a 20% greater risk, a ratio of 0.7 implies a 30% lower risk and 1.0, no different risk from the comparison group**]**

Over the last 30-40 years, the evidence for ambient pollutant related adverse health outcomes has accumulated, with the seminal papers in 1989 (Pope et al., 1989) and the Six Cities Study in 1993 (Dockery et al., 1993). There is little doubt that pollutants associated with coal production and combustion increase the risk for cardiorespiratory disorders, including acute and chronic effects, as well as cancer risk and increased mortality. Data from the Global Burden of Diseases estimates suggest that global deaths attributable to $PM_{2.5}$ rose from 3.5m (95% confidence intervals (CI): 3m-4m) in 1990 to 4.2m (95% CI: 3.7m - 4.8m) by 2015 (GBD 2015, 2016).

Estimates for South Africa indicate that in 2000 15 619 (95% uncertainty interval (UI): 8 958-21 849) deaths were attributable to $PM_{2.5}$ exposure, with 19 672 (95% UI: 11 526 – 27 086) and 19 507 (95% UI: 11 318 – 27 111) in 2006 and 2012 respectively (Roomaney et al., 2022).

In 2018, the Lancet Commission on Pollution and Health classified the pollution-disease "pollutome" into three zones: established relationships with robust effect estimates (Zone 1); emerging but not quantified health effects of known pollutants (Zone 2) and inadequately

characterized effects of emerging pollutants (Landrigan et al., 2018). The report states that "causal associations are seen between $PM_{2.5}$ and cardiovascular and pulmonary disease" (Landrigan et al., 2018). Because of the extensive body of literature over the past three decades on some of these associations, this report largely focuses on findings that have emerged in the last 3-4 years.

For the purposes of this report, recognizing the strong cases made for these "Zone 1" disorders, a summary of estimates of dose-responses is provided where these are available from meta-analytic studies, as opposed to report estimates from individual epidemiological studies. The value of meta-analytic studies is the systematic approach in selecting multiple studies, and providing a grouped estimate of effect across multiple studies for specific pollutants and diseases. Of note, since the Lancet Commission report, there have been many meta-analyses conducted and reported in the peer-reviewed literature not just on Zone 1, but also of Zone 2. This is critical to document in a report of this nature, to ensure that the broader adverse health outcomes likely to be experienced by the exposed LCPP communities, but not adequately characterized in their contexts, are not ignored.

Pollutant-adverse health outcome relationships are complex both at an individual and population level: understanding the nature of the pollutant and the character of the exposure, and the pathogenesis of the disease is essential. Ambient pollutants impact across their concentration spectrum (from low exposure to high exposure), from steady state exposure through to peak exposures, single pollutant exposures to multiple exposures. Similarly, health effects are seen at a bio-molecular level (genetic and epigenetic (intra-generational) changes) (for example tests looking at biomarkers), subclinical (abnormal lung function tests in asymptomatic individuals or IQ tests below population average in otherwise normal individuals), acute presentation of symptoms (acute wheezing, myocardial infarcts (heart attacks), strokes, emergency department visits), chronic disorders (asthma, chronic obstructive pulmonary disease), cancers and death. Epidemiologists, in attempting to understand these complexities design studies that adjust for these variations, recognising that both exposure and health outcomes exist across a continuum in individuals and communities.

Respiratory Outcomes

Respiratory outcomes associated with the pollutants of concern, include new cases of asthma (incidence cases); presentation of severe respiratory symptoms requiring emergency room visits; chronic obstructive pulmonary disease (COPD), pneumonia and lung cancer.

The risk (odds ratio) for new cases of asthma was estimated in a meta-analysis to be 1.07 (95% CI 1.02 to 1.13) per 10 $\mu g/m^3$ increase in NO_2, and 1.16 (95% CI 0.98 to 1.37) per 10 $\mu g/m^3$.increase in $PM_{2.5}$ (Anderson et al., 2013). Based on these risk estimates, 16 million (95% CI: 9 to 19 million) new paediatric asthma cases are likely to occur globally each year due to $PM_{2.5}$ (Anenberg et al., 2018). A meta-analysis across 21 studies including in excess of 4 million participants, indicated that for every 10 $\mu g/m^3$ increase in $PM_{2.5}$, there was 1% increase (95%CI: 0.5–1.5) and 0.4% (95%CI: 0.2–0.6) for PM_{10} in hospital admissions or emergency room visits for pneumonia (Yee et al., 2021). Similar meta-analytic findings were reported for other emergency visits for respiratory symptoms (Wang et al, 2021) and non-specific respiratory disorders (Li et al., 2021).

The impacts on lung function are important, particularly on children, because these effects may not present clinically at the time of assessment, but ongoing exposure could result in COPD as the child ages. In one of the two first meta-analyses in 2022, a 10 $\mu g/m^3$ increase in $PM_{2.5}$, forced vital capacity (FVC), forced expiratory volume in the first second (FEV_1) and peak expiratory flow (PEF) decreased by 21.39 ml (95% CI: 13.87-28.92), 25.66 ml (95% CI: 14.85-36.47) and 1.76 L/min (95% CI: 1.04-2.49), respectively. These effects were also noted as a lagged effect – the preceding day's (or preceding few days) exposure caused similar decreases (Zhang et al., 2022). The other study focused on PM_1, and found for every short-term 10 $\mu g/m^3$ increase, FVC and FEV_1

decreased by 31.82ml (95% CI: 20.18, 43.45) and 32.28 ml (95% CI: 16.73, 48.91) respectively. Long term averaged exposure resulted in similar significant decrease in lung function (Zong et al., 2022)

The timing of exposure is an important predictor of longer term disease. In a meta-analysis of prenatal exposure, $PM_{2.5}$ significantly increased the risk of childhood asthma and wheezing (OR = 1.06, 95% CI 1.02-1.11; per 5 μg/m^3). (Yan et al., 2020)

Oxides of nitrogen, a key pollutant associated with coal combustion, is associated with a variety of respiratory outcomes, including decreases in lung function, increased presentations to emergency departments, increased symptoms of wheeze and exacerbation of asthma (US EPA, 2018).

Cardiovascular Outcomes

According to the Lancet Commission classification, the pollutant-related effects associated with the heart and blood vessels are “Zone 1”. These conditions include myocardial infarction (heart attacks), hypertension, abnormal heart rhythms (arrhythmias) and a failing heart (congestive heart failure) (Landrigan et al., 2018). These findings have been strengthened through new meta-analyses conducted after the Commission Report.

In one meta-analysis, involving 84 cohorts with more than 28m participants in total, the hazard ratio (HR) for cardiovascular disease generally was 1.10 (95% CI: 1.02-1.19) for a 10μg/m^3 increase in $PM_{2.5}$.This pattern was retained for specific cardiovascular disorders: acute coronary events (HR 1.15 (1.12, 1.17)), stroke (HR 1.13 (1.06, 1.19)), and hypertension (HR 1.07 (1.01, 1.14)). Cardiovascular mortality risk was similarly increased. PM_{10} and NO_2 showed increases in the similar range for these various outcomes. NO_2 was also associated atrial fibrillation (HR 1.01 (1.01, 1.02)), an irregular, rapid beating heart which requires urgent medical intervention (Pranata et al., 2020). In another meta-analysis, the relative risk of myocardial infarction (a type of acute coronary event) was 1.02 (95% CI 1.01-1.03) for every 10μg/m^3 increase in short term exposure to $PM_{2.5}$ (Farhadi et al., 2020). A subsequent meta-analysis (Zhu et al., 2021), that included 7.2m participants, considered long-term exposure. In this report, for every 10 μg/m^3 increase in $PM_{2.5}$, there was a 10% increase in myocardial infarction (HR = 1.10, 95% CI: 1.02-1.18) (Zhu et al., 2021).

A recent report pooling together data from multiple cohort studies in Europe (Effects of Low-Level Air Pollution: A Study in Europe (ELAPSE)), brought together data for 137 148 participants, provides new insights about cardiovascular outcomes and particulate and oxides of nitrogen exposure. Stroke risk was increased with both $PM_{2.5}$ (HR=1·10 (95% CI 1·01-1·21) per 5 μg/m^3 increase) and NO_2 (HR=1·08 (1·04-1·12) per 10 μg/m^3 increase). This study is particularly important because of its findings at levels below the EU standards of 25 μg/m^3 for $PM_{2.5}$ and 40 μg/m^3 for NO_2 (in 2010), and because in their analysis, they could not identify levels below which no risk was present (Wolf et al., 2021).

Maternal and Adverse Birth Outcomes

The impacts of coal-related pollutants on newborns and growing infants is particularly concerning. Antenatal exposures result in a range of adverse pregnancy effects and birth outcomes. These outcomes in themselves become risk factors for adverse health as the child grows into adulthood (Johnson et al., 2021). Two meta-analysis separately report an increased risk of pre-eclampsia (a condition associated with elevated blood pressure during pregnancy, which results in foetal risks) due to $PM_{2.5}$ and NO_2, (OR=1.31 (95% CI 1.14–1.50) per 5 μg/m^3 and OR = 1.31 (95% CI 1.07–1.61) per 10 μg/m^3 increase in $PM_{2.5}$ respectively and OR=1.07 (95% CI 1.02–1.13) per 10 mg/m^3 increase in NO_2 (Pedersen et al., 2014; Sun et al., 2020). Adverse birth outcomes such as low birth

weight (LBW), small for gestational age (SGA) and preterm birth (PTB), stillbirths and spontaneous abortion have all been associated with coal-related pollutants. In a pooled analysis of over 700 000 pregnancies across six studies, relative risks of 1.20 (95%CI: 1.01–1.40) and 1.09 (95%CI: 1.02–1.15) were reported for $PM_{2.5}$ and PM_{10} associated spontaneous abortions, respectively (Zhu et al., 2022). For each 10 μg/m^3 increase in ambient $PM_{2.5}$, there was a 22g lower birth weight (95% CI: 12 – 32g), an increased risk of LBW (OR=1.11 95% UI: 1.07-1.16) and an increased risk of PTB (OR=1.12, 95% UI: 1.06-1.19) (Ghosh et al., 2021). In another meta-analysis, exposures during different trimesters in pregnancy varied the risk for LBW across different pollutants. A statistically meaningful pooled estimate was seen for the entire pregnancy and in the first trimester for $PM_{2.5}$ (OR=1.06; (95%CI: 1.03-1.09) and OR=1.07 (95%: 1.00 – 1.15) respectively) (Hung et al., 2021). $PM_{2.5}$ significantly increased the risk of PTB and SGA during the exposure in entire pregnancy (RR = 1.09, 95% CI: 1.06-1.12) and RR = 1.101, (95% CI: 1.06-1.15), respectively (Ju et al., 2021).

Maternal exposures remain a concern, not just for specific birth outcomes, but also for health risks as the newborn ages. According to a detailed review, there is substantial evidence that exposure to $PM_{2.5}$ during the antenatal period is associated with a range of adverse health as the child ages. These include hypertension, obesity, metabolic diseases, including diabetes and non-alcoholic fatty liver disease. (Sun et al., 2022).

Neurological and Neurocognitive Outcomes

The impact of coal-related pollutants on the nervous system (the brain and the peripheral nerves), will probably be considered in “Zone 2” of the Lancet Commission. However, the evidence is increasing in well-designed epidemiological studies. Once again, the advantage of using evidence from meta-analyses as opposed to single studies, shows consistency of findings for specific outcomes and that findings are not specific to a particular sample.

In a recent comprehensive systematic review (Thompson et al., 2023), exposures of PM and oxides of nitrogen showed mixed evidence for specific neurocognitive function. In young children, there was limited evidence for general cognition; IQ, memory and learning and processing speed, while modest support for executive cognitive skills. In adults, there was moderate support for $PM_{2.5}$ and general cognition. Despite limited studies lending itself to a meta-analysis, among adults, a 1 μg/m^3 increase in NO_2 and $PM_{2.5}$ resulted in declines in performance on general cognitive battery scores (points lost = −0.02, (95 % CI: −0.04, −0.01) and −0.02, (95 % CI: −0.03, −0.00) respectively), while a 1μg/m^3 increase in $PM_{2.5}$ significantly reduced verbal fluency by−0.05 words (95 % CI:−0.08,−0.01, −0.02) and decrease in executive function tasks (95 % CI: −0.03 points, −0.01, $p < 0.001$) (Thompson et al., 2023). These findings are supported from an earlier review of studies, which have reported dose-response adverse outcomes with $PM_{2.5}$ (Weuve et al., 2021).

Although no meta-analysis has been presented on the outcome, over the past 10 years several studies on autistic spectral disorders have reported important increases in risk (odds ratios) due to $PM_{2.5}$ exposure, in a dose-related manner. In the most recent study, the increased odds ratios associated with an IQR increase for PM_1, $PM_{2.5}$ and PM_{10} were 1.86 (95%CI: 1.09-3.17), 1.78 (95%CI: 1.14- 2.76) and 1.68 (95%CI: 1.09-2.59), respectively (Chen et al., 2018). This supported an earlier study (Volk et al., 2013) which showed that nitrogen dioxide, $PM_{2.5}$ and PM_{10} were also associated with autism during gestation exposures (NO_2: OR= 1.81 (95% CI, 1.37-3.09); $PM_{2.5}$: OR= 2.08 (95%CI: 1.93-2.25); PM_{10}: OR= 2.17 (95% CI,1.49-3.16). The findings from both studies provide support that these estimates become larger when exposures in the early years of life are considered as compared to antenatally.

Increased pollutant related risks have been identified for other neurological outcomes, including attention deficient hyperactive disorder (ADHD), Parkinson’s, Alzheimer’s and epilepsy. No meta-analyses have been conducted for ADHD, but in a recent study of over 800 000 Danes, early life exposure to NO_2 and $PM_{2.5}$ was associated with 38% increase (95%CI: 35 to 42%) per 10 μg/m^3

increase in NO_2 and a 51% increase (95%CI: 41 to 62%) per 5 μg/m^3 increase in $PM_{2.5}$ (Thygesen et al., 2020). In a meta-analysis of over 200 000 patients with Parkinson's Disease, while increased risks were noted from the pooled data, these were not statistically significant (Han et al., 2020). In a meta-analysis of four cohort studies, a 10 μg/m^3 increase in exposure to $PM_{2.5}$ was associated with increased risk for dementia (HR=3.26 (95% CI: 1.20, 5.31) and Alzheimer's (HR=4.82 (95% CI: 2.28, 7.36) (Tsai et al., 2019). Epilepsy, with a global prevalence of 45.9m cases, is a leading chronic disease. Although a meta-analysis of 43 000 cases of childhood epilepsy in China, did not show meaningful associations with $PM_{2.5}$, two studies have reported short term lagged (previous day) effects, suggesting a short-term exposure association for this outcome (Cheng et al., 2022; Bao et al., 2019)

In addition to this evidence for the association between particulates and oxides of nitrogen and neurological and neurocognitive effects, associations of these outcomes with other coal-related pollutants such as mercury, volatile organic compounds and polycyclic aromatic hydrocarbons are well established. Deposition of mercury in soil and water sources are of the greatest concern. Consumption of contaminated fish becomes an important source of biological entry. Prenatal exposure through maternal consumption results in exposure to the foetus, and consequently resulting in neurodevelopmental compromise. This is quantified with reduced performance on tests of neurologic function in children, including tests of cognitive development, attention and behaviour, and motor skills. (Axelrad et al., 2007). It is estimated that 12 million IQ points are lost each year globally due to mercury exposure (Zhang et al., 2021). Depressed verbal IQ index scores were increased three-fold in children with higher levels of PAH biomarkers evident in cord-blood at the time of their birth (Jedrychowski et al., 2015). In another birth cohort, maternal PAH exposure was associated with ADHD in children followed up to the age of nine (Perera et al., 2014).

Mortality and Cancer

Apart from its association with specific diseases, coal-related pollutants are directly associated with mortality endpoints. Several meta-analytic studies since 2020 have provided pooled estimates of risk for these pollutants. In a meta-analytic study of all Chinese research to date, excess risks per 10 μg/m^3 increase in short term $PM_{2.5}$ were 0.70% (95% CI: 0.37%, 1.03%), 0.59% (95% CI: 0.34%, 0.83%) and 0.31% (95% CI: 0.04%, 0.58%), for mortality related to cardiovascular diseases, stroke and ischaemic heart disease, respectively. This changed substantially when considering long term exposure, as a 10 μg/m^3 increase in long-term $PM_{2.5}$ exposure resulted in 15.1%, 11.9% and 21.0% increase in cardiovascular, stroke and lung cancer mortality (Luo et al., 2023). In a further meta-analysis specifically investigating short term exposures (one hour to seven days) for particulates and nitrogen dioxide, positive associations between PM_{10} and NO_2 and all-cause mortality, cardiovascular mortality, respiratory mortality and cerebrovascular mortality were reported (Orellano et al., 2020). In another major pollutant-mortality study, consisting of more than 28m participants from several cohorts in Europe (ELAPSE), significant positive associations between non-accidental mortality and key pollutants were seen. A 5 μg/m^3 increment in $PM_{2.5}$ and a 10 μg/m^3 increment in NO_2 was associated with a HR = 1·05 (95% CI 1·02-1·08) and HR=1·04 (1·02-1·07) respectively. Associations with cardiovascular mortality, non-malignant respiratory mortality and lung cancer mortality where all statistically significant, with estimates strongest for lung cancer. (Straffogia et al., 2022)

The International Agency for Research on Cancer has determined that airborne particulate matter and ambient air pollution are proven group 1 human carcinogens. A meta-analysis of the available studies showed that the $PM_{2.5}$ and PM_{10} meta-relative risks for lung cancer was 1.09 (95%CI: 1.04-1.14) and 1.08 (95%CI: 1.00-1.17) respectively (Hamra et al., 2014). The meta-analyses of North American and European studies have provided additional support for PM-related lung cancer (Turner et al., 2020). The latter study also identified a $PM_{2.5}$ risk for bladder cancer mortality (HR

=1.13; 95% CI: 1.03-1.23). An increased $PM_{2.5}$ risk for gastro-intestinal cancers were reported (RR = 1.12; 95% CI: 1.01- 1.24), with strongest evidence for liver and colorectal cancer (RR = 1.31; 95% CI: 1.07-1.56 and RR = 1.35; 95% CI: 1.08- 1.62 respectively) (Pritchett et al., 2022; Wu et al., 2022)

Emerging Evidence for Other Outcomes associated with Coal-related Pollutants

Recent meta-analytic studies and epidemiological research presents increasing evidence for PM and NO_2 exposure associated with kidney disease. A pooled estimate across four studies found that for each 10 $\mu g/m^3$ increase in $PM_{2.5}$ and PM_{10}, there was an increased risk for chronic kidney disease (RR=1.10 (95% CI: 1.00, 1.21) and RR=1.16 (95% CI 1.05, 1.29) respectively). Both these pollutants were associated with important declines in kidney function (Wu et al., 2020). The latter finding was supported in an epidemiological study of over 2.5m young adults in China, which found a 0.8% decline in kidney function per 10 $\mu g/m^3$ increment in $PM_{2.5}$ exposure (Rasking et al., 2022)

Another area of concern is the evidence suggesting impacts on the immune system. Biologically plausible mechanisms provide support that PM and NO_2 can cause disruption of the immune system resulting in disorders referred to as auto-immune and auto-inflammatory – essentially the body reacting against its own tissues, through triggering immune responses in the body. Oxides of nitrogen have been associated with systemic lupus erythematosus (SLE) and rheumatoid arthritis (Zhang et al., 2023), while short-term exposure to $PM_{2.5}$ have been associated with increased outpatient visits for osteoarthritis in China (Chen et al., 2021).

In keeping with the hypothesis of PM stimulating biological immune responses, a similar mechanism may be at play with the increased reports of atopic dermatitis, an inflammatory skin disorder, with pollutant exposure (Fadadu et al., 2023). Among 214 000 children in Chongqing, China, each 10 $\mu g/m^3$ increase in $PM_{2.5}$, PM_{10}, SO_2 and NO_2 was associated with 0.7%, 0.9%, 11% and 5.5% increase in atopic dermatitis outpatient visits (Luo et al., 2022), with similar findings reported from a study of 34 000 participants in Shangai, China (Ye et al., 2022).

Studies of Coal-affected communities

The preceding review of coal-pollutant related health effects was based on the specific pollutants known to be associated with coal production and combustion. However, there have been numerous studies that report health effects from communities living in close proximity to the coal-industry related activities (either mining or power generation). In a review of 21 studies of children living in proximity to coalfired power plants, adverse birth outcomes, lower IQ scores; mercury associated autism and lower lung function was reported (Cortes et al., 2018). In a systematic review, searching for coalfired or power plant and health outcomes, forty studies were identified over a twenty year period, 1998-2018. All of these epidemiological studies were conducted in communities living in close proximity to a coalfired power stations in Canada, Israel, Thailand, India, Turkey, Italy, China, Slovakia, Malaysia, United States, Chile and Croatia. The health outcomes for which these studies presented evidence, included respiratory, cardiovascular, cancer, neurocognitive, birth outcomes, increased mortality, dental fluorosis outcomes (Amster, 2021). For those studies that presented estimates of effect for the various outcomes, these were generally in the range described in the preceding review, showing consistency in estimates across different study types and jurisdictions.

Expected Human Health Impacts due to emissions and ambient air quality levels as assessed by LCM AQIA and by CREA

CER Mandate: *A description of the expected impacts on human health due to emissions and ambient air quality levels reported in LCM's AQIA and CREA's pending report. Please comment on increases, if any, in disease such as asthma, mortality rates, incidence of emergency room visits or hospital admissions, and any other relevant health outcomes.*

There are no published peer-reviewed epidemiological studies that describe coal and related exposure-outcome relationships in the Limpopo area, specifically, or in the areas of the South African coal industry (production and combustion), including Mpumalanga or KwaZulu-Natal provinces. The few available studies do not appropriately assess either outcome or quantify exposure to determine estimates of effect. Published environmental epidemiological studies have been conducted in limited areas in South Africa. This was identified in a recent review of air pollution and respiratory effects in sub-Saharan Africa (Glenn et al., 2021). Of the 14 studies that met the selection criteria for inclusion, nine were from South Africa, with seven of these focusing on Durban and two from Cape Town. Therefore, to understand the effects of the emissions and air quality impact from the LCPP, extrapolation from studies outside these coal industry areas have to be considered, borrowing from the extensive international literature.

Having outlined the risks to health in the preceding section, the challenge is to extrapolate this to the LCPP. It serves no purpose in basing health risk estimates on the emissions of the LCPP alone. It is necessary to consider collectively, all emissions from all sources experienced by the communities within the impact zone of the LCPP. The AQIA does not provide these estimates. CREA was able to determine the short term and annual collective exposure for these communities including existing levels (CREA Report, Table 5). The AQIA provides lower estimates of exposure than CREA, due to the methodological shortcomings identified by CREA.

The CREA Report (Tables 6 and 7) provides the mortality and morbidity impacts of the anticipated exposures, based on their models. Based on the estimates of risk provided in the preceding review, outcomes not calculated for the CREA report (eg. lung function declines; incidence of pneumonia; neurocognitive and neurodevelopmental effects; congestive cardiac failure, autoimmune disorders and other cancers etc), are likely to be present within the affected communities. For example, the estimate of a 0.4% increase in presentations of pneumonias per 10 $\mu g/m^3$ increase in $PM_{2.5}$ (Yee et al., 2022), at the exposure levels calculated for the communities affected by the LCPP, one will expect to see a substantial increase in cases due to $PM_{2.5}$ alone. For many conditions, there will be almost a silent escalation in adverse health, which will not present at health services: for example, loss in lung function only becomes clinically evident when losses of up to 20% is experienced. However, with an expected 25ml loss in forced expiratory volume in one second (FEV_1) for every 10 $\mu g/m^3$ increase in $PM_{2.5}$ (Zhang et al., 2022), (a volume at the lower end compared to the loss due to a one year increase in age), it takes several years before an individual presents clinically with symptoms. At the modelled levels of exposure in the LCPP, declines in lung function are likely to exceed these declines, resulting in clinical presentations a few years after exposure. Similarly, losses in IQ points, while likely to be substantial in the exposed communities, will not present clinically, and therefore likely to be undetected.

A recent report from the Health Effects Institute (HEI) provides a sobering insight into the use of effect estimates, and the emphasis on air quality standards (Brunekreef et al., 2021). Analysing the data from six European cohorts, with well validated robust exposure estimates, Brunekreef and colleagues reported on the morbidity and mortality effects of long term low dose exposure to $PM_{2.5}$, NO_2, O_3, CO and BC (the ELAPSE study). Reporting on findings from over 300 000 cohort participants and a further 28 million individuals from administrative datasets, $PM_{2.5}$, NO_2 and BC

were significantly associated with natural cause, respiratory, cardiovascular and diabetes mortality, as well as asthma incidence, stroke and COPD admissions. Almost all participants had residential exposures below the 2010 annual EU Air Quality Standards for $PM_{2.5}$ and NO_2 (25µg/m^3 and 40 µg/m^3 respectively), with more than 50 000 residents with levels below 12 µg/m^3 (the US EPA standard). The researchers describe a supralinear relationship between pollutant and outcome, implying that effects were greater at the lower levels of exposure, and that no threshold for no effect was observed, with effects being seen at $PM_{2.5}$ levels lower than 10 µg/m^3 (Brunekreef et al., 2021).

The HEI report brings into sharp focus the AQIA dependence on the use of the South African Air Quality Standards. While these may represent legal values, in the health risk assessment context they are of no value.

Based on the effect estimates reported in the literature, the predicted levels of exposure, the estimates of morbidity and mortality in the CREA Report, the exposed communities are likely to experience substantial ill-health. Most of the reported studies discussed in this report which showed a dose-response relationship, were studies with much lower levels of exposure than the LCPP region. This will imply that the communities within the LCPP range of exposure can expect a greater burden of disease than the reported literature. However, because for the majority of these outcomes (respiratory, cardiovascular, neurocognitive, birth outcomes, cancers etc), there is no single cause, attributing individual adverse outcomes to a coal-related pollutant is not possible, and therefore, not likely to be reported as such clinically. Thus, with the predicted increase in exposure levels in these communities, the burden of disease will increase, as detailed in the CREA Report.

In the existing low-socioeconomic context that this community finds itself, the added burden of managing household members with ill-health results in further marginalization. The economic costs set out by the CREA Report translates differently at a household level, where limited resources must be further distributed to cater for ill-health, and the subsequent cycle of impoverishment: frequent school absence leads to compromised post-school education, poor employability, employment instability and further financial resource challenges.

The lack of investment in social infrastructure and networks, such as schools and clinics capable of managing the new illnesses likely to present within the community, further compromises the general health of the community.

Assessment of the AQIA Conclusions on Human Health

CER Mandate: *Your assessment of the air quality impact assessment's conclusions about impacts to human health.*

The AQIA provides a generic and somewhat dated summary of the literature on health effects associated with the selected pollutants (PM, NO_2 and SO_2), however, fails to extrapolate its predicted levels of exposure (both for mitigated and unmitigated concentrations) to expected health outcomes likely to be experienced by the exposed communities around the LCPP project (AQIA, FEIAR Vol 3). The Community Health Impact Assessment (CHIA) report (CHIA, FEIAR Vol 4), also provides no input into the pollutant-related health risks, and instead refers the reader back to the unreported estimates in the AQIA. Thus FEIAR reports provide no quantifiable estimates on the health risks that can be anticipated in the affected and exposed communities.

Apart from the above flaw in the AQIA, there are additional shortcomings that must be pointed out, some of which have been raised in the CREA Report:

a) not considering exposures to metals, particularly mercury

b) not considering the total burden of exposure that the community will experience

c) in a similar vein, not considering the use of the coal in other nearby power plants, and the contribution of this to the health burden

d) a focus on Lephalale, but limited attention to communities that may be outside this jurisdiction, or those in small informal villages around the mine

e) persistent reference to the SA Air Quality Standards and exceedances thereof. These standards are considerably weaker than the World Health Organisation guidelines, with extensive international data to show that substantial health effects are evident at the SA NAAQS. The WHO provides detailed arguments about why it was necessary to move toward stricter standards based on the adverse health effects seen at the old guidelines (WHO, 2021). The ongoing use of the SA NAAQS calls into question whether the AQIA had health or legal compliance as its objective.

The CHIA, apart from its absence in quantifying health impacts associated with exposure from the LCPP, uses a non-health framework to assess the health impacts on the affected communities. In using the International Finance Corporation's (a member of the World Bank Group) framework, the report claims that the most important factors contributing to health impacts are "influx of job seekers …..increasing the demand on ….infrastructure….." and "….the introduction of infectious diseases….". In listing the health impacts, exposure to pollutants from the operation are included, but not discussed any further. All other impacts listed are externalized from the LCPP – focus on household vulnerability, social and health infrastructure and communicable diseases. This externalization approach then places the burden for redress on the public sector. In addition, much of the "potential health impacts" are superficially presented as a narrative, with little or no scientific basis for their conclusions, with no reference to any literature or reports to support their methods of findings. For example, this report uses the population distribution from 2009, but provides no indication of how this outdated data may influence its findings.

The CREA Modelling and Risk Assessment Findings

CER Mandate: *comment on what CREA's modelling and risk assessment means for people of various ages and health conditions in Limpopo*

The CREA Report addressed the important shortcomings that emerged in the AQIA and the CHIA. From a health perspective, the CREA Report considered key coal-related pollutants (particulates and other criteria pollutants and mercury). The report considered exposures likely to be experienced by the affected communities in its totality, not just the isolated emissions from the LCPP. The report provided predicted exposure concentrations for the various criteria pollutants and mercury, which allowed for comparison with international standards and international peer-reviewed literature. In addition, together with the use of well-established concentration response functions, the calculated exposure levels by CREA allowed for the determination of health outcomes. In all of the modelling and predictions, the CREA Report provides the scientific basis for the work, and argues assumptions made, where necessary.

The report did not consider other metals, such as arsenic, nor did it consider the volatile organic compounds and polycyclic aromatic hydrocarbons. The report also did not consider health effects identified in the preceding literature review (eg. lung function declines, neurocognitive effects, pneumonias, autoimmune diseases etc). I believe that the reasons for these omissions are because the data is not available in a form that allows for them to be integrated into available predictive models, or that it is impossible to assign a measurable endpoint or quantify an economic value – for example, while lung function declines over time will adversely affect health of the individual, until this becomes clinically significant (eg. presenting as chronic obstructive airways disease), there is no tangible "community" impact (eg. loss of work days, increased use of health services and economic costs). However, I believe that these omissions in the report are unlikely to have had any significant impacts on their final conclusions of health impacts in terms of mortality, disability adjusted life years or economic impacts that arose from these adverse health states. It is possible that because of these omissions, the estimates in the CREA Report are likely to be more conservative that the real health effects experienced.

Despite the invaluable information provided by the CREA Report in terms of mortality and morbidity for the affected communities, there is no understanding of who bears the extrapolated costs from these health impacts as reflected in Table 8. Clearly this is not a shortcoming of the report, as it is beyond the authors' remit to address a purely social and societal question. However, in the absence of allocating responsibility of these costs to specific parties, the owner/implementer of the proposed LCPP, redistributes these "unfunded liabilities" to external stakeholders. While the state may assume responsibility for some of these liabilities, a disproportionate amount cascades to the community and households within these communities. In those households that are affected by the exposures (for example, homes with newborns with adverse birth outcomes, asthmatic children and adults, immunocompromised individuals, or those with severe chronic disorders etc), unbudgeted expenses emerge: transport of ill individuals to and from health facilities; cost of health services utilisation and medication; loss of income through sickness absence or family responsibility leave; etc. Longer term costs then escalate, once again borne by affected households directly: reduced available income within the household impacts on the wellbeing of the healthier children in the household, in terms of diets, school performance etc; poor school performance within the affected household influences post-secondary education and subsequent employability and income.

Thus, the very large financial costs calculated in the CREA Report must be understood in its social context, and this unfunded liability clearly accounted for in terms of the Risk Assessment conducted by the project implementer.

Conclusion

In summary, there is overwhelming scientific evidence for adverse health outcomes due to coal-related pollutants. These are mostly dose-dependent. The communities within the LCPP area are likely to be exposed to substantial levels of exposure, consistently above international benchmarks or levels known to cause ill-health. The AQIA and the CHIA provides no estimates of the burden of ill-health likely to be experienced by these communities. The CREA Report clearly indicates the high levels of mortality and morbidity that will be experienced in these areas. In calculating the economic burden of this ill-health as a result of the exposure, the CREA Report presents a disturbing picture. The challenge is, in proceeding with a project that is, without doubt, going to result in ill-health, how will this “unfunded liability” be managed by the relevant stakeholders. Marginalised communities in the LCPP region should not be expected to carry these costs.

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