The Urban Impact Project acknowledges the Traditional Custodians of the many lands we call Australia, and pay respects to their Elders – past, present and future.

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The Urban Impact Project is an independent health research consultancy based in Fremantle, Western Australia. It is our stance that human health and wellbeing are inseparable from environmental health.

We believe that the problems we face on the climate crisis are systemic, and therefore we work to identify and advocate for bold new approaches to policy and governance.

We acknowledge that we meet and work on the lands of the Nyoongar people.

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EXECUTIVE SUMMARY

"few risks have a greater impact on global health today than air pollution"

Dr Maria Neira, World Health Organization Director of Public Health, Environmental and Social Determinants of Health.

Since the 1960's a number of heavy industries that rely on or extract fossil fuels, or manufacture agricultural products have been approved and developed in the Burrup Peninsula in Western Australia. This includes the:

- Dampier Port, one of the world's busiest cargo ports;
- Rio Tinto's iron ore transfer facilities;
- Woodside North West Shelf Joint Venture (including the Karratha Gas Plant)
- Woodside Pluto LNG; and
- Yara fertiliser and technical ammonia nitrate plants.

Individually and collectively, they all create and release significant point-source air pollution such as Sulphur Dioxide (SO_2), Nitrogen Dioxide (NO_2), Ozone (O_3), particulate matter (PM_{10} and $PM_{2.5}$) and Volatile Organic Compounds (VOCs) into the Burrup air-shed.

Over the coming years, proposals to extend Woodside LNG facilities, as well as the creation of other new industries will be put forward. These industries will also release all their point-source emissions into the same air-shed.

Despite this large-scale industrialisation, and almost 23,000 people living and working in the proximity of the Burrup Peninsula, the Western Australian Department of Water and Environmental Regulation (DWER) have not installed open-source ambient air quality monitors to measure the cumulative impacts of all these pollutants.

Industry-funded monitors, such as the Woodside Burrup Ambient Air Monitoring Program (BAAMP), do not make their data publicly available.

This is concerning. For there are no reasonable and practicable ways to determine if the air quality is 'safe' for the people living and working in the Burrup air-shed. There is also no overarching public health responsibility between the polluting industries, DWER and the Department of Health WA (DoH), such as coordinated health warnings, if and when the quality of air degrades due to industrial activities.

What is well-known is that significant respiratory health injustice already exists in the area. Data from the 2011-2015 Pilbara Health Profile* confirms the respiratory vulnerability, and verifies that:[1]

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*The Pilbara Health Profile data includes the towns of Port Hedland, Newman and Karratha.

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- children living in the Pilbara have 1.7-11 times more hospitalisations for asthma and bronchiectasis (respectively) when compared to the children in the rest of WA; and
- COPD was the most frequent preventable cause for hospitalisation in people aged 60 years and over. It accounted for almost a third of all the preventable hospitalisation in this age group, and that this figure was nearly three times the WA average.

To better understand the relationship between the industrial air pollutants _and_ the significant respiratory injustice in the Burrup air-shed; a two-pronged literature review was conducted to assess the short-term and long-term health effects of SO_2, NO_2, O_3, PM_{10}, $PM_{2.5}$ and VOC's.

The process involved:

- reviewing policy-relevant publications from key national and international agencies, including NEPC, USEPA, WHO and the UK Committee Medical Effects of Air Pollution (COMEAP); and
- searching PubMed and Google Scholar to identify additional or new research for each pollutant.

The key finding was that air pollution is carcinogenic; and that there is no safe level of pollution. Children, the elderly and those with existing cardiovascular and respiratory conditions will certainly be more vulnerable to the effects of these air pollutants. However, their health burden will not be directly proportional to the quantity of exposure. Instead, their response will depend entirely on their individual respiratory and cardiovascular response.

The literature review confirmed that breathing SO_2, NO_2, O_3, PM_{10}, $PM_{2.5}$ and VOC's either when industrial exceedance occurs (short-term), or on a daily basis (ambient long-term) will inherently create an altered life experience for many of the 23,000 people living and working on the Burrup Peninsula.

- SO_2 levels as low as 10ppb (24-hour average) might cause respiratory and cardiovascular mortality;
- Long-term exposure to NO_2 has been linked to deficits in lung function growth;
- NO_2 levels as low 14-2ppb (annual average) in a child's first year of life might increase their chance of getting asthma;
- O_3 levels as low as 60-70ppb might decrease pulmonary function in healthy adults;
- Long-term exposure to O_3 is likely to be one of many causes of asthma development;
- $PM_{2.5}$ is carcinogenic and it is the fifth-ranking mortality risk factor in the world, with an estimated 4.2 million deaths and 103.1 million disability-adjusted life-years in 2015.
- Short-term exposure to $PM_{2.5}$ is more serious. It can aggravate asthma and other respiratory symptom, leading to increased hospital admissions. It can also cause cardiovascular morbidly and mortality such as acute heart failure, acute myocardial infarction, ischemic stroke, venous thromboembolic disease and cardiac arrhythmias.
- Some VOC are carcinogenic. Exposure to high concentrations of VOCs, and for extended periods of time, could lead to increase in chronic headaches, loss of coordination, chronic nausea; liver, kidney and central nervous system damage.

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From birth through adolescence, the effects of breathing air pollution will accumulate over time, into their adulthood and beyond. They will experience more wheezing, more exacerbation of asthma, more respiratory infections, decline in respiratory function, new cases of asthma, more COPD; higher blood pressure, chest pain, thrombotic events, arrhythmias, ischemic heart failure, and maybe cancer.

This burden will be felt throughout the health, education, economic and social paradigms. For they may have to take more days off school/work; may incur extra medical costs and expenses; may utilise more health services such as doctors' visits, outpatient visits, emergency room visits and hospitalisation.

Figure 1: Summary of health burden from air pollutants in Burrup air-shed
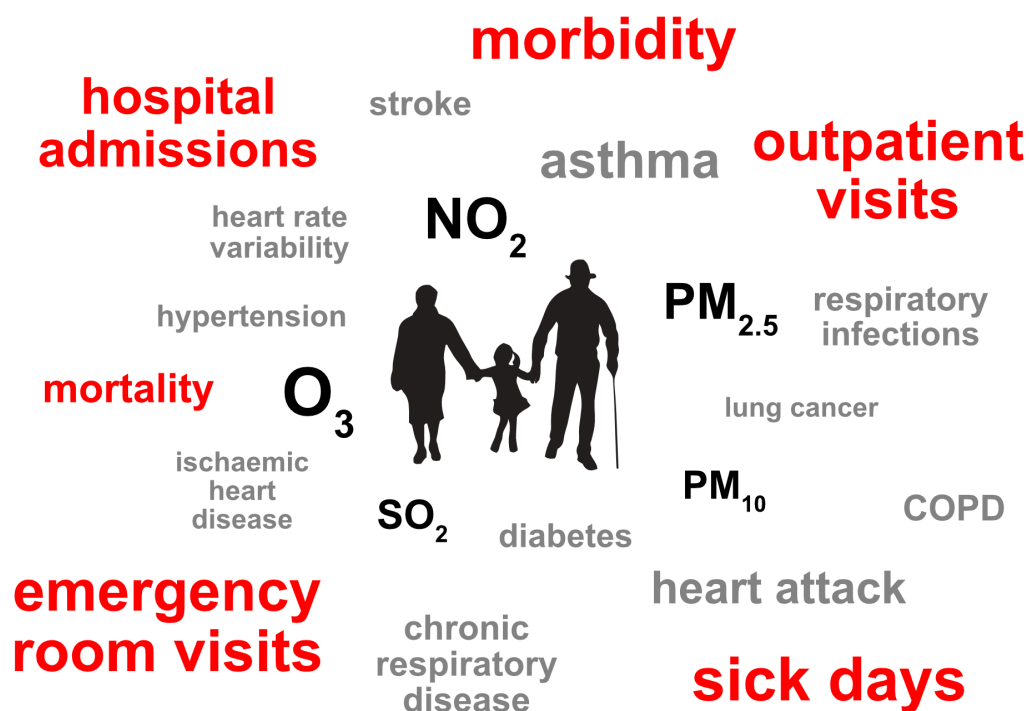
While uncoordinated and fragmented approaches between industries and Government agencies have been normal practice to-date (business-as-usual), we face unprecedented challenges as the climate impacts reach multiple and simultaneous tipping points.

The burden from the industrial air pollution could be dangerous to human health and wellbeing to a degree that might unwind years of progress of other improvements in public health outcomes.

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Therefore, there is an urgent need to resolve the General Public Health Duty omissions as defined in the Public Health Act of WA of 2016; and urgently start addressing the cumulative impacts of air pollution in the Burrup air-shed.

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Section 34: General Public Health Duty

Section 34 of the Public Health Act of WA, 2016 states that "*a person must take all reasonable and practicable steps to prevent or minimise any harm to public health that might foreseeably result from anything done or <u>omitted to be done</u> by the person*".

</div>

Recommendations

The range of possible futures for every child born today in the Burrup Peninsula will largely depend on the approvals and policies that get implemented in the year 2020.

We therefore call that **<u>no new industrial licenses be issued in the Burrup Peninsula</u>** until there is:

1. An Environmental Protection Policy for the Burrup Peninsula. This includes:
   - extending the DWER Air Quality Management Program for Air NEPMS in the Burrup air-shed; and
   - installing a network of DWER open-source air quality monitors in the towns of Karratha and Dampier to accurately assess the ambient air quality.

2. A comprehensive Health Impact Assessment of all the air pollutants in the Burrup air-shed.

3. Open source access to all the Woodside and BAAMP datasets (past, present and future).

4. An effective and responsive public health management plan for the Burrup Peninsula. This includes:
   - Updating and upskilling all public health and medical health services and service-providers, so that they can moderate, mitigate, document and plan their responsiveness to any short-term exceedances and long-term ambient pollution health risks.

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INTRODUCTION

The release of toxic atmospheric pollutants from anthropogenic activities such as fuel combustion, industry, mining, agricultural pesticides and fertilisers, trains, shipping, road and air travel all contribute to climate change and damaging human health. It appears that air pollution is, and will remain, an inevitable consequence as long as global industrialisation relies on fossil fuels, even though the science shows that there is no safe level of air pollution (Raaschou-Nielsen, 2003).

The World Health Organization Director of Public Health, Environmental and Social Determinants of Health Dr Maria Neira, painted a very accurate, and equally worrying picture at the COP25 UN climate change conference in 2019 when she said:

"The true cost of climate change is felt in our hospitals and in our lungs. The health burden of polluting energy sources is now so high… the risks from air pollution are now far greater than previously thought or understood, particularly for heart disease and strokes...Few risks have a greater impact on global health today than air pollution; the evidence signals the need for concerted action to clean up the air we all breathe."

The reality is air pollution is hard to escape, no matter where you live. It is all around us. Microscopic pollutants in the air will slip past our body's defence, penetrate deep into our respiratory and circulatory system, and cause damage to our lungs, heart and brain.

The Australian Institute of Health and Welfare (AIHW, 2016; Begg, 2007) have estimated that about 3000 deaths (equivalent to about 28,000 years of life lost) are attributable to air pollution in Australia each year. This represents almost 2.3% of the total deaths in the whole country per year – more than the number of deaths from car accidents on our roads.

The health costs from mortality alone is estimated to be in the order of $11–24 billion per year (Begg, 2007; Access Economics, 2008). A health risk assessment found that the most severe effects, in terms of overall health burden, were linked to long-term exposure to high levels of particular matter (PM), and that older people, children and people with pre-existing health conditions were most vulnerable to air pollution (Golder Associates, 2013).

This report focuses on the health risks of breathing ambient air pollution (short-term and long-term) in the Burrup Peninsula air-shed. The report includes a:

1. Summary of the current legislation, guidelines and application of air quality assessments in Australia and Western Australia;
2. An overview of the population demographics, the scale of industrialisation, and the current efforts to monitor ambient air quality in the Burrup Peninsula Region;
3. Literature review of the health effects of the most common air pollutants; and
4. Summary and recommendations framed around general public health duty.

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HOW IS THE QUALITY OF AIR WE BREATHE REGULATED IN AUSTRALIA?

This section sets out the current legislation, policy and guidelines that are applicable to air quality assessment in Australia and in Western Australia.

National Environment Protection (Ambient Air Quality) Measure

The National Environment Protection Council Act 1994, together with mirror legislation from State and Territories, established the National Environmental Protection Council (NEPC) to serve two primary functions:

- to make National Environment Protection Measures (NEPMs); and
- to assess and report on the implementation and effectiveness of NEPMs in participating jurisdictions.

In 1998, the NEPC created and released the first national air quality framework and standards – and the National Environmental Protection Measure for Ambient Air Quality (Air NEPM) – was created for the key pollutants, then known, to poses significant human health risk:

- carbon monoxide (CO);
- ozone (O_3);
- nitrogen dioxide (NO_2);
- sulphur dioxide (SO_2);
- lead (Pb); and
- particles (PM_{10}).

Over the past twenty years, the NEPMs have undergone a few of reviews and variations, with the key changes to the Air NEPM being:

- addition of monitoring and reporting fine particles $PM_{2.5}$; and
- updating the monitoring standards for the pollutants to reflect public health scientific evidence.

In 2019, NEPC released, and consulted, on the variation to Air NEPMs for O_3, NO_2 and SO_2. The final standards to be adopted by Air NEPM will be released in 2020 (NEPM, 2019). The documents relating to these can be accessed at http://www.nepc.gov.au/nepms/ambient-air-quality.

A summary of the current, and the proposed draft variations to Air NEPMs for O_3, NO_2, and SO_2 are shown in table 1. The text in purple shows the proposed variations that are still under review (NEPC, 2019).

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Table 1: The current, and proposed draft Air NEPM variations to O_3, NO_2, and SO_2

Pollutant	Averaging period	Max conc in parts per billion (ppb)	Max conc in mcg per cubic metre µg/m3	Maximum allowable exceedances
Carbon monoxide (CO)	8 hours	9000		1 day a year
Nitrogen dioxide (NO_2 and NO_x)	1 hour 1 year	120[a]; 90[b]; 80[c] 30[a]; 19[b]; 15[c]		1 day a year[a]; none[b] none
Ozone (O_3)	1 hour 4 hours 8 hours	100[a]; remove limit[b] 80[a]; remove limit[b] n/a[a]; 65[b]		1 day a year 1 day a year none
Sulphur dioxide (SO_2)	1 hour 24 hours 1 year	200[a]; 100[b]; 75[c] 80[a]; 20[b] 20[a]; remove limit[b]		1 day a year[a]; none[b] 1 day a year[a]; none[b] none
Lead	1 year		0.50	none
Particles PM$_{10}$	24 hours 1 year		50 25	none none
Particles PM$_{2.5}$	24 hours 1 year		25 8	none none

a: current value
b: proposed variation in NEPC, 2019 consultation - to be met 2020
c: proposed variation in NEPC, 2019 consultation - to be met by 2025

Air Quality Monitoring in Western Australia

The responsibility for licensing, compliance and enforcement of emissions and discharges of pollution in Western Australia lies with the Western Australian Department of Water and Environmental Regulation (DWER).

In translating and implementing the Air NEPM, DWER chose to operate and maintain ambient air quality monitors at 14 strategic locations across WA. Table 2 shows these locations and the type of ambient air monitoring occurring at each location.

The live air quality results from those monitors are open-source, and can be accessed at https://www.der.wa.gov.au/your-environment/air/air-quality-index. DWER also publish their annual WA Air Quality Reports (accessible at https://www.der.wa.gov.au/your-environment/air/203-air-quality-publications).

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Table 2: Place and type of ambient air monitoring conducted by DWER in WA

Location of monitor	CO	NO$_2$	O$_3$	SO$_2$	PM$_{10}$	PM$_{2.5}$
Caversham	◆	◆	◆		◆	◆
Duncraig	◆	◆			◆	◆
Mandurah	◆	◆	◆		◆	◆
North Rockingham		◆	◆	◆		
Rolling Green	◆	◆				
South Lake	◆	◆	◆	◆	◆	◆
Swanbourne		◆	◆			
Wattleup				◆		
Albany					◆	
Bunbury					◆	◆
Busselton						◆
Collie					◆	
Geraldton					◆	◆
Kalgoorlie				◆	◆	◆

Issues with NEMPs and air quality monitoring in WA

While DWER provides open-source data to the general public, it is important to understand that these results show an "average" representation of the general air quality in that specific location only. This is important for a number of reasons:

Environmental injustice: few people actually breathe the ambient air that is being monitored. Most of the air quality monitors are placed much higher and far from air pollution "hot-spots". There is significant research to show that people living close to freight routes, busy roads, intersections, certain industries and mining activities are affected more by air pollution health impacts, and thus suffer greater environmental injustice (Higginbotham, 2010; VEPA 2012).

Average representation: some pollutants like SO$_2$ are known to cause respiratory morbidity and decreased lung function very quickly. Studies have found that people with existing asthma, or anyone breathing through their mouth (for example when exercising) could suffer wheezing, chest tightness and shortness of breath within the first few minutes of exposure to SO$_2$ (NEPM, 2019; WHO, 2006). Therefore, the average representation, shown as AQI, tends to dilute the data, prevents documentation of the exact quantity and time of exceedance, and is unreliable when deciding whether it is safe/unsafe to go outside.

Incomplete air quality monitoring: ambient air monitoring should be an integral part of an effective air quality management system so as to safe guard public health from ALL six pollutants. However, only one location in the whole of WA (in South Lake) monitors all six pollutants. Pollutants such as NO$_2$, PM and O$_3$ co-exist in the atmosphere in complicated equilibriums, and the quantity of each pollutant at one time depends on various independent factors including volatile organic compounds and sunlight, as seen in figure 2 (Zhang, 2019). Therefore, the incomplete air monitoring in WA, especially in the areas with industry emissions, could underestimate the quality of the air.

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Figure 2: Equilibrium between oxides of nitrogen (NO, NO$_2$) and formation of Oxone (O$_3$)
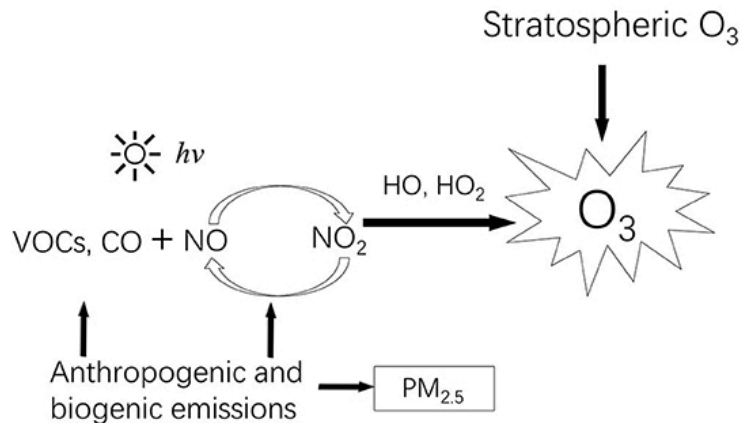
The NEPMs are advisory and not enforceable under law: due to the complex intergovernmental arrangements involving Commonwealth, State and Territory governments, there is no direct responsibility or penalty if the air quality standards are breeched.

Definition of region: The NEPM gives a very broad definition of "regions" where the air quality (for a particular pollutant) should be determined, leaving the exact determination to the various state-based jurisdictions.

In 2001, the WA Peer Review Committee accepted the following definition of a region when creating the WA Ambient Air Quality Monitoring Plan:

- Type 1 - a large urban or town complex with a population in excess of 25,000 requiring direct monitoring and contained within a single airshed;
- Type 2 - a region with no one population centre above 25,000, but with a total population above 25,000 and with significant point source or area-based emissions so as to require a level of direct monitoring;
- Type 3 - a region with population in excess of 25,000 but with no significant point source or area-based emissions, so that ancillary data can be used to infer that direct monitoring is not required.

This approach is highly insensitive, for it fails to monitor and regulate regional industrial air-sheds that create huge and dangerous levels of industrial point-source and area-based air pollution – for example in the Burrup Peninsula Region.

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The Dampier Archipelago is located on the coast of the Pilbara region in Western Australia, containing the towns of Karratha and Dampier. The Burrup Peninsula (27km long by 5km wide) is the largest in the island chain (figure 3).

This section lays out an overview of the population, the scale of industrialisation, the inconsistent efforts to monitor ambient air quality in the Burrup Peninsula Region.

Figure 3: Map of the industries and residential proximity in the Burrup Peninsula region

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The people living and working in the Burrup Peninsula region

In 2018, the City of Karratha, which encompasses the towns of Karratha and Dampier, was home to 22,195 people. It is estimated that the number of people that will reside in these towns will increase to 28,000 by 2040 (Karratha, 2019).

The population-age distribution of Karratha compared to the rest of Western Australia is noteworthy (figure 4).

- Almost a third of the population is under the age of 20 years, with almost 20% being under the age of 10 years; and
- Only 5.5% of the population is over the age 60 years, compared to the WA average of 18%.

Figure 4: The 2018 population- age distribution in City of Karratha and Western Australia
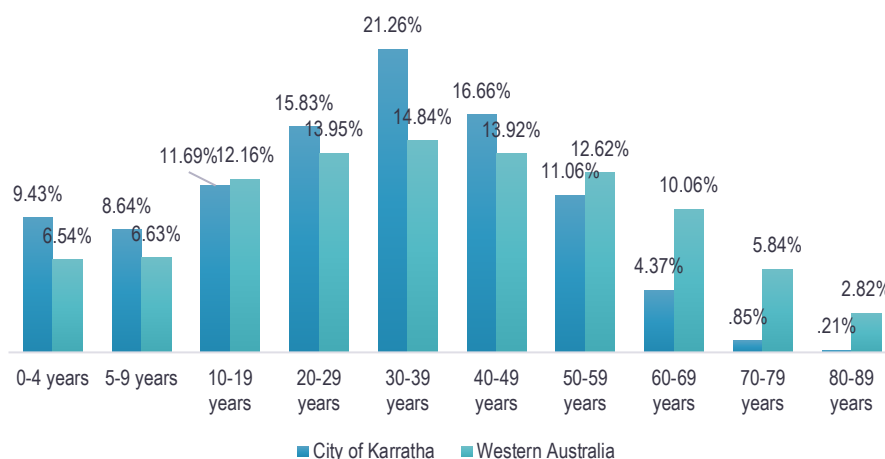
Public health concern

Children are particularly vulnerable to air pollution because they breathe more air more per unit body weight, and therefore inhale more airborne toxicants than adults exposed to the same amount of pollution – all at a very critical time in their life when their brains and bodies are still developing. They also spend more time being active outdoors than most adults.

Therefore, the high proportion of children, in particular younger children, living in the City of Karratha is of concern. There is strong evidence that exposure to short and long-term ambient air pollution will worsen lung function and slower lung function growth in children; which in turn may limit their ability and capabilities in adulthood.

The Pilbara Health Profile report from 2011-2015 identified that there were significantly higher potential preventable hospitalisations (PPH) for respiratory causes in children aged 0-14 years, when compared to the whole of WA. The report showed that in children aged 0-14

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years asthma was the fourth leading PPH. The rate of asthma PPH was 1.7 times higher in the Pilbara when compared to the rest of WA; and the rate of PPH of chronic bronchiectasis was 11.5 times higher (Anderson, 2018). It is however, important to note that this data was for the whole of Pilbara, which includes the towns of Port Hedland, Newman and Karratha (table 3).

Table 3: Pilbara leading PPH in children aged 0-14 years (2011-2015)

PPH Condition	Number of PPH	% all child PPH	SRR
ENT infections (acute)	473	26%	1.4
dental conditions (acute)	451	25%	1.0
cellulitis (acute)	216	12%	2.7
asthma (chronic)	180	10%	1.7
convulsions and epilepsy (acute)	132	7%	1.1
urinary tract infections, including pyelonephritis (acute)	106	6%	1.0
pneumonia and influenza (vaccine)	55	3%	2.0
bronchiectasis (chronic)	46	3%	11.5
diabetes complications (chronic)	21	1%	0.8
gangrene (acute)	20	1%	5.7
All Potentially Preventable Hospitalisations (PPH) (0-14 years)	**1,813**	**100%**	**1.4**

Note: The standardised rate ratio (SRR) is the ratio between a particular health region (or district) and the State. A ratio of 1 means the regional rate is the same as the State, a value of 2 indicates the regional rate is twice that of the State, and an SRR of 0.5 indicates the rate in a region is half that of the State population.

Note: Local rates are compared to the rate of all residents of the State using the SRR and the 95% confidence interval of the SRR. Those that are significantly different to the State rate (1.0) have black font, and those that are not significantly different to the State have white font. Those between 1 and 1.5 times the State rate are highlighted orange, higher than 1.5 times the State are highlighted red while those rates less than the State are highlighted green.

Source: DoH, Health Tracks[v]

Another vulnerable group to effects of air pollution are the elderly i.e. people aged 65 years and above. Even though only a small percentage of the populations stays in the area once they reach retirement age, The Pilbara Health Profile report also showed that the most frequent PPH condition between 2011-2015 was chronic obstructive pulmonary disease (COPD). It accounted for almost a third of all PPH's in this age group, and was nearly three times the State rate.

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Table 4: Pilbara leading PPH in 65 years and over (2011-2015)

Condition	PPH	% all PPH (65 years and over)	SRR
chronic obstructive pulmonary disease	208	29%	2.75
congestive cardiac failure	98	14%	1.49
urinary tract infections, including pyelonephritis	90	13%	1.53
cellulitis	68	9%	2.16
diabetes complications	43	6%	1.07
All PPH (65 yrs and over)	717	100%	1.55

Note: The standardised rate ratio (SRR) is the ratio between a particular health region (or district) and the State. A ratio of 1 means the regional rate is the same as the State, a value of 2 indicates the regional rate is twice that of the State, and an SRR of 0.5 indicates the rate in a region is half that of the State population.

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Source: DoH, Health Tracks[v]

This review did not enquire further into the longer-term trends of respiratory morbidity in the Burrup Peninsula. More recent data was also not available to analyse and comment on.

To investigate this respiratory burden further, a full health review of all the Medicare, Pharmaceutical Benefits Scheme and private health data would be warranted; which was beyond the scope of this study.

However, there is a growing public health concern, that the scale and the rate of industrialisation that extracts and utilises fossil fuels, or releases significant point-source emissions from agricultural fertiliser industries, may be responsible for these respiratory health inequalities in children and the elderly.

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Industrialisation of the Burrup Peninsula Region

Since the 1960's, a number of intense, heavy industries with significant point source and area-based emissions have been approved in the Burrup Peninsula, and future industrialisation is planned. Figure 5 shows a very topical timeline of the some of the key industries in the area.

Figure 5: Industrialisation of the Burrup Peninsula Region

1960s

Dampier Port is developed.

Rio Tinto begins iron ore transfer through the port.

Dampier Salt is formed.

1970s

Dampier Port operations expand.

1980s

Woodside North-West Shelf (NWS) project commences with an offshore domestic gas platform and on-shore processing train at the Karratha Gas Plant. Two LNG processing trains are completed in 1989.

1990s

Woodside NSW: a third LNG train is added at the Karratha Gas Plant and a second off-shore production platform is comissioned. Oil production starts at the Cossack Pioneer FPSO.

2000s

Woodside NSW: a fourth and fifth LNG production train are added to the Karratha Gas Plant and a third off-shore platform is commissioned.

Woodside Pluto LNG: work commences on the Burrup Penisinsula onshore facilities.

Yara Pilbara Fertilizer liquid ammonia plant is built and commences production and export of liquid ammonia.

2010s

Woodside NSW: Cossack Pioneer FPSO is replaced with Okha FPSO. A fourth off-shore gas platform is added.

Woodside Pluto LNG facility commences operations, processing gas from an off-shore platform in the Pluto and Xena gas fields. A truck loading facility is added.

Rio Tinto significantly increase iron ore output through Dampier Port.

Yara Pilbara Nitrates Technical Ammonium Nitrate (TAN) plant is built.

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Future developments in the Burrup Peninsula region

In 2019, Woodside announced their vision to link the Pluto LNG and the NSW project facilities to create the 'Burrup Hub', providing the infrastructure to develop gas resources from Scarborough, Browse, Pluto and North West Shelf (NWS) and enable future development of third-party resources. The Burrup Hub vision includes:

- An extension of the NSW project - enabling processing of third-party gas through the Karratha Gas Plant, and extending the life of the facility for 30+ years;
- The Pluto Train 2 - a brownfield expansion of Pluto LNG through the construction of a second gas processing train, to be completed by 2024;
- The Pluto-KPG Interconnector - a pipeline to transport gas from Pluto LNG to the NWS Project's Karratha Gas Plant; and
- Offshore developments of the Scarborough and Browse natural gas resources.

Two other projects are also proposed in the Burrup Peninsula region. They are:

- Yara liquid ammonium nitrate will recommence (operations closed in 2017 due to issues with cooling towers);
- urea plant by Perdaman Industries; and
- methanol plant by a joint venture comprising Coogee Chemicals, Mitsubishi and Wesfarmers.

Ambient Air Quality Monitoring in Burrup Peninsula Region

Even though the above-listed industries are continually expanding their operations and output, creating significant point source and area-based air pollution, only a handful of ambient air quality studies have been conducted in that region to date.

In the early 2000s, the Western Australian Government sponsored the Pilbara Air Quality study (PAQS, 2004). One of the study objectives was to obtain reliable air quality and meteorological data in order to provide a sound basis for the assessment of the air quality impacts of existing industries and proposed industrial developments in and around Pilbara coastal towns. A few years later, the West Australian Government sponsored another air quality monitoring study in 2004–2005 and 2007–2008 (CSIRO, 2008). However, this study ended before Woodside Pluto went into production.

In 2008 Woodside established the Burrup Ambient Air Monitoring Program (BAAMP), which continued till 2011. Then, as part of the Pluto project, Woodside continued to the BAAMP program until the end of 2015 (Jacobs 2019). However, the BAAMP data was not made available to the public.

This is concerning because there is no reasonable and practicable way of identifying the frequency and degree of risk people living and working in the Burrup Peninsula experience as a consequence of the breathing the air pollution.

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Access to real-time ambient air quality data in the Burrup Peninsula would bring peace of mind, and fulfil public health duty. For it would:

- provide environmental regulators information on ambient air quality trends;
- enable public health specialists and the medical sector opportunity to moderate and mitigate human health risks through warning systems such as asking people to stay indoors - if and when ambient air pollution exceedances occur;
- allow the health services to be more responsive in planning their services and workforce, depending of the nature, extent and duration of exceedances and corresponding respiratory health emergencies; and
- allow full documentation of the true health burden - such as asthma exacerbation, doctor visit, days of school/work/activities, emergency room visits, hospital admissions, and mortality - as a direct consequence of the exceedance in emissions.

Conclusion

In summary, there are almost 23,000 people that call the City of Karratha home, and many of them suffer greater respiratory burden compared to the rest of Western Australia. A number of industries that emit large quantities of point-source pollution in the Burrup air-shed have been approved in the past few decades, and more will be approved in the coming decade.

Yet, there is no real-time ambient air quality monitoring in the Burrup Peninsula. The only monitors available, are industry funded and they do not release their data to the public.

This is concerning. There is no reasonable and practicable way of identifying the frequency and degree of risk people living and working in the Burrup Peninsula experience as a consequence of the breathing the air pollution.

To better understand the health impacts of the key air pollutants in the Burrup air-shed, a literature review was conducted. The aim was to determine the health effects of short-term and long-term exposure to the most common air pollutants.

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LITERATURE REVIEW – HEALTH EFFECTS OF AIR POLLUTION

Methodology

A two-pronged literature review to assess the short-term and long-term health effects of $S0_2$, $N0_2$, O_3, PM_{10}, $PM_{2.5}$ and VOC's was conducted. This involved:

- Reviewing policy-relevant publications from key national and international agencies, including NEPC, USEPA, WHO and the UK Committee Medical Effects of Air Pollution (COMEAP); and
- Searching PubMed and Google Scholar to identify additional or new research for each pollutant.

Deliberate attention was taken to ensure that a wide range of OECD data was extracted and used in this review. Research and data from developing countries was only used when another author had pooled, reviewed or compared their data in meta-analyses. The decision to avoid non-OECD data in this review was to prevent over-indicating health risks from areas and people who suffer greater environmental injustice, complex etiologies, and different baseline health statuses.

To ensure accuracy and consistency, systematic reviews with meta-analysis, structured reviews, controlled human exposure studies and experimental studies were given more emphasis. Data from epidemiological studies was to identify long-term or chronic risks of ambient air pollution.

To ensure coherency of methodology, studies that did not identify or distinguish health effects for specific pollutants were excluded. However, this created a limitation of the review. It is a well-known fact that various co-pollutants exist in the atmosphere in complicated equilibriums, especially NO_x, O_3 and PM as seen in figure 2 (Zhang, 2019). The reality is we breathe all the air, and the potential burden of the multiple pollutants could be much worse for morbidity and mortality risk. Therefore, by excluding the broader air quality studies, the findings of this review could potentially be underestimated.

The key findings from the policy-relevant documents, together with studies identified in this literature review were collated and summarised for each of the key pollutant.

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Breathing Sulphur Dioxide (SO₂) Pollution

Sulphur dioxide is a colourless gas with a nasty, sharp odour. It reacts easily with other substances to form harmful compounds such as sulphuric acid, sulphurous acid and sulphate particles; which are associated with acidification of lakes and streams, accelerated corrosion of monuments and buildings, and reduced visibility. When SO_2 reacts with ammonia, the neutralisation process creates bisulphates and sulphates, which contribute significantly to $PM_{2.5}$ concentrations in some airsheds.

A large amount of literature focusing on short-term and long-term health effects to exposure to SO_2 in ambient air have been reviewed and summarised by (NEPC, 2019; USEPA, 2008; WHO, 2013). The key findings from these policy documents, together with the studies identified in the literature review are summarised below.

Health effects due to short term exposure to SO₂

Human clinical studies have provided the strongest evidence for a relationship between respiratory morbidity and short-term exposure to SO_2. These studies showed that there were increases in respiratory symptoms and decreases in lung function after peak exposures of SO_2 for 5-10 minutes duration (WHO, 2006). People with asthma were particularly sensitive, and the symptoms were worse when the subjects were breathing through their mouth rather than the nose (i.e. when exercising). The exact duration of exposure was not critical because the symptoms occurred very rapidly, within the first few minutes of exposure, and continued exposure did not further increase any of the effects. Statistically significant decrements in lung function accompanied by respiratory symptoms, including wheezing, chest tightness and shortness of breath were demonstrated following short-term exposure to 400-600ppb $S0_2$ (NEPC, 2019).

Intervention studies provide further evidence of an association between short-term exposure SO_2 and respiratory morbidity (USEPA, 2008).

- The Hong Kong intervention study compared the effects of reducing SO_2 (up to 80% in polluted districts) and sulphate (38% in polluted districts) levels on bronchial responsiveness in primary school children living in two districts (polluted and less polluted). The authors found a greater decrease in bronchial hyper-reactivity and bronchial reactivity in schoolchildren in the polluted district than in the less polluted district (Wong, 1998).
- In a case-crossover study of air pollution and child respiratory health undertaken in five Australian and two New Zealand cities, Barnett et al. found a statistically significant increase in hospital admissions and short-term exposure (1-hour) to SO_2. The ambient levels recorded during the study for 1-hour SO_2 ranged from 3.7 to 10.1ppb, and for the mean 24-hour value ranged from 0.9 to 4.3ppb. In the 1–4year age group there was evidence of seasonal impacts on pneumonia and acute bronchitis admissions for SO_2 with higher effects observed in the warmer months (Barnett, 2005).

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Epidemiological studies have reported links between short-term exposure to SO_2 exposure and daily mortality, respiratory effects, cardiovascular effects; and for emergency department and hospitalisation.

- Many studies from various parts of the world, including the United States, Canada and Europe, have reported associations between exposure to ambient levels of SO_2 and increases in all-cause (non-accidental) and respiratory and cardiovascular mortality, often at 24-hour average levels of less than 10ppb (Biggeri, 2005; Samet, 2000a,b; Dominici, 2003a,b; Burnett, 1998a, 2000, 2004; Katsouyanni, 1997, 2003; Samoli 2001, 2003; Stieb, 2002, 2003; USEPA, 2008).
- There are consistent and robust associations between ambient SO_2 concentrations and emergency department visits and hospitalisations for all respiratory causes, particularly among children and older adults (65+ years), and for asthma and COPD (USEPA, 2008). Warmer months were more likely to show evidence of an association with adverse respiratory outcomes in children, while older adults appeared to be more likely to be affected during the cooler months (NEPC 2019).

Health effects due to long term exposure to SO_2

Effects respiratory morbidity, asthma, bronchitis to long-term exposure to SO_2 were summarised in the USEPA report (2008). They stated that there was a positive association between exposure and respiratory health symptoms in children, and that there was no association with lung function. However long-term exposure to $S0_2$ in animal toxicological studies have shown damage to the epithelium of the airways, leading to epithelial hyperplasia. These changes are similar to those seen in chronic bronchitis in humans. Other studies conducted in a range of animal species have shown that long-term exposure to SO_2 produces bronchoconstriction (WHO, 2006).

Epidemiological evidence on effect of long-term exposure to $S0_2$ on mortality was limited because it was not possible to distinguish the potential confounding co-pollutants in the two major US epidemiological studies (Dockery, 1993; Pope, 1995).

Low birth weight, premature birth or other measures of foetal growth restriction have been associated with high exposure to $S0_2$ in a number of studies. While there was variation in demographics, study exposure periods and maximum dose-responses; several well-conducted studies suggest that SO_2 is associated with reduced birth weight, independent of co-pollutants.

- A Canadian study found that first trimester exposures in the highest quartile for SO_2 and PM_{10} suggested an increased risk of delivering a low-birth-weight infant (Dugandzic, 2006).
- In Korea, Leem (2006) also found an association between low birth weight and low levels of air pollutants, including SO_2.
- In the USA, a time-series study undertaken by Sagiv (2005) found evidence of an increase in pre-term birth risk with exposure to PM_{10} and SO_2 which were consistent with prior investigations of spatial contrasts.

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- A significant dose-response relationship was observed between SO_2 and total suspended particulates and gestational age in a prospective cohort study that examined the highest exposure levels. The gestation age distribution curve was more skewed toward the left (shorter gestation) on high pollution days, suggesting that pregnancies at high risk for pre-term delivery may be particularly susceptible to adverse effects of air pollution (NEPC, 2019).

Reproductive and development effects have been linked to relatively high air pollution. Evidence shows that long-term exposure can produce male reproductive toxicity, which manifests as adverse effects on semen quality, sperm chromatin integrity and biochemical parameters in the testis (OEHHA, 2011). A few studies have found that exposure to SO_2 is directly associated with increase in the DNA damage of sperm cells (NEPC, 2019).

Summary of health effects due to SO_2 pollution

Short-term exposure to SO_2 (<24hours) has been shown to be associated with increase in daily mortality, hospital and emergency department admissions, mainly due to respiratory causes including wheezing, chest tightness and shortness of breath.

Epidemiological evidence on the effect of long-term exposure to SO_2 is limited, but there is association between exposure and respiratory morbidly, low birth weight, premature birth or other measures of foetal growth restrictions.

Children, people over 65 years of age, and people with existing disease (respiratory, cardiovascular and asthma) were found to be the most susceptible to the effects of SO_2.

Breathing Nitrogen Dioxide (NO_2) Pollution

Nitrogen Dioxide (NO_2) is a reactive gas which has a reddish-brown colour at typical atmospheric temperatures. It is a precursor to photochemical smog, and contributes to secondary particle formation by reaction with ammonia to form nitrate salts.

In the atmosphere, nitric acid (NO) is oxidised to NO_2. NO and NO_2, collectively referred to oxides of nitrogen (NO_x) then co-exist in complex equilibrium that is influenced by the presence of atmospheric oxidants such as O_3, VOCs, sunlight and other factors. The concentration of NO_2 in the atmosphere at any given time are determined by the rates of competing chemical reactions, as shown in figure 2 (Zhang, 2019).

In recent years there has been a significant increase in the number of studies that have investigated and reviewed the effects of NO_2 on health (WHO 2013b; USEPA 2016; NEPC 2019). The review and policy documents have all found considerable evidence on the health effects of NO_2, and that these effects are independent of other pollutants, including PM.

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Health effects due to short term exposure to NO_2

Exposure to short-term NO_2 is associated with hospital admissions, emergency department visits, asthma and chronic obstructive pulmonary disease. The evidence is supported by human controlled studies, some of which show increased inflammation of airways and hyperresponsiveness at NO_2 levels at 200ppb in health individuals (NEPC, 2019). The NEPC, 2109 report summarised key Australian studies and noted that there were similar associations between hospitalisation for respiratory effects, including asthma, and daily NO_2 as overseas studies (Barnett, 2005; Erbas, 2005; Jalaludin, 2004; Morgan, 1998a; Rodriguez, 2007).

Panel studies in children with asthma found associations between NO_2 and reductions in lung function, increase in cough, night-time asthma and school absenteeism (WHO 2013b; USEPA 2016; NEPC 2019). In a meta-analysis of five Australian and two New Zealand cities Barnett et al. analysed hospital admissions for three age groups of children. They found significant increases in hospital admissions for respiratory disease (1–4 and 5–14 years) and asthma (5–14 years) in either 1-hour or 24-hour NO_2. The largest association reported was a 6.0% increase in asthma admissions with a 5.1ppb increase in 24- hour NO_2. The effect was not reduced by inclusion of PM_{10} in the analysis (NEPC, 2019). The USEPA reported that there are also sufficient epidemiological studies to show a strong association between short term exposure to NO_2 and increase in symptoms in asthmatic children, increase in airway inflammation, and hyper-responsiveness (USEPA, 2016).

Short-term exposure to NO_2 has been linked to cardiovascular mortality. In 2015, Mills et al., conducted a systematic review to assess the evidence from epidemiological time-series studies published worldwide to determine whether and to what extent short-term exposure to NO_2 is associated with increased numbers of daily deaths and hospital admissions, and found clear evidence of health effects associated with short-term exposure to NO_2 (Mills, 2015). Another meta-analysis, of the associations between pollutants and cardiovascular hospital admissions in the elderly in Brisbane, Canberra, Melbourne, Perth, Sydney, Auckland and Christchurch, found significant associations between CO, NO_2, and particles and five categories of cardiovascular disease admissions. The two largest statistically significant increases were for cardiac failure, with a 6.9% increase for a 5.1ppb increase in NO_2 and a 6.0% increase for a 0.9 ppm increase in CO (Barnett et al., 2006).

Health effects due to long term exposure to NO_2

There is a strong evidence of an association between long-term exposure to NO_2 and the incidence of asthma and wheeze. In fact, evidence suggests that NO_2 exposure may actually cause asthma rather than just exacerbate existing asthma (USEPA, 2016; WHO, 2013a). The strongest evidence for these statements comes from the several high quality, single and multicity studies, conducted in children from birth to ages up to 12 years. Regardless of the methodological variations and setting (such as country, length of study) there was causal relationship between long-term NO_2 exposure and respiratory effects for increase in asthma incidence, decrease in lung function and partially irreversible decrease in lung function growth.

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In some studies, the associations were linked with average NO_2 concentrations over multiple years ranging from as low as 14-21ppb (NEPC, 2019; USEPA, 2016).

The NEPC (2019) report also highlighted several meta-analyses that have studied the long-term effects of NO_2 on mortality and morbidity, including the incidence of asthma (Barone-Adesi, 2015; Faustini, 2014;).

- Faustini et al. (2014) conducted a meta-analysis of the long-term studies that were conducted between 2004 and 2013 evaluating the relationship between NO_2 and mortality. The study found that the long-term effects of NO_2 on mortality were as great as that for $PM_{2.5}$. For all cause (non-traumatic) and cardiovascular mortality the effect estimates based on interquartile ranges were greater for NO_2 compared with $PM_{2.5}$ – 6% NO_2 compared with 3% $PM_{2.5}$ for all-cause mortality and 29% for NO_2 compared with 16% for $PM_{2.5}$ for cardiovascular mortality.
- Hoek et al. (2013) conducted a review and meta-analysis of studies to evaluate the association between cardio-respiratory mortality and air pollution. The pooled effect estimate for NO_2 for all-cause mortality was a 5% increase (95% CI 3%, 8%) per 10 μg/m3 increase in annual average NO_2.
- Barone-Adesi et al. (2015) looked at the effects of long-term exposure of traffic related pollutants, including NO_2, and lung function in children. The meta-analysis included 9 studies including the Child Heart and Health Study in England (CHASE). The results of the meta-analysis showed that a 10 μg/m3 increase in long-term NO_2 exposure was associated with a 0.7% decrease in FEV1 in children. The authors concluded that this would translate to an increase of 7% (95% CI: 4% to 12%) in children with abnormal lung function. The observed effect was not modified by a reported asthma diagnosis.

The Australian ACHAPS cross-sectional study showed consistent evidence of respiratory adverse effects of NO_2 for both recent and life-time exposure (SCEW, 2012). These adverse effects are manifested as increased risk of asthma-like symptoms (in particular, wheeze), increased airway inflammation and reduced lung volumes.

## Summary of health effects due to NO_2 pollution

Short-term exposure to NO_2 has been linked to increases in all-cause, cardiovascular and respiratory mortality. The recent studies have provided evidence that has strengthened the association with hospital admissions and emergency department visits for respiratory disease including all respiratory causes, asthma and chronic obstructive pulmonary disease. Studies of children with asthma show associations between NO_2 and reductions in lung function, increases in cough, night-time asthma and school absenteeism. An increase in symptoms in asthmatic children and increases in airway inflammation and hyper-responsiveness have also been observed.

Epidemiological studies of long-term effects of NO_2 exposure on mortality (both respiratory and cardiovascular causes) and on children's respiratory symptoms and lung function also support the conclusion that NO_2 has an independent effect on health. Long-term exposure to

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NO$_2$ has been linked to deficits in lung function growth. There is also strong evidence of an association between long-term exposure to NO$_2$ and the incidence of asthma and wheeze. This new evidence suggests that NO$_2$ exposure may actually cause asthma rather than just exacerbate existing asthma.

The biological health effects that are related to NO$_2$ exposure were diagrammatically summarised by the USEPA. The solid arrows and dark boxes represent pathways for which there is consistent evidence. The dotted lines and white boxes represent uncertain pathways because evidence is limited or inconsistent (USEPA, 2016).

Figure 6: Relationships of short and long-term nitrogen dioxide exposure
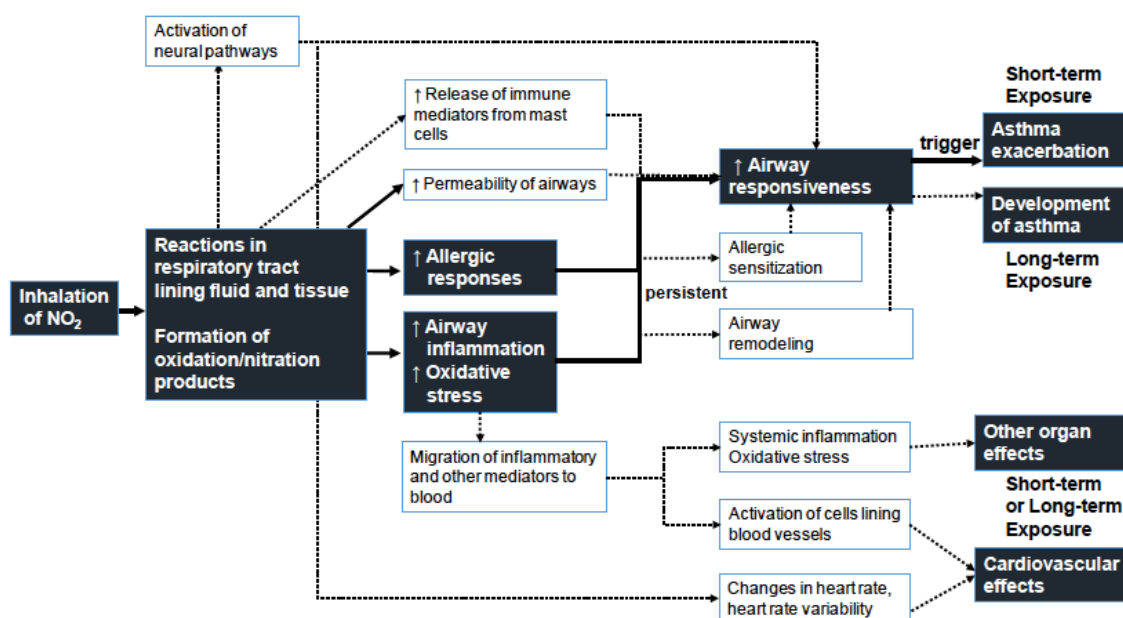
Breathing Ozone (O$_3$) Pollution

Ozone (O$_3$) at ground level, not to be confused with the ozone layer in the upper atmosphere, is one of the major constituents of photochemical smog. It is highly reactive, oxidative gas with a low solubility in water.

The ozone formation mechanism, as shown in figure 2 shows why elevated ozone concentrations are found in an increasing number of places around the world where anthropogenic emissions of NO$_x$, VOCs, and CO have been increasing.

There is a very large volume of literature focusing on the acute and chronic effects of ozone exposure. Furthermore, the short-term and long-term health effects to exposure to O$_3$ in ambient air have been reviewed and summarised by (EPA, 2012; EEA, 2015; COMEAP, 2015; NEPC 2019; USEPA, 2016; WHO, 2013a). The key findings from these policy documents, together with the studies identified in the literature review are summarised below.

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Health effects due to short-term exposure to O_3

Many studies focused on short-term effects of ozone exposure and respiratory health under various conditions such as controlled exposures at rest or during exercise, exposure at various ambient levels, and in subjects with or without pre-existing pulmonary diseases, such as asthma or chronic bronchitis (McDonnell, 2012; NEPC, 2019). Regardless of the setting, the results showed that when people are exposed to higher outdoor ozone levels (outdoor workers, children in summer camps, exercising adults), they experience acute lung function decrement (Brunekreef, 1994; Chan, 2005; Kinney, 1996; Thaller, 2008). In the 2013 review, the USEPA concluded that exposure to O_3 as low as 60-70ppb is associated with a statistically significant group mean decrease in pulmonary function in healthy adults (USEPA, 2013).

Results from epidemiological studies also show robust, positive and significant associations between fluctuations in ambient ozone levels and increased morbidity. Different large multicity analyses at varying O_3 levels have examined hospital admissions or emergency department visits for asthma, respiratory tract infections and exacerbation of other respiratory diseases, school absenteeism. Associations between morbidity and O_3 levels were frequently observed in children, elderly people, asthmatics and people with COPD, especially during the warmer months (Nuvolone, 2018).

Two large multicentre studies: the Air Pollution and Health: a European and North American Approach study (Katsouyanni, 2009) and the Public Health and Air Pollution in Asia study found that short-term exposure to O_3 increases respiratory mortality (HEI 2010, 2011; Wong, 2010).

Nuvolone et al. found a 6.3% (95% CI 1.2–11.7%) increase in out-of-hospital coronary deaths (cardiovascular mortality) for a 10 µg/m3 increase in ozone, suggesting higher risks for females, elderly and patients that have previously been hospitalized for cerebrovascular and artery diseases.

Health effects due to long-term exposure to O_3

Effects respiratory morbidity and long-term exposure to O_3, such as increase in asthma incidence, asthma severity, hospital care for asthma and lung function growth have been summarised in many policy reports (NEPC, 2019; USEPA, 2013; WHO, 2013a). Locally, two multi-city studies conducted in Australia reported a positive association between increase in O_3 levels of 1ppb and respiratory admissions (Barnett, 2005; Simpson, 2005a). A few other international cohort and epidemiological studies also found consistent and significant associations between reduced airway function and long-term ozone exposure (Ihorst, 2004; Künzli, 1997; Tager, 2005).

Multi-community prospective cohort studies demonstrated that long-term exposures to O_3 is also associated with new onset of asthma in children as well as increases in respiratory symptom effects in children with asthma (Chen, 2016; McConnell; 2002; McDonnell, 1999; Peters, 1999). In the Children's Health Study, McConnell et al (2002) prospectively followed

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more than 3500 non-asthmatic children aged 9–16 from 1993 to 1998 in the US. The results showed that in communities with high levels of ozone, there was significant association between developing asthma and the number of hours spent outside, with a relative risk of 3.3 (range 1.9–5.8). In another US study, 3091 non-smoking adults were followed for 15-year follow-up period and a 20-year ozone exposure history was assigned to each participant. Asthma onset was significantly associated with long-term ozone increase (McDonnell, 1999). A more recent study conducted in Adelaide by Chen et al, found that the largest effect for O_3 was in the warm season, with a 5-day cumulative effect on an 11.7% increase in the risk of asthma hospital admissions in children ages 0-17 years for every 10ppb increment in O_3 (Chen, 2016).

Atkinson et al. recently published a systematic review on the effects of ozone on long-term mortality, and concluded that there was a suggestive, positive association between long-term ozone exposure, especially during warmer months, and cardiopulmonary and respiratory mortality (Atkinson, 2016).

Summary of the health effects due to O_3 pollution

Human exposure to O_3 occurs almost exclusively by inhalation, and evidence shows that is a well-established respiratory irritant. Because of its low solubility in water, ozone is not effectively removed by the upper respiratory tract. Therefore, once inhaled it can cause formation of secondary oxidation in the lung, inflammation, alteration of epithelial barrier function, sensitisation of bronchial smooth muscle, changes in immunity and airway remodelling.

Short-term exposure to O_3 can cause adverse respiratory effects such as difficulty of breathing (e.g., shortness of breath and pain when taking a deep breath) and inflammation of the airways. There is also an association between short-term O_3 exposure and cardiovascular and respiratory mortality, as well as cardiovascular and respiratory hospital admissions (COMEAP, 2015; USEPA, 2013; WHO, 2013a).

Long-term exposure to O_3 is likely to be one of many causes of asthma development. Evidence shows that exposure to O_3 can also aggravate pre-existing lung diseases such as asthma, emphysema, and chronic bronchitis/COPD (Nuvolone, 2018).

Breathing Particulate Matter (PM$_{10}$ and PM$_{2.5}$)

Particulate matter (PM) usually consists of complex solid and liquid particles of organic and inorganic substance suspended in the air, with the major components being nitrates, sulphates, polycyclic aromatic hydrocarbons, endotoxin, and metals such as iron, copper, nickel, zinc, and vanadium. PM is subclassified according to particle size into:

- coarse (PM$_{10}$, diameter <10μm),
- fine (PM$_{2.5}$, diameter <2.5μm), and

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- ultrafine ($PM_{0.1}$, diameter <0.1µm).

Coarse particles, PM_{10} and above, generally do not penetrate deep into the lungs. However, fine and ultrafine particles, generally produced from combustion of fossil fuels and other industrial activities, are a greater threat to human health. For they can penetrate into the small airways and alveoli and lodge themselves deep inside the lungs.

There is a very large volume of literature focusing on the acute and chronic effects of PM_{10} and $PM_{2.5}$. With new and emerging evidence from animal studies, human controlled studies, observational and epidemiological analysis, this topic is growing at a very rapid pace.

PM particulate matter has been listed as a carcinogen by the International Agency for Research on Cancer, and a global assessment of mortality estimated $PM_{2.5}$ as the fifth-ranking mortality risk factor, leading to 4.2 million deaths and 103.1 million disability-adjusted life-years in 2015.

It is now a well-accepted fact that there is no "safe" threshold under which increases in PM are not associated with increased death. The key findings from policy documents, together with the studies identified in the literature review are summarised below.

Health effects due to short term exposure to PM_{10} and $PM_{2.5}$

There is a large amount of research in short-term exposure to $PM_{2.5}$ and adverse health effects.

Atkinson et al. conducted a comprehensive and systematic meta-analysis of 110 peer-reviewed studies and found positive correlation between adverse health effects of short-term exposure to $PM_{2.5}$ across a range of respiratory and cardiovascular health outcomes, diseases and age groups. Their review found that short-term exposure to $PM_{2.5}$ was associated:

- with asthma and respiratory hospital admissions in children under the age of 14 years;
- cardiovascular and respiratory hospitalisations in people aged 65 years and over.

The summary estimates for $PM_{2.5}$ exposure and specific cardiovascular and respiratory disease in ages 65+ years, and children aged 01-14 years are shown in figure 7 (Atkinson, 2014).

Another meta-analysis of data from 36 panel studies observed that short-term exposure to PM air pollution was associated with episodes of asthma symptoms in asthmatic children (Weinmayr, 2010).

Several large, multi-city studies have been conducted in the US and Europe, such as the National Morbidity, Mortality, and the Air Pollution Study and the Air Pollution and Health: A European Approach. Regardless of the seasonal and regional variations, sources of pollutants, meteorological conditions and population differences, they findings from these studies were remarkably consistent. They show that PM levels are significantly associated with daily all-cause cardiovascular and respiratory mortality (Bell, 2008; Katsouyanni, 1997, 2001; Peng, 2005).

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Hamanaka et al, analysed the results of the multi-city studies, and suggested that there is no "safe" threshold under which increases in PM are not associated with increased death (Hamanaka, 2018).

Figure 7: Summary estimates (95% confidence intervals) for cardiovascular and respiratory hospital admissions and PM$_{2.5}$ concentrations (Atkinson, 2014)
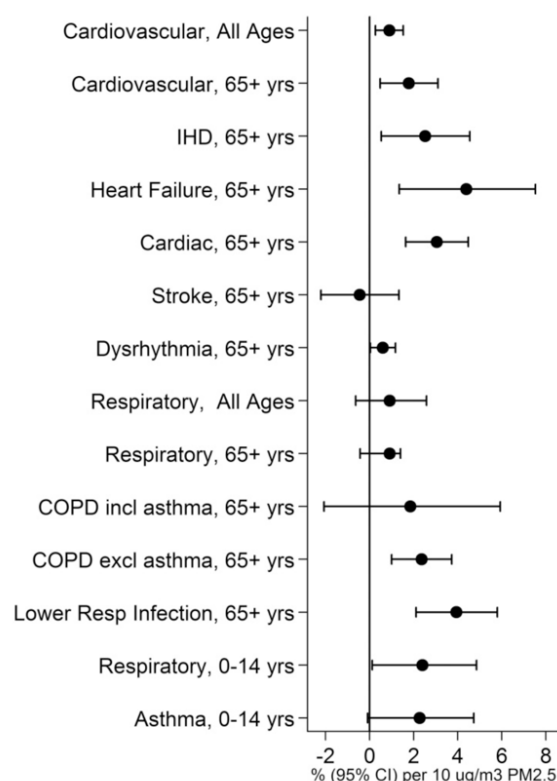

To better understand how PM$_{2.5}$ is associated with cardiovascular symptoms, Hamanaka et al. in 2018, conducted a review of over 200 articles, and identified three main hypotheses by which PM-exposure could exert its biological effects in cardiovascular disease.

- Their first hypothesis proposes that PM inhalation activates inflammatory responses in the lung leading to a "spill over" effect and systemic inflammation, which promotes thrombosis, endothelial dysfunction, and atherosclerosis.
- Their second hypothesis suggests that inhaled PM activates sensory receptors in the lung, leading to imbalance of the autonomic nervous system, favouring sympathetic pathways and leading to alterations in heart rate, vasoconstriction, endothelial dysfunction, and hypertension.
- Their third hypothesis proposes that some particles, particularly ultrafine particles (PM$_{0.1}$) can enter the circulation from the lung and interact directly with target tissues.

Hamanaka et al. summarised their research into an infographic (figure 8), highlighting that regardless of which hypothesis stands true, the short-term exposure to PM$_{2.5}$ is associated with clinical indicators such as acute heart failure, acute myocardial infarction, ischemic stroke, venous thromboembolic disease and cardiac arrhythmias (Hamanaka, 2018).

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Figure 8: Current evidence for the mechanisms by which particulate matter air pollution causes cardiovascular health effects.

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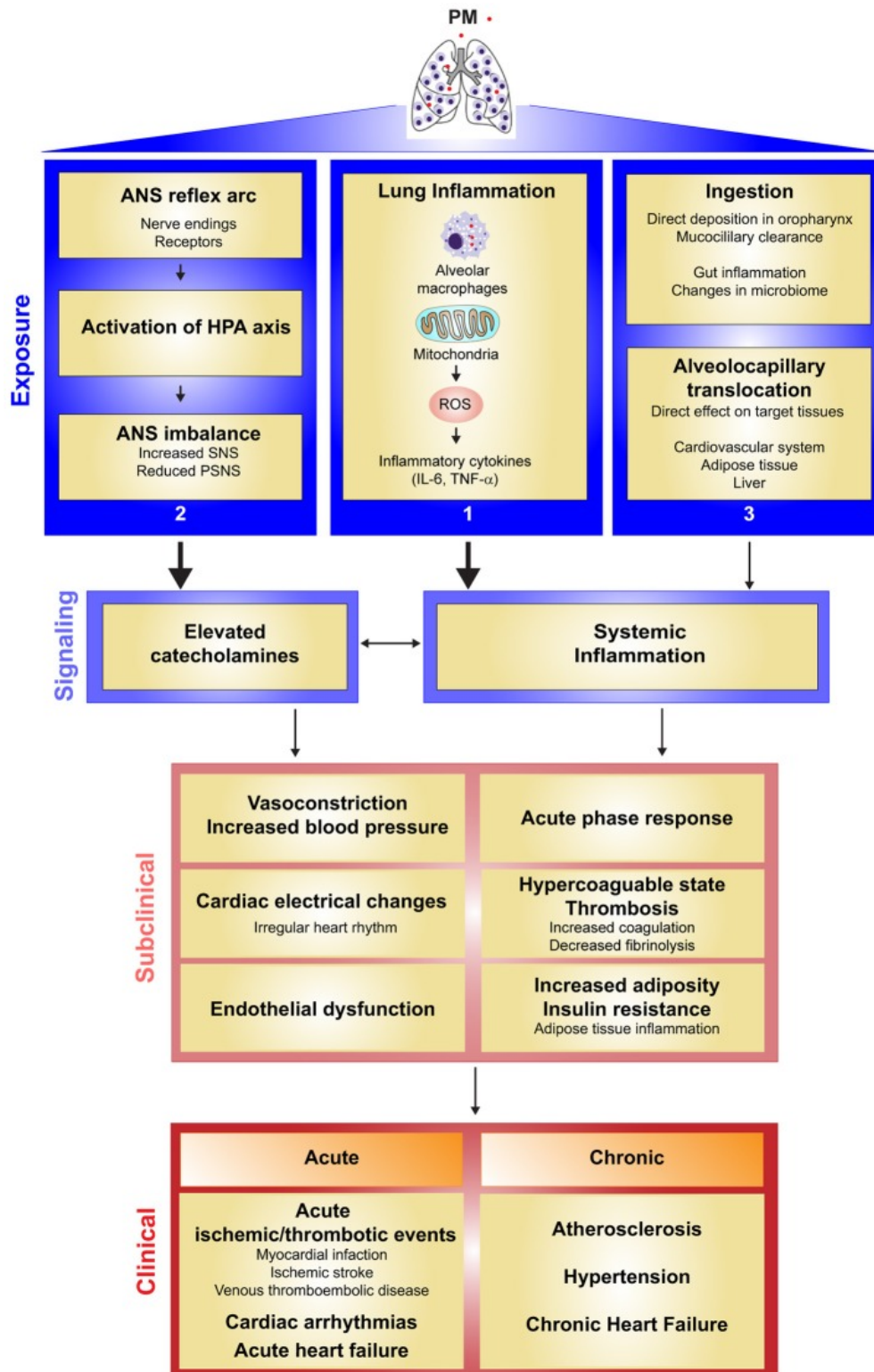
Health effects due to long term exposure to PM$_{10}$ and PM$_{2.5}$

Long-term exposure to PM$_{2.5}$ is strongly associated with respiratory and cardiovascular morbidity and mortality (Hime, 2018). A global assessment of mortality, carried out the Global Burden of Diseases, Injuries, and Risk Factors Study 2015 estimated that PM$_{2.5}$ is the fifth-ranking mortality risk factor, leading to 4.2 million deaths and 103.1 million disability-adjusted life-years in 2015 (Cohen, 2018).

Data from three European cohort studies show evidence that long-term exposure to PM is associated with asthma prevalence (Cai, 2017). The Framingham Heart Study cohort in the US also observed an association between PM and decreased lung function in children and adults (Rice, 2015). This finding was also observed in cohorts participating in the multi-country European Study of Cohorts for Air Pollution Effects (ESCAPE) project, which found that long-term exposure to PM air pollution is associated with:

* decreased lung function in children (Gehring, 2013)
* decreased lung function in adults (Adam, 2015)
* prevalence and incidence of chronic obstructive pulmonary disease (Schikowski, 2014) and
* with childhood pneumonia (MacIntyre, 2014).

In the ACS Cancer Prevention II, both PM$_{2.5}$ and SO$_2$ were positively correlated with all-cause, lung cancer, and cardiopulmonary mortality. Every 10µg/cm3 increase in PM$_{2.5}$ was associated with a 4, 6 and 8% increase in risk of all-cause, cardiopulmonary, and lung cancer mortality, respectively. These findings were similar to those of the Harvard Six Cities Study, where PM$_{2.5}$ (and SO$_2$) was positively associated with death from lung cancer and cardiopulmonary diseases (Hime, 2018; Smith, 2009).

Many other observation studies and pooled reviews show that long-term exposure to PM air pollution is strongly associated with the incidence of acute coronary events (myocardial infarction and unstable angina), stroke and heart failure as seen in figure 8 (Atkinson, 2014; Hamanaka, 2018, Hime, 2018, Nawrot, 2011).

Thurston et al found that exposure to PM air pollution include exacerbations of COPD, impaired vascular function, high blood pressure, stroke, myocardial infarction and neurological diseases such as Alzheimer's, Parkinson's disease and diabetes (Thurston, 2017, WHO, 2013). These results were confirmed by Wei et al, when they conducted indepth case crossover analysis with conditional logistic regression of hospital admissions and associated PM$_{2.5}$ exposure.

Emerging evidence also suggests that PM exposure affects timing of puberty and reproductive health in both men and women, and low-term birth weight (Hamanaka, 2018; Liu, 2019; Pederson, 2013)

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Summary of health effects due to PM$_{10}$ and PM$_{2.5}$ pollution

The International Agency for Research on Cancer classified PM's as carcinogenic (IARC, 2013).

Short-term exposure to PM$_{10}$ can cause mild to moderate respiratory distress such as shortness of breath, pain when inhaling deeply, wheezing, coughing, eye and nose irritation. Depending of the person's vulnerability, short-term exposure to PM could also inflame breathing passages, decrease the lungs working capacity and increase susceptibility to illness.

Short-term exposure to PM$_{2.5}$ is more serious, and is associated with:

- respiratory morbidity (such as aggravation of asthma, respiratory symptoms and increase in hospital admissions) and mortality.
- cardiovascular morbidly and mortality such as acute heart failure, acute myocardial infarction, ischemic stroke, venous thromboembolic disease and cardiac arrhythmias.

Long-term exposure to PM is a major contributor to respiratory and cardiovascular morbidity and mortality. A global assessment of mortality estimated that PM$_{2.5}$ is the fifth-ranking mortality risk factor, leading to 4.2 million deaths and 103.1 million disability-adjusted life-years in 2015.

In terms of respiratory effects, evidence shows that long-term exposure to PM can cause decreased lung function in children and adults, affect lung developments in children, increased prevalence and incidence of chronic obstructive pulmonary disease and childhood pneumonia.

In terms of cardiovascular morbidity and mortality, evidence shows that long-term exposure to PM can cause high blood pressure, stroke and myocardial infarction.

There is also new evidence to show association between long-term exposure to PM and neurological diseases such as Alzheimer's and Parkinson's disease.

It now well accepted that the health effects of PM are not only dependent on exposure, but also on vulnerability of the people. Vulnerability could be as a result of age, pre-existing health conditions or underlying asymptomatic factors.

Breathing Volatile Organic Compound Pollution

VOCs are an important group of air pollutants, and are often referred to as toxic or hazardous air pollutants. The term VOC is used for both indoor and outdoor air quality, and the term is defined differently to reflect its predominant concern in each context. They play an important role in the formation of O$_3$ and PM$_{2.5}$ by photochemical smog reaction.

The VOC's benzene, formaldehyde, benzo(a)pyrene (as a marker for Polycyclic Aromatic Hydrocarbons (PAH)), toluene and xylene are regulated using the Air Toxins NEPMs in Australia.

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Health effects due to short term exposure to VOCs

The effects of benzene, toluene, xylenes, formaldehyde and PAHs on health are well documented.

Inhaling VOCs will cause short-term health effects in most people. The most common reactions will be term localised such as irritation of the eyes, nose and throat, and more systemic sides effects such as headaches, loss of coordination and nausea. Evidence shows that exposure to VOC could also trigger allergic skin reactions in some people (USEPA, WHO 2000).

The reaction varies from person to person, either due to individual response, or due to level and length of exposure.

Health effects due to long term exposure to VOCs

Long-term exposure to ambient VOCs contributes to some of the most serious health-related impacts such as cancer, neurological effects, respiratory irritation and eye irritation.

Benzene is classified as a known human carcinogen. It is considered to be a genotoxic carcinogen for which no safe threshold has been established (US EPA 2000, WHO 2000). The critical human health effects from long term exposure to benzene are bone marrow depression and leukaemia, specifically acute non-lymphocytic leukaemia (also known as acute myeloid leukaemia).

There is strong evidence to show that long-term exposure to toluene and xylene could result in serious neurosis, Chronic toluene exposure leads to devastating neurological disorders, such as dementia. Other studies have linked evidence that that exposure to toluene could cause vision impairment. Similarly, evidence shows that long-term exposure to xylene may cause neurological side effects such as headaches, extreme tiredness, tremors, impaired concentration and short-term memory (Singh, 2018).

While one the key complexities in evaluating the health effects of PAHs is that they exist as a mixture of compounds not individual compounds, several epidemiological studies have shown increased association of mortality due to cancer due to PAH exposure.

Summary of health effects due to VOC pollution

Short-term exposure to VOC can cause irritation of the eyes and respiratory tract, headaches, dizziness, visual disorders and memory impairment.

However, exposure to in high concentrations and for extended periods of time can have longer term health effects, such as prolonged eye, nose and throat irritation; chronic headaches as well as loss of coordination and chronic nausea; liver, kidney and central nervous system damage. Some VOCs are carcinogenic.

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Key findings from Literature Review

There was an extensive international body of literature on the health impacts of air pollution, reporting a wide range of adverse health outcomes including: worsening of asthma and chronic lung disease, increasing risks of heart attack, stroke and lung cancer, and affecting lung development. While some studies from Australia were identified, and they have been reviewed in the appropriate sections, it was evident that the field of public health and air pollution research is still emerging.

A compressive global health review conducted by the Forum of International Respiratory Societies' Environmental Committee, Part 1: The Damaging Effects of Air Pollution summarised the mechanisms by which different pollutants cause tissue damage. Their data is summarised in table 5 (Schraufnagel, 2019).

Table 5: How Different Types of Air Pollution Damage Tissue

Pollutant	Injury Determinants	Tissue Affected
Sulphur dioxide	Highly soluble	Upper airway and skin damage
Nitrogen dioxide and Ozone	Less soluble (nitrogen dioxide and ozone are irritating)	Deeper lung penetration Bronchial and bronchiolar injury
Particulate matter (PM10, PM2.5, PM0.1)	Size, structure, and composition determine toxicity	Course PM10: mucous membranes, upper airways. Fine PM2.5 bronchioles and alveoli Ultrafine PM0.1: systemic tissue reactions

$PM_{0.1}$ = particulate matter with an aerodynamic diameter < 0.1 μm; $PM_{2.5}$ = particulate matter with an aerodynamic diameter < 2.5 μm; PM_{10} = particulate matter with an aerodynamic diameter < 10 μm.

The key research and evidence identified in the literature review was themed and is summarised in table 6 to show the health outcomes of ambient air pollution.

Table 6: Health outcomes of ambient air pollution

Ambient air pollution	General air pollution	Ozone (O_3), Sulphur dioxide (SO_2), Nitrogen Dioxide (NO_2)
	Fine particle matter	Particulate matter (PM10, PM2.5), VOCs
Health Outcomes	Respiratory disease	Asthma, respiratory infections, respiratory disorders, chronic obstructive pulmonary disease, lung cancer
	Cardiovascular disease	Hypertension, heart rate variability, heart attack, ischaemic heart disease, stroke,
	Chronic disease	Diabetes, dementia, cancer
	Health records	Morbidity, hospital admissions, outpatient visits, emergency room visits, sick days, mortality
	Most vulnerable	Children, elderly, existing chronic health condition

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Air pollution is carcinogenic. There is no safe level of air pollution. The findings of the literature review show that:

- SO_2 levels as low as 10ppb (24-hour average) might cause respiratory and cardiovascular mortality;
- Long-term exposure to NO_2 has been linked to deficits in lung function growth;
- NO_2 levels as low 14-2ppb (annual average) in a child's first year of life might increase their chance of getting asthma;
- O_3 levels as low as 60-70ppb might decrease pulmonary function in healthy adults;
- Long-term exposure to O_3 is likely to cause asthma development;
- $PM_{2.5}$ is carcinogenic and it is the fifth-ranking mortality risk factor in the world, with an estimated 4.2 million deaths and 103.1 million disability-adjusted life-years in 2015.
- Short-term exposure to $PM_{2.5}$ is more serious. It can aggravate asthma and other respiratory symptom, leading to increased hospital admissions. It can also cause cardiovascular morbidly and mortality such as acute heart failure, acute myocardial infarction, ischemic stroke, venous thromboembolic disease and cardiac arrhythmias.
- Some VOC are carcinogenic. Exposure to high concentrations of VOCs, and for extended periods of time, could lead to increase in chronic headaches, loss of coordination, chronic nausea; liver, kidney and central nervous system damage.

The short-term and long-term exposure to ambient SO_2, NO_2, O_3, PM_{10}, $PM_{2.5}$ and VOCs were found to be strongly associated with:

- Increase in daily mortality
- Increase in emergency room and/or hospital admissions for:
    - Respiratory causes such as wheezing, exacerbation of asthma, respiratory infections, decline in respiratory function, COPD; and
    - Cardiovascular causes such as high blood pressure, chest pain, thrombotic events, arrhythmias, ischemic heart failure.

Children, the elderly and those with existing cardiovascular and respiratory conditions are particularly vulnerable to the effects of ambient air pollution, and that their health burden is not dependent on amount of pollution exposure, but on their individual respiratory and cardiovascular response.

The people living and working in areas with higher than average air ambient pollution suffer greater health injustice, with more:

- days off school/work;
- medical expenditures;
- health service utilisation such as doctors' visits, outpatient visits, emergency room visits, hospitalisation; and even death.

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LIVING WITH UNCERTAINITY IN THE BURRUP PENINSULA

Almost 23,000 people residing in the City of Karratha are at risk from exposure to the air pollution from industries on the Burrup Peninsula.

The literature review confirms that both short-term and long-term exposure to ambient SO_2, NO_2, O_3, PM_{10}, $PM_{2.5}$ and VOCs are strongly associated with:

- increase in daily respiratory and cardiovascular mortality;
- increase in emergency room and/or hospital admissions for:
    - respiratory causes such as wheezing, exacerbation of asthma, respiratory infections, decline in respiratory function, COPD; and
    - cardiovascular causes such as high blood pressure, chest pain, thrombotic events, arrhythmias, ischemic heart failure.

Children, the elderly and those with existing cardiovascular and respiratory conditions are more vulnerable to the effects of ambient air pollution; and their health burden is not dependent on amount of pollution they breathe, but instead it depends on their individual respiratory and cardiovascular response.

It is for this reason; the general advice is that there is no safe levels of air pollution.

Health vulnerability in the Burrup Peninsula

Children: almost a third of the population in the City of Karratha is under the age of 20 years. They are more vulnerable to respiratory distress from ambient air pollution because they breathe more air (and toxins) per unit body weight.

The 2011-2015 Pilbara Health Profile report showed that children living in the Pilbara had 1.7-11 times more hospitalisations for asthma and bronchiectasis (respectively) when compared to the children in the rest of WA.

The findings from the literature review confirm that short-term and long-term exposure to SO_2, NO_2, O_3, and $PM_{2.5}$ can:

- exacerbate the symptoms of asthma (wheezing, breathlessness, increase in night-time cough, decreased lung function) in children;
- increase the risk of hospital admission for asthma and/or respiratory symptoms in in children, the elderly and those with existing conditions; and
- long-term exposure to NO_2 and O_3 could cause new cases of asthma.

Elderly: only a very small percentage of the population (5%) stay back to live and retire in the City of Karratha once they turn 60 years of age. Therefore, the actually morbidity and mortality data due to air pollution related symptoms cannot fully be determined.

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The 2011-2015 Pilbara Health Profile report showed that COPD was the most frequent preventable cause for hospitalisation. It accounted for almost a third of all PPH's in this age group, and was nearly three times the WA average.

The findings from the literature review confirm that short-term and long-term exposure to SO_2, NO_2, O_3, and $PM_{2.5}$ can:

- exacerbate the symptoms of asthma in the elderly;
- increase the risk of hospital admission for asthma, COPD and respiratory symptoms in the elderly; and
- increase the risk of hospital admissions for cardiovascular symptoms in the elderly.

General public health duty

The General Public Health Duty (section 34) of the Public Health Act of Western Australia, 2016 states that *"all reasonable and practicable steps to prevent or minimise any harm to public health that might foreseeably result from anything done or omitted to be done by the person"*.

Since the 1960's a number of heavy industries that rely on or extract fossil fuels, or manufacture agricultural products have been approved and developed in the Burrup Peninsula.

Individually and collectively, they create and release significant point-source air pollution, including SO_2, NO_2, O_3, PM_{10}, $PM_{2.5}$ and VOCs, into the Burrup air-shed. In the coming years, there will be new proposals to expand the LNG facilities, as well as create new industries. They too will also release their point-source emissions into the same air-shed.

Despite large-scale industrialisation of the area, and population proximity living and working in the Burrup air-shed, DWER have not installed long-term open-source ambient air quality monitors in the area. Industry-funded monitors, such as the Woodside BAAMP data, do not make their data available to anyone.

Therefore, the true nature, extent and duration of health burden caused by short-term or long-term pollution exceedances from the multiple industries is unknown.

The omission of general air pollution public health duty in industrial air-sheds is not unique to the Burrup Peninsula.

The case-study below, using Woodside BAAMP, is just one of many just such examples, that highlights the inadequacy in existing relationships between industry, governing bodies and health services.

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CRITICAL REFLECTION OF WOODSIDE BAAMP AND THE NWS EXTENSION AIR QUALITY IMPACT ASSESSMENT

DWER's Air Quality Management Program for Air NEMP's does not extend to the Burrup air-shed.

In 2008 Woodside established the Burrup Ambient Air Monitoring Program (BAAMP) to determine the ambient air quality in Karratha and Dampier. The BAAMP continued until 2015, as part of the Pluto license (Jacobs, 2019). However, their ambient air quality monitoring data was not accessible to anyone.

In December 2019, Woodside submitted their NWS Project Extension Air Quality Impact Assessment as part of their Environmental Review Document to the EPA (Jacobs, 2019)

In reviewing the information provided by Jacobs *et al.* in Appendix E of the NWS Extension Air Quality Impact Assessment document to the EPA; my critical assessment concludes that the information provided by Woodside is outdated and distorted.

Information provided to the EPA is outdated

The BAAMP data from 2009-2015 is presented by Woodside in their Air Quality Impact Assessment. There is no data to show the quality of ambient air in Karratha and Dampier since 2015. Therefore, this dataset is four years out-dated.

Woodside have used the 2014 dataset for assessing air quality and modelling studies for the fifty-year NWS extension i.e. until 2070.

The National Pollutant Inventory shows that Woodside have increased their NO_2 point-source emissions since 2014-2015, and will continue to increase their emissions (figure 9).

Figure 9: Increases in oxides of nitrogen emission from the Pluto Onshore Processing Plant between 2015-2018. Data obtained from National Pollutant Inventory.

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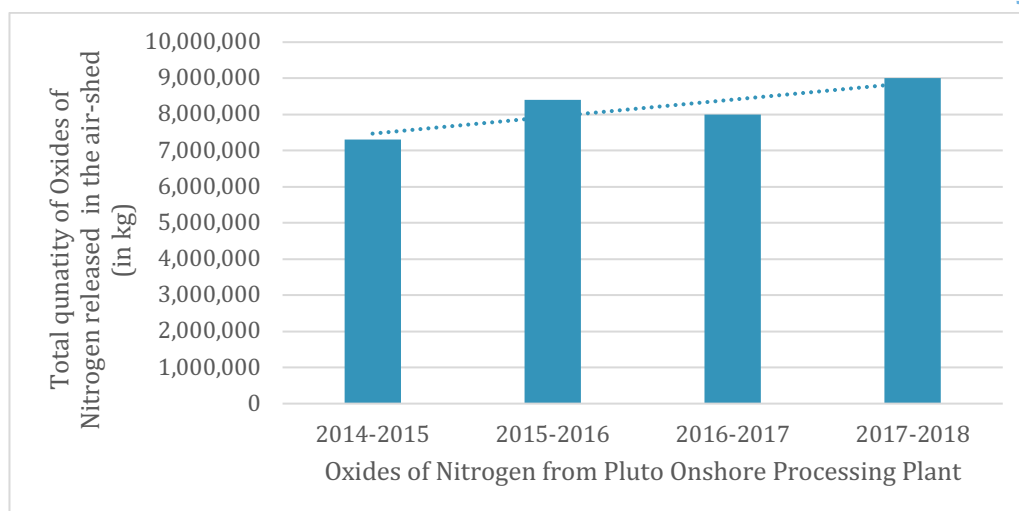
However, Woodside have omitted to mention the annual increase in point-source NOx, and consequent increase in ambient NO_2 pollution between 2014-2018 in their modelling and future projections to the EPA.

Q. What are the predicted increases in NOx emissions between 2020-2070 in the NWS project?

Q. What are the predicted cumulative increases in NOx emissions from all Woodside projects in the Burrup Peninsula between 2020-2070?

Information provided to the EPA is distorted

Woodside have not released the actual BAAMP datasets containing the 1-hour or 4-hour emissions data. Instead, Jacobs *et al.* state on pg 356 that they re-analysed all the BAAMP data capture for this document.

International standards for air quality highlight that for strict comparison, data capture should be >90% over a calendar year.

Tables 3.1, 3.2 and 3.2 on pg 357 show a number of NO_2 data capture sets less than 90%, and in case of O_3 missing and thus indicated by ND (No Data) at Burrup Road (as shown in figure 10).

Figure 10: Yellow highlight shows data capture sets less than 90% in Appendix E of the NWS Extension proposal to the EPA.

Table 3-1: Karratha Air Quality Monitoring – Data Capture NO_2 and O_3

Substance	2009	2010	2011	2012	2013	2014	2015
NO_2	91.8%	93.1%	92.4%	94.8%	94.4%	91.5%	94.6%
O_3	70.8% (year) 94% (1 April to 31 Dec)	94.3%	90.6%	90.1%	91.3%	89.0%	91.2%

Table 3-2: Dampier Air Quality Monitoring Results – Data Capture NO_2 and O_3

Substance	2009	2010	2011	2012	2013	2014	2015
NO_2	89.2%	86.9%	86.9%	87.4%	92.2%	89.6%	92.4%
O_3	3% (year) 51% (10 Dec to 31 Dec)	90.9%	95.4%	94.5%	95.3%	92.5%	95.9%

Table 3-3: Burrup Road Air Quality Monitoring Results – Data Capture NO_2 and O_3

Substance	2009	2010	2011	2012	2013	2014	2015
NO_2	82.7%	91.5%	84.0%	88.4%	94.7%	92.6%	91.3%
O_3	8.8% (year) 94.3% (24 Oct to 27 Nov.)	ND	ND	ND	ND	ND	ND

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Without actual 1-hour or 4-hour emissions datasets, it is not possible to determine if, or why, the data capture for NO_2 in Dampier and Burrup was less than 90%.

Q. Were all the data-sets included when Jacobs *et al* 're-analysed' the data for this report?

Q. If no, would this affect the average and 99th percentile data shown in Figure 3.2 and 3.3 on pg. 358-359?

The new proposed draft variations to NO_2 NEPMS state that 1-hourly limit should not exceed 80ppb; and the annual average should not exceed 15ppb. These variations were suggested because WHO and the USEPA clearly state that NO_2 exposure to ambient levels as low as (14-21ppb) may actually cause asthma rather than just exacerbate existing asthma and symptoms such as wheezing.

Woodside have not revealed the annual averages for NO_2 between 2009-2015 in their NWS Project Extension Proposal.

Q. What were the annual averages of NO_2 between 2009-2015 at all three monitoring sites?

Q. Were there any exceedances in the annual averages of NO_2 between 2009-2015 at all three monitoring sites? If yes, how many and by how much?

Issues with O_3 reporting in the NWS Project Extension Proposal

Woodside have presented the annual averages for the 1-hourly O_3 data capture at Dampier and Karratha stations. They have not disclosed the O_3 levels at Burrup Road in their NWS Project Extension Proposal, as shown in table 3.3 on pg. 357.

The WA Department of Environment Regulation Air Quality Modelling Guidance Notes (DWER, 2006), outlines the expectations for air quality assessment. It states that:

"cumulative impacts must be considered and the assessment must include "existing concentrations caused by other sources plus (if significant) the background concentration (whether man made or natural)".

On pg. 360 Jacobs *et al*. explains that there were high levels O_3 on Burrup Road. Yet Woodside does not disclose the exact amount NWS Project Extension Proposal

Q. What was the actual O_3 data from between 2009-2015 from the Burrup Road station?

The new draft variations to O_3 NEPMS state that the limit on 1 hour and 4 hours should be removed, and should instead focus on 8-hourly reporting, with the limit set to 65ppb. This is because evidence shows that O_3 is a well-established respiratory irritant, and long-term

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exposure can cause asthma, as well as aggravate pre-existing lung diseases such as asthma, emphysema, and chronic bronchitis/COPD.

O_3 has a low solubility in water, and therefore it is not effectively removed by the upper respiratory tract. Therefore, once inhaled it can cause formation of secondary oxidation in the lung, inflammation, alteration of epithelial barrier function, sensitisation of bronchial smooth muscle, changes in immunity and airway remodelling.

Woodside have not revealed the 8 hourly O_3 data.

Q. What was the 8-hourly O_3 data between 2009-2015 at the three monitoring stations?

Q. Were there any exceedances in the NO_2 between 2009-2015 at all three monitoring sites? If yes, how many and by how much?

Issues with PM_{10} and $PM_{2.5}$ reporting in the NWS Project Extension Proposal

In pg. 362 of the NWS Extension Project Extension Proposal, Woodside acknowledge the cumulative PM_{10} and $PM_{2.5}$ burden in the Burrup air-shed, including the exceedances in Dampier and Karratha. They allocate these exceedances to dust storms, bushfires and other industries in the Burrup air-shed.

However, they have not provided in the submission any of the PM_{10} and $PM_{2.5}$ data from BAAMP. The WHO clearly asserts that there is no safe level of expose to $PM_{2.5}$ (WHO, 2006). For every increase of 10 µg/m^3 in $PM_{2.5}$ concentration, there is an increase of six percent in cardiopulmonary disease, and eight percent in lung cancer mortality.

Q. What was the 24-hour and 1-year PM_{10} and $PM_{2.5}$ data between 2009-2015 at the three monitoring stations?

Q. Where there any exceedances in 1-year PM_{10} and $PM_{2.5}$ data between 2009-2015 at the three monitoring stations? If yes, how many and by how much?

Precise location coordinates and height of BAAMP monitors

Page 440 of NWS Project Extension Proposal shows the geographical location of the BAAMP.

- the precise location coordinates of the BAAMP air quality monitors are not disclosed;
- the monitors are located further away from the residential town centres, and in high prevailing wind conditions; and
- the heights of air quality monitors have also not been disclosed.

Q. What were the precise location coordinates for the three BAAMP stations?

Q. What height above ground was the air monitored at the three BAAMP stations?

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DWER (WA) are responsible for open-source ambient quality monitoring in some parts of Western Australia. However, not all air-sheds are monitored, even though some contain multiple large-scale industries with significant point source or area-based emissions; e.g. the Burrup Peninsula.

The evidence from the literature review highlights that there is no safe level of air pollution. While some groups, such as children, elderly people, pregnant women and those with pre-existing health problems, are more vulnerable, everyone exposed may be affected.

Their health burden will not be directly proportional to amount of pollution exposure. It will be unpredictable; because it depends on each individual's respiratory and cardiovascular response.

Therefore, whether the 23,000 people living and working in the Burrup air-shed endure occasional short-term exceedances, or long-term ambient air pollution; they will face an altered life experience.

From birth, through adolescence and beyond, the cumulative air pollutants SO_2, NO_2, O_3, PM_{10} and $PM_{2.5}$ will damage their heart, their lungs and other vital organs in their body.

This health injustice will be seen in more tangible burdens such as:

- more days off school/work;
- greater medical expenditures from increased incidence of respiratory and cardiovascular disease;
- more health service utilisation such as doctors' visits, outpatient visits, emergency room visits, hospitalisation; and
- maybe even death.

To date, the efforts to monitor ambient air quality in the Burrup Peninsula have been either inconsistence, or restricted. DWER have not installed any open-source ambient air quality monitors in the Burrup air-shed. Industry-funded ambient air quality monitors, such as the Woodside BAAMP, restrict public access to their air quality data.

Therefore, the nature, extent and duration of harm caused by short-term or long-term exceedances from all the cumulative polluting industries in the Burrup air-shed is unknown.

There is no overarching public health responsibility between polluting industries, DWER and DoH when it comes to protecting human health. This is a significant omission of responsibility, according to the Section 34: General Public Health Duty of the Public Health Act of WA, 2016.

While uncoordinated and fragmented approaches between industries and Government agencies have been normal practice to-date (business-as-usual), we face unprecedented challenges as the climate impacts reach multiple and simultaneous tipping points.

There is an urgent need to resolve the General Public Health Duty omissions, and start addressing the cumulative impacts of air pollution in the Burrup air-shed.

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RECOMMENDATIONS

We call that no new polluting licenses be issued in the Burrup Peninsula until there is:

1. An Environmental Protection Policy for the Burrup Peninsula. This includes:

    - extending the DWER Air Quality Management Program for Air NEPMS in the Burrup air-shed; and
    - installing a network of DWER open-source air quality monitors in the towns of Karratha and Dampier to accurately assess the ambient air quality.

2. A comprehensive Health Impact Assessment of all the Air NEPMS and Air-Toxins NEMPS in the Burrup air-shed.

3. Open source access to all the Woodside and BAAMP datasets (past, present and future).

4. An effective and responsive public health management plan for the Burrup Peninsula. This includes:

    - Updating and upskilling all public health and medical health services and service-providers, so that they can moderate, mitigate, document and plan their responsiveness to any short-term exceedances and long-term ambient pollution health risks.

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