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Attention: Bryan Beudeker

Peer Review: Dr Ewald Report

1.0 Introduction

Environmental Risk Sciences Pty Ltd (enRiskS) has been engaged by the Australian Energy Council Environmental Working Group (EWG) to undertake a peer review of the following report:

- The health burden of fine particle pollution from electricity generation in NSW, prepared by Dr Ben Ewald, dated November 2018.

It is understood that the report was commissioned by and prepared for Environmental Justice Australia (EJA) and there is no evidence that the report has been peer-reviewed prior to publication. Dr Ben Ewald is noted (at the start of the report) to be a GP in Newcastle and Senior Lecturer at the University of Newcastle School of Medicine and Public Health.

The scope of the review presented in this letter is as follows:

- **Task 1:** conduct a critical peer review of the report, where the following will be undertaken:
 - Critique of the methodology adopted including the papers relied upon for the characterisation of health impacts;
 - Critique and review of the assumptions adopted, and the calculations undertaken (this will include verification of the calculations presented). The uncertainties presented in the report will also be reviewed and considered; and
 - Critique of the conclusions presented in the report.
- **Task 2:** recommendations
 - Comments and recommendations on what a more rigorous health impact assessment scope of works may involve in relation to characterising potential impacts of fine particulate emissions from coal fired power generators.

The report aims to provide a quantitative assessment of health impacts from fine particulates (i.e. PM_{2.5}) derived from coal fired power stations in NSW. To enable a quantitative assessment to be undertaken the following key aspects need to be defined or characterised:

- Source of emissions
- Area or region where exposure may occur, and characteristics of the population in that area
- Exposure concentrations that are relevant to the area being evaluated – that relate to the source of emissions



- Identifying key health endpoints that are relevant to (and have been causally linked with) exposures to fine particulates, and identifying the most appropriate exposure-response relationship to utilise
- Characterisation of health impacts for the population in the area evaluated

The Ewald report states that it utilises the best published estimates of the components of air pollution, the risks of disease and the population exposures at the time of writing. This statement will be evaluated as part of this review.

2.0 General comments on report

The Ewald report has no section numbers which makes it difficult to reference sections of the report. In addition, the Ewald report is poorly referenced, with many sections providing statements with no references as to the basis of such statements.

The Ewald report is not sufficiently transparent, hence the detailed calculations undertaken cannot be checked and verified. This is especially important where the conclusions of the report make claims regarding specific sources being directly attributable/responsible for mortality.

It is expected that such technical reports would include all references (and data and interpretation) to support statements, and that sufficient information is available to ensure that calculations are transparent and can be verified. This is standard practice for scientific writing and would mean this report would not be acceptable for publication in a scientific journal.

In addition, the report comments on a number of occasions that there is certainty in the estimates being used. This is unusual. It is standard practice for scientific assessments to discuss the uncertainties in an estimate and indicate whether those uncertainties are sufficient to make a difference to the outcome of a calculation or the conclusions of a study. Given that the estimates used in this review are based on adjustments to estimates made by others, at least a discussion of the uncertainties raised by the others would have been appropriate to include. Just making statements that a value is certain without evidence to support such a conclusion is highly unusual in a scientific assessment and would not be acceptable in most situations.

3.0 Source of emissions

The Ewald report indicates that the source of fine particulates being considered is emissions from current electricity generation. The 5 power stations considered in the assessment are Bayswater, Liddell, Eraring, Vales Point and Mount Piper.

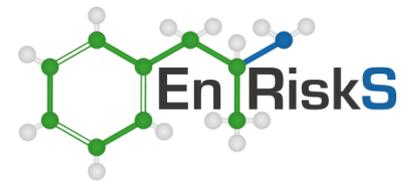
4.0 Area/region for evaluating exposure

The Ewald report has considered 71 Local Government Areas, identified as those that are within 200 km of a coal-fired power station. It is unclear why a distance of 200 km was selected for the analysis, as no reference or basis has been provided. There is no understanding of whether this population matches in with the method used to estimate exposure. Based on the review undertaken the exposure estimate is very rough and has made numerous assumptions to allow the data to be applied to the population selected.

5.0 Characteristics of the population

Population data for each of the LGAs was noted to have been obtained from the Australian Bureau of Statistics (ABS) for 2015. It is not clear why the 2016 census data has not been used – no comment was provided by the author.

Health data was noted to have been retrieved from NSW Centre for Epidemiology and Evidence in 2018, with mortality data obtained from the ABS for all ages. The value was adjusted to estimate the >30 years



mortality rate. The population and health data are not presented so these adjusted values cannot be checked for each LGA. It is noted that data that is specific for mortality for ages 30 years and older can easily be requested from the Australian Institute of Health and Welfare (AIHW) and would then not require the value to be estimated. An estimated value adds to the uncertainty of the overall assessment.

It is noted, however, that the mortality rates adopted for the areas evaluated are within the range expected for various areas in Sydney and NSW even though they cannot be checked for each LGA.

6.0 Health endpoints and exposure response functions

6.1 General

There are numerous publications available that provide statistical associations between exposure to particulate matter (specifically $PM_{2.5}$) and a variety of health outcomes. These evaluations come from epidemiological studies. However, not all epidemiological studies are of the same quality, or show a consistency of health outcome from the pollutant exposure. Quality and consistency limit how confident an assessor can be about the true nature of the health outcome assessed and so affect the ability of a particular epidemiological study to be used. For those studies where there is strong evidence that $PM_{2.5}$ exposure is related to the health outcome, that health outcome is considered a core health outcome.

A core health outcome is defined as one where a strength or weight of evidence approach has been undertaken when analysing the epidemiological evidence defining the health outcome, and this strength or weight has been assessed as strong. In other words, we are sure that what has been reported really is what the data indicates and is not just due to random chance, bias or confounding. It is also important that the health outcomes considered have been determined to be causal – i.e. exposure to $PM_{2.5}$ has caused the health effects. This is different from an association where this is simply whether there is a statistical association. Without understanding if it is causal, such an association may or may not be solely related to $PM_{2.5}$.

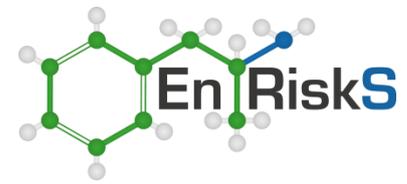
In the conduct of a health impact assessment, it is important that the health effects considered and characterised relate to core health outcomes. These core health outcomes are most clearly determined by detailed reviews of the available literature by recognised experts for organisations like the World Health Organisation or the US Environmental Protection Agency. Such reviews typically following a documented robust approach to appraise the published epidemiological studies and supporting mechanistic studies to determine core health outcomes that may be considered causal.

The section titled “Health outcomes assessed” does not provide any critical evaluation of the health effects associated with exposure to $PM_{2.5}$, nor any discussion as to why the 3 health endpoints assessed were chosen. The report has selected mortality (all causes, for ages 30 years and older), incidence of Type 2 diabetes (ages 49 and older), and low birth weight. The report has selected the studies to rely on, stating that these studies are “convincing” without detailed discussion as to why it is considered that they are convincing.

6.2 Mortality

It can be agreed that the available literature and critical reviews have clearly shown that exposure to $PM_{2.5}$ is causally linked with mortality, and that this is a core health endpoint. It is also agreed that the exposure-response relationship (Krewski et al. 2009) adopted in this assessment is current, robust and appropriate for the assessment of all-cause mortality.

The Ewald report, however, has not included or considered any other key health endpoints that recognised reviews have shown to be causally linked with exposure to $PM_{2.5}$, including cardiovascular and respiratory mortality and morbidity (Morawska et al. 2004; USEPA 2009, 2018).



6.3 Other health endpoints

The Ewald report, however, has included diabetes and low birth weight. These are not the usual endpoints considered in assessment of potential health risks from exposure to PM_{2.5}. Consequently, the inclusion of these health endpoints requires much more justification and discussion as there is currently no consensus that these are causally linked with PM_{2.5} exposures.

The published literature has a wide range of health effects that have been associated with changes in PM_{2.5}. There are a number of critical reviews available in relation to PM_{2.5} that should have been considered and referenced, however, these have not been included.

In the most recent update to the NEPM Ambient Air Quality, a review was undertaken (Jalaludin & Cowie 2012) to determine the most appropriate health endpoints and exposure-response relationships for the assessment of health impacts and burdens from population exposures to particulate matter (as well as nitrogen dioxide, ozone and sulfur dioxide), including consideration of reviews undertaken by other key organisations such as the USEPA, European Commission and the World Health Organisation. This review has not identified diabetes as a key health endpoint, and in relation to low birth weight, the review did not identify any suitable exposure-response functions due to the inconsistency in the outcomes from the available studies.

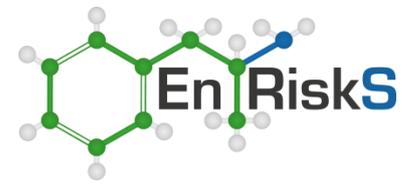
The most current review (draft) from the USEPA (USEPA 2018) has evaluated the available data in relation to the incidence of type 2 diabetes and low birth weight, including the robustness of the studies, biological plausibility and supporting toxicological evidence. These findings are discussed below.

Type 2 diabetes

In relation to type 2 diabetes, when all the available studies are considered they do not consistently report positive associations, however, some high-quality epidemiological studies have reported positive associations between PM_{2.5} exposures and incidence of type 2 diabetes. The USEPA (2018) evaluation concluded the evidence was suggestive but not sufficient to infer a causal relationship. The evaluation included consideration of the Eze et al (Eze et al. 2015) meta-analysis, but not the Bowe et al. (Bowe et al. 2018) study – the only 2 publications referenced in the Ewald report.

Eze et al (2015) was a meta-analysis of 13 studies for a range of air pollutants. Only 3 of these studies (2 of which evaluated males and females and one only evaluated females) included analysis of an association with PM_{2.5}. The paper identifies a range of issues that can result in bias in the results of these studies, with the paper concluding that while an association has been identified there is a high risk of bias in the outcomes. One of the key factors identified was the diabetes risk factors which were not considered in these studies. If there are other significant factors that could be related to this disease that are not included in a study, then it is possible to overestimate how much exposure to PM_{2.5} could relate to diabetes.

The study by Bowe et al (2018) was utilised in the report as the basis for quantitative estimates of health impacts. The study was a longitudinal cohort study that followed up participants over a median time period of 8.5 years. PM_{2.5} exposure was based on the regional air quality data from the county they resided in at the time they started participation in the study. Only a limited assessment of changes in residence or PM_{2.5} exposures over the duration of the study were considered so the study has not well defined PM_{2.5} exposures for the cohort for the duration of the study (despite the report suggesting that PM_{2.5} exposures were well characterised). There is also no consideration of individual PM_{2.5} exposures or changes in exposures at localised levels. Hence, it is not possible to conclude that the PM_{2.5} exposure for all participants was well characterised or defined. In addition, there is no evidence that the study adjusted for co-exposures to other air pollutants, considered differences in socioeconomic status or physical activity. The inclusion of a negative outcome control is helpful as it suggests the study was of high quality. The paper provides evidence of a positive association between PM_{2.5} exposure and incidence of type 2 diabetes. However there remains



sufficient variability in the available studies (as summarised by the USEPA (2018) and Bowe et al (2018)) to question whether the data is sufficiently robust to indicate a causal link between PM_{2.5} exposure and the incidence of type 2 diabetes, and if the exposure-response relationship from the Bowe et al (2018) paper is appropriate to use to characterise health impacts.

Low birth weight

In relation to low birth weight the USEPA (2018) review concluded there is evidence that low birth weight (LBW) is associated with PM_{2.5} exposure, and there is biological plausibility for this association (and other associations such as foetal growth and preterm birth). However, overall, the USEPA considered that the evidence is suggestive of, but not sufficient to infer, a causal relationship between PM_{2.5} exposure and low birth weight (and other reproductive and developmental outcomes).

The USEPA (2018) review included consideration of the study by Pederson et al (Pedersen et al. 2013) which was identified in the Ewald report for use in the quantitative assessment, as well as all other available studies. Review of the other studies available, where large populations from the US, Canada or Europe have been evaluated, shows mixed outcomes both in terms of associations (positive or negative) and the relative risk or odds ratios that come from these studies.

The Pedersen et al (2013) study considered data from 12 European countries over the period 1994 – 2011 and considered exposure to PM_{2.5} and NO₂. Exposure concentrations of PM_{2.5} were based on estimated (modelled) values for the mother's home address. Traffic intensity on local roads was also considered (within 100 m of major roads) but no localised or individual PM_{2.5} exposure concentrations were considered. The study is considered to be of high-quality considering exposures over the duration of pregnancy and collecting a range of data and information throughout the pregnancy. The study provided a quantitative odds ratio of 1.18 (95% CI 1.06–1.33) per 5 µg/m³ increase in PM_{2.5}.

A meta-analysis of 4 earlier studies (Sapkota et al. 2012) suggested a 9% increase in risk of LBW for a 10 µg/m³ increase in PM_{2.5} (combined odds ratio, 1.09; 95% CI, 0.90–1.32). Similarly, another earlier meta-analysis (Dadvand et al. 2013) that considered 7 studies identified an odds ratio of 1.10 (95% CI: 1.03, 1.18) per 10 µg/m³ increase in PM_{2.5}. The USEPA (2018) review identified odds ratios (for larger population studies) in the range of <1 (i.e. no positive association) to an upper level of the Pedersen et al (2013) study.

These studies illustrate that the quantitative exposure-response relationships are variable for low birth weight as a health endpoint. Other published studies and pooled meta-analyses provide less conservative odds ratios than the study adopted in the Ewald report. There is no discussion in this report as to why the exposure response relationship from the Pedersen et al (2013) study was the most appropriate/robust to choose.

6.4 Overall

Overall, the current science supports the characterisation of all cause mortality to be causally linked with PM_{2.5} exposures and considers this a core health outcome for such assessments. The exposure-response relationship adopted is robust and one which is used in numerous other assessments of PM_{2.5} exposures in Australia.

The science, however, does not support the quantification of the incidence of type 2 diabetes or low birth weight as core health outcomes. The quantification of these other health outcomes, and the basis upon which exposure-response relationships has been selected has a very high degree of uncertainty and should not be relied on.

The calculations and conclusions presented in relation to all-cause mortality have been further reviewed.

7.0 Exposure concentrations

7.1 Approach taken

The Ewald report has taken an approach to estimate the contribution of coal-fired power stations to measured levels of PM_{2.5}. In particular, the assessment has considered:

- the formation of secondary particulates as a result of atmospheric transformations of sulfur dioxide and nitrogen dioxide, based on the studies undertaken by ANSTO on particulate characterisation in the Upper Hunter Valley, Lower Hunter Valley and Sydney (Cohen et al. 2016; Hibberd et al. 2016; Hibberd et al. 2013);
- emissions data available from the National Pollutant Inventory (NPI) (for power stations and industry within 50 km of the power stations); and
- annual average PM_{2.5} concentrations measured at OEH monitors in NSW.

It is not clear why primary PM_{2.5} has not been considered for exposures to populations close to the power stations. No commentary has been included in the report in regard to this matter. For populations close to the power stations, this would be an important contribution to exposures.

7.2 Review of approach – Malfroy and Nelson

The approach adopted has a number of flaws and issues, many of which are outlined in the paper by Malfroy and Nelson (Malfroy & Nelson 2018). This paper provides a critique of the methodology adopted in the Ewald report for the estimation of the contribution of coal-fired power station emissions to measured PM_{2.5}, with the key issues identified being:

- The limitations within the particulate characterisation studies undertaken, particularly in being able to clearly define specific sources of PM_{2.5}, including being able to determine the trajectory of emissions from specific sources that are a long way from Richmond.
- A proper understanding of the complexities of secondary particulate formation within a local area vs a larger airshed that is further away from the sources. This is very important as the Ewald report has mixed localised emissions from the NPI within 50 km of the 5 power stations and data from the larger air shed characterisation studies. In addition, there has been no consideration of primary PM_{2.5} emissions from the power stations and their impact on local areas.
- Assuming that the power stations operate consistently at full load (i.e. at the highest emission rate as reported to the NPI).
- Assuming that emissions from the larger number of power stations considered in the ANSTO reports (8 power stations) remains the same, despite the closure of some of these power stations. It is important to note that the Ewald report has simply made the statement that the “remaining power stations are running at higher capacity factors, so sulfur emissions have hardly changed.” This statement has no reference or data to support it. The Malfroy and Nelson review provides a graph of SO₂ emissions from NSW coal-fired power stations over the years (see below). This shows a reduction over time for the Upper Hunter and western region plants and a plateau of emissions from the Central Coast facilities. Hence such a statement is not correct and assuming the particulate characterisation studies that utilised data when all 8 power stations were operating is representative of current conditions is not correct.

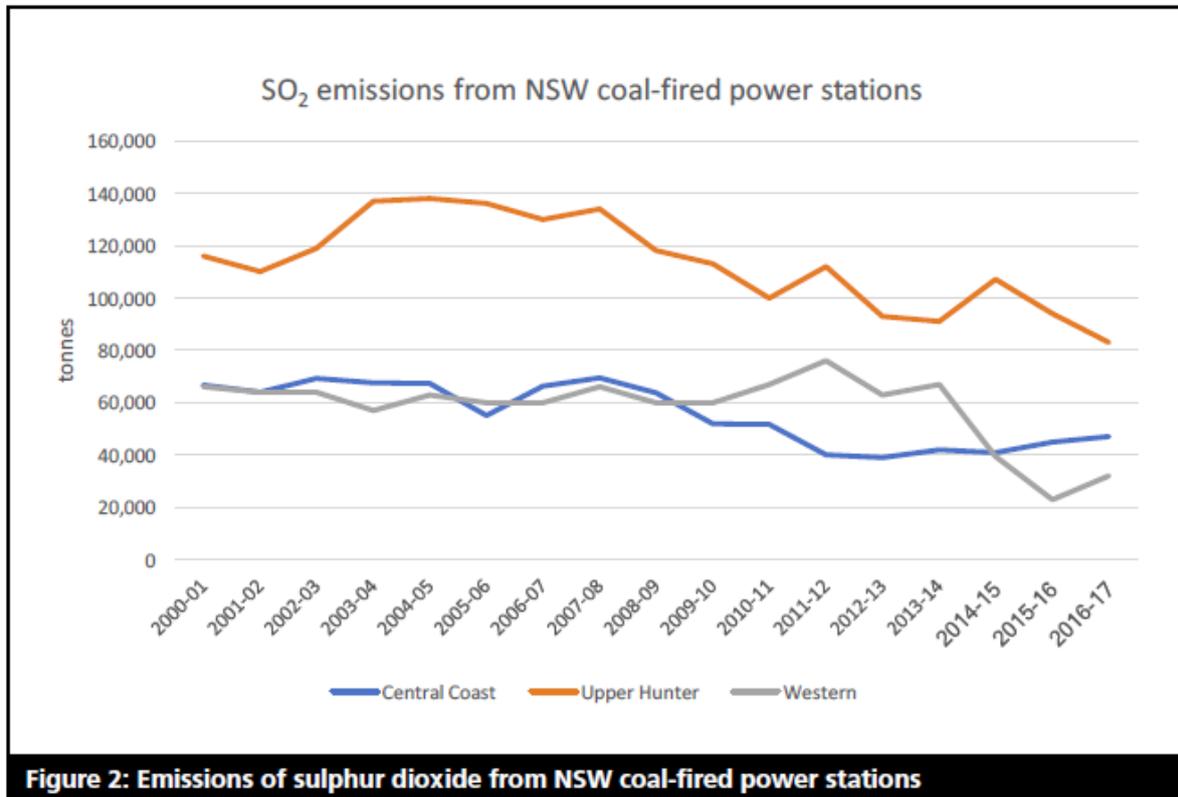


Figure 2: Emissions of sulphur dioxide from NSW coal-fired power stations

From Malfroy and Nelson (2018)

- Assuming the data collected from Richmond is representative of all areas in Sydney and the rest of NSW (outside of the Hunter Valley and Central Coast). The data collected from Richmond has a number of localised characteristics, in particular nocturnal inversion, that limit its applicability to other areas.
- No consideration of potential impacts arising from other sources which contribute an estimated 84% of the particulates as measured at Richmond.

The Malfroy and Nelson (2018) review also highlights the 2003 air modelling study that was undertaken, is publicly available and has not been referenced or discussed in the Ewald report. The 2003 modelling study (Malfroy et al. 2005) was a complex model designed to characterise the contribution of coal-fired power station emissions on PM_{2.5}. The modelling was complex and considered the chemistry associated with the transformation of SO₂ and NO₂ to secondary particulates and the long range transport of these pollutants.

The modelling was set up to consider more power stations than are currently operating, and uses conservative assumptions about how these are operating (i.e. always at full load). This more detailed modelling of emissions and movement and transformation of pollutants is more accurate than the generalised estimation approach adopted in the Ewald report. This detailed modelling estimates coal-fired power stations contribute, as an annual average, 0.3 µg/m³ to PM_{2.5} averaged over Sydney (Malfroy et al. 2005), much lower than the estimate of 1.2 µg/m³ in the Ewald report. This work was undertaken when there were 7 coal fired power stations operating, compared with the 5 currently operating. The complex modelling is currently being revisited by the NSW OEH. It would be appropriate to consider the outcomes of that modelling when it becomes available within any assessment of health impacts.



7.3 Review of the use of data from the NPI

General

The Ewald report has referenced and relied on emissions data from the NPI.

The National Pollutant Inventory (NPI) is an initiative of the governments of Australia and has been established under a National Environment Protection Measure (NEPM). These measures are established under the National Environment Protection Council Act 1994. There are also a range of other such Measures made under the same Act relating to air quality, movement of controlled waste, assessment of site contamination and used packaging (NPI 2015). The purposes of the NPI NEPM are to:

- Collect a broad range of information on emissions and transfers of a range of chemical substances;
- Disseminate that information in a useful, accessible and understandable form (NPI 2015).

The Measure requires:

- Companies that meet certain requirements (size, types of chemicals used, amounts of chemicals used) to report various statistics for the chemicals they use;
- State governments to collect the data from companies and aggregate the data; and
- Commonwealth government to:
 - compile supporting information
 - collate all the data across Australia
 - provide the data on line in an accessible and understandable form (NPI 2015).

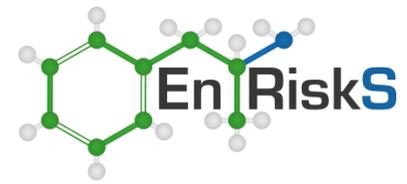
The NPI was developed to track pollution across Australia and to respond to community right to know about chemical substances released in their local environment. Similar systems are used in a number of countries including:

- USA;
- Canada;
- UK;
- Netherlands;
- Sweden;
- Switzerland;
- Mexico;
- Chile; and
- France.

The first step in setting up such a system was to decide which chemical substances needed to be covered. In 1997, a group of experts was asked to develop a risk based approach to choosing the chemical substances of most importance. They developed criteria regarding the potential of a chemical substance to have effects on people or the environment or for it to be released into the environment in large quantities. They applied this system to 420 chemical substances on a master list and ranked them. The top 90 were included in the reporting list for the Inventory – the idea being to keep track of the most important chemical substances (NPI 2015).

For each of the chemical substances, a threshold was set for the amount of a substance to be used in one year by an individual company that would trigger a requirement for a company to report. Also the NPI team at the Commonwealth Department of the Environment prepared a factsheet for each chemical (NPI 2015).

Companies are required to report their emissions of chemical substances to air, water or land if they exceed the usage triggers. They can estimate their emissions using a number of methods based on:



- Various indicators of the size of their operation (e.g. energy used or how much material was manufactured in a year);
- Measurements at various times of the year which are combined with yearly production; or
- A combination of these methods (NPI 2015).

The NPI team have prepared manuals explaining how emissions are to be estimated. These manuals are available on line for the companies who have to report and for the community to see the recommended methods for estimating the emissions of chemical substances. The manuals are based around different industry types so that they cover emission types and chemical substances relevant for a particular industry (NPI 2015).

If there are industries or activities that do not exceed the usage triggers, they do not need to report emission to the NPI. Hence, the NPI inventory does not include all sources of specific pollutants. There can be numerous small sources of pollutants that are not included.

Power stations

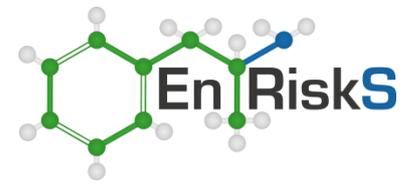
For power stations data, the following should be noted in relation to the data presented in the NPI (NSW EPA 2018):

- The NSW EPA does not regulate power stations via the NPI program. Power stations are regulated on the basis of Environment Protection Licences (EPLs), and air emissions testing is undertaken and reported in accordance with EPL conditions.
- Emissions reported by power stations to the NPI are estimated based on the use of approved emissions estimation techniques, which may or may not include measurement of emissions from a facility (NPI 2012).
- Review of the use of the approved emissions estimates by the NSW EPA determined that:
 - all power stations used approved methods,
 - minor errors were found in most power station emissions estimates for most years and
 - the particle emissions were found to follow the annual stack test results.

In addition, the review identified that there were some differences in the methods used to estimate emissions from each power station. It was also highlighted that PM_{2.5} was estimated from PM₁₀ emissions using standard factors rather than measurements.

A further review of emissions as reported by the NPI by the Australian Energy Council (AEC 2018) indicates the following:

- The NPI data implies that 100% of emissions originate from industry. The NPI does not include emissions from diffuse sources such as motor vehicles, household activities (e.g. woodfired heaters) or biogenic/natural sources. For PM_{2.5}, the review quotes the CSIRO who indicate only 10% of PM_{2.5} is from man-made sources (with 90% from biogenic and natural sources). This means that if power generation is assumed responsible for 27% of PM_{2.5} emissions based on the NPI, the actual figure would be much less and could be 2.7% if biogenic and natural sources were included. As a result, using the NPI data to estimate a proportion of measured PM_{2.5} that comes from a specific source, will be misleading and will overestimate the contribution. The Ewald report has used information provided by both the NPI and the Particle Characterisation Studies to determine the contribution from power stations. This will result in an overestimation of the contribution.
- In relation to PM_{2.5}, the NPI only includes combustion sources (that meet the trigger levels). It does not include any other types of PM_{2.5} sources such as the handling of materials or earthworks.



- The data provided in the NPI only reflects estimated emissions (i.e. what is released from the top of a stack). It does not mean or reflect what exposure may occur within the community at ground level where people breathe. Actual exposures that occur as a result of emissions from power generators will depend on the dispersion of emissions from each power generator. In addition, where other data is being used from the NPI to determine a proportion from all industry sources, actual community exposures will depend on the dispersion characteristics for each individual source. This makes using only the emissions data from the NPI misleading in terms of community exposures.
- The NPI does not include agricultural emissions. This is of particular importance for the assessment of the formation of secondary particulates (as has been assessed in the Ewald report). Ammonia emissions from agriculture play an important role in the generation of secondary particulates from SO₂ and NO₂. Without understanding these sources and the complex chemistry behind the generation of these secondary particulates, particularly over a large air shed, it is difficult to rely solely on NPI emissions estimates of SO₂ and NO₂ emissions from industry alone to determine a contribution for power stations.

7.4 Overall

Overall the approach adopted in the Ewald report for estimating the contribution of PM_{2.5} that may be from secondary particulates from emissions from coal fired power stations is flawed and misleading, not based on good science and will have resulted in a significant overestimation.

The Ewald report, however, concludes that the approach they undertook to determine the contribution of power stations emission to PM_{2.5} in Sydney “are regarded as certain”. This statement cannot be supported at all based on the above comments.

To be able to properly determine the potential contribution of power station emissions to PM_{2.5} in any air-shed outcomes from detailed modelling of both primary and secondary PM_{2.5} should be considered. If the modelling completed in 2003 (where more coal-fired power stations were operating) were used the contribution to the total PM_{2.5} would be 25% of that estimated in the Ewald report. It is this type of detailed modelling that should be the source of any estimation of potential exposure concentrations within the community, and any risk calculations.

8.0 Health impact calculations undertaken

As no detail is presented on how the calculations were undertaken for each LGA in this report, these calculations cannot be directly checked. However, in relation to mortality, a basic calculation check has been undertaken as follows (using the equation from the Ewald report – which is appropriate):

$$\text{Population incidence} = (1 - 1/\exp(\beta \times \Delta X)) \times B \times P$$

β = coefficient for a 1 ug/m³ increased in PM = 0.0058 (from the Krewski et al 2009 paper)

ΔX = change in PM_{2.5} concentration assessed (ug/m³ – see below)

B = baseline incidence of the health endpoint (mortality all cause, ages 30 years and over) (per person)

P = population exposed (see below)

For the parameters listed in Table 9 of the Ewald report, the following table provides an overview of a check of the calculations for the population incidence.

Table 1: Check of population health incidence calculations

Parameter	Sydney	Central Coast	Lower Hunter	Upper Hunter	Remainder
Calculations based on emissions estimates from Ewald report					
Population aged 30-99 (from Table 9)	2657209	211514	350056	38649	714658
All cause mortality 30-99 years (from Table 9)	21709	2065	3555	383	6824
Comment – not able to be checked but data consistent with mortality rate for ages 30 years + for Sydney and NSW					
Population weighted average PM _{2.5} (all sources) (from Table 9)	7.7	6.1	7.4	8.4	6.8
Comment: not able to be checked as insufficient data available – however the values appear reasonable					
Incidence – all sources of PM _{2.5} (calculated from the above parameters)	948	72	149	18	264
Comment: calculated value slightly lower than in Table 9 for some areas, as it is not possible to repeat the calculation for every LGA. Values are essentially the same					
Proportion of total PM _{2.5} that is from power generation (from Table 9) (%)	16%	34%	34%	37%	16%
Incidence – PM _{2.5} from power generators (calculated from the above parameters)	152	24	51	7	42
Comment: calculated value slightly lower than in Table 9 for some areas, as it is not possible to repeat the calculation for every LGA. Values are essentially the same					
Revised calculation of Incidence – based on emissions from detailed PM_{2.5} modelling (primary and secondary particulates)					
Proportion of total PM _{2.5} that is from power generation (from Malfroy et al 2005) (%)	4%	10%	24%	21%	4%
Incidence – all sources of PM _{2.5} (calculated from the above parameters)	38	7	37	4	12
Comment: The calculated numbers are significantly lower than presented in the Ewald report					

In relation to the calculation of years of life lost (YLL), the calculations presented in the Ewald report cannot be verified as there is insufficient information provided to replicate the calculations undertaken. However, where data available for Australia (population and health data) and United Kingdom (Office of National Statistics (ONS) life tables template (Chiang method (II))(ONS 2011)) are utilised, the calculated YLL is significantly lower than presented in the Ewald report (for the data as presented in the Ewald report as well as the revised calculations).

The Ewald report alleges a “certainty” for the mortality estimates provided, indicating these range from low (for the remainder of the study area) to high for Sydney. The certainty in the calculations should not be considered anything other than low for all areas evaluated given the issues raised above. The calculations presented should all be considered to have a high level of uncertainty.

No comments are provided on calculations presented for type 2 diabetes and low birth weight as these health endpoints are not considered sufficiently robust to include any quantitative assessment.

The Ewald report also includes a range of scenarios evaluated in the sensitivity assessment, including an assessment of health impacts from individual power stations and total health burden of the operation of these power stations for the remainder of their operational life. This additional analysis is even more uncertain than the calculations presented in the main part of the Ewald report and should not be relied on for any conclusions.

It is noted that, in addition to the calculation of a population incidence, it is possible to also calculate the population or community risk associated with PM_{2.5} exposures associated with emissions from coal-fired



power stations. Where such risks are calculated, based on emissions from the detailed PM_{2.5} modelling, the calculated risks are consistent with those that are calculated from urban activities/sources such as vehicle emissions from new major road infrastructure (e.g. NorthConnex and WestConnex).

It is important that any assessment of the health impacts from any one source is also considered in the context of other key urban sources. This would assist in better understanding and contextualising the health impacts of these sources.

The Ewald report concluded that the health impacts/burden is “substantial”. There is no definition of what substantial means, other than providing some absolute numbers, none of which are discussed in the context of the population size, variability within the population or other sources.

9.0 Conclusions

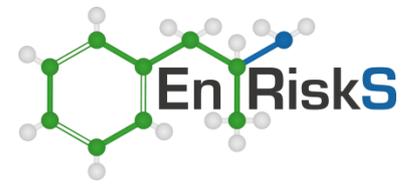
Review of the Ewald report has identified a range of issues that call into question the outcomes presented as well as the level of certainty placed on the outcomes presented. These issues include:

- The area evaluated appears to be randomly chosen and does not specifically relate to any method for estimating exposures. The baseline incidence for mortality is only estimated, rather than being based on specific age-specific data which is known to be available.
- In relation to the health effects considered in the report, the current science supports the characterisation of all-cause mortality as this is a core health outcome which has been shown to be causally linked with PM_{2.5} exposures. The science, however, does not support the quantification of the incidence of type 2 diabetes or low birth weight as core health outcomes. The quantification of these other health outcomes has a very high degree of uncertainty and should not be relied on. The Ewald report has not evaluated other key/core health effects that have also been causally associated with exposure to PM_{2.5}.
- The Ewald report has estimated emissions, and potential exposure concentrations (i.e. PM_{2.5} exposures in large population areas that may be from power generators) using a very rough approach. The assessment has only focused on exposure to secondary particulates. No assessment of primary particulate emissions has been included. Review of the approach indicates that it should be considered as flawed and misleading, not based on good science and will have resulted in a significant overestimation.
- As a result of the issues noted above, any health impact calculations will carry through the same issues. The calculations undertaken to estimate population incidence are considered to be mathematically correct (though the detail cannot be verified as insufficient detail is provided in the report), however, the inputs and assumptions adopted are the issue and means the quantitative results cannot be relied upon. The calculated years of life lost presented in the Ewald report cannot be verified and is likely to have been overestimated based on the issues identified above.

It is important to note that the Ewald report consistently makes statements that the assumptions and approach adopted are “certain”. This is not the case. The approach adopted has a very high level of uncertainty, which is not recognised or considered in the report.

10.0 Recommendations

It is entirely appropriate that health impacts from various sources are understood and characterised to assist in risk and policy communication. Where an assessment of the potential health impact of emissions from coal-fired power stations were to be undertaken, the following should be considered to provide a more robust evaluation:



- Clearly defining and understanding the populations of interest in relation to these emissions – and having a good basis for determining these, including making sure these population areas coincide with the populations and regions considered in the estimation of exposure.
- Use of current OEH modelling of emissions, obtaining information relevant to both primary and secondary particulates (as derived from current coal-fired power stations) as an average in various population areas of interest.
- Evaluation of key health endpoints that relate to PM_{2.5} exposure, preferably those that are causally associated, or where there is sufficient consistent weight of evidence to consider the association to be strong. To evaluate these health endpoints, data from NSW Health should be obtained to ensure that the baseline health statistic used are the most current and relevant to the population ages and health endpoint being evaluated.
- Where such an assessment is undertaken all the calculations should be provided so that the assessment is robust and transparent, and others can verify the calculations.
- The assessment should also provide context to the health impacts evaluated and considered, with impacts from other key sources in urban areas also considered so that the outcomes can be weighed up against other types of common exposures. This is especially the case for pollutants like PM_{2.5} which come from a range of sources including naturally occurring and man-made sources (diffuse and localised).

11.0 Limitations

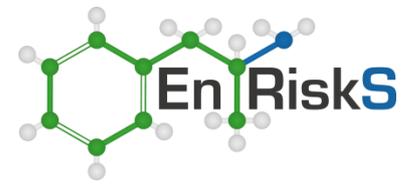
Environmental Risk Sciences has prepared this report for the use of the Australian Energy Council in accordance with the usual care and thoroughness of the consulting profession. It is based on generally accepted practices and standards at the time it was prepared. No other warranty, expressed or implied, is made as to the professional advice included in this report.

It is prepared in accordance with the scope of work and for the purpose outlined in the **Section 1** of this report.

The methodology adopted and sources of information used are outlined in this report. Environmental Risk Sciences has made no independent verification of this information beyond the agreed scope of works and assumes no responsibility for any inaccuracies or omissions.

This report was prepared between December 2018 and March 2019 and is based on the information provided and reviewed at that time. Environmental Risk Sciences disclaims responsibility for any changes that may have occurred after this time.

This report should be read in full. No responsibility is accepted for use of any part of this report in any other context or for any other purpose or by third parties. This report does not purport to give legal advice. Legal advice can only be given by qualified legal practitioners.

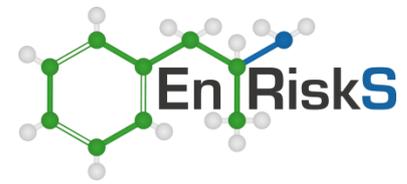


If you require any additional information, please do not hesitate to contact me on (02) 9614 0297 or 0425 206 295.

Yours sincerely,

A handwritten signature in black ink, appearing to read 'Jackie Wright', is positioned below the text 'Yours sincerely,'.

Dr Jackie Wright (Fellow ACTRA)
Principal/Director
Environmental Risk Sciences Pty Ltd



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