Human Health Risk Assessment

Portland Energy Recovery Facility

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Human Health Risk Assessment

Portland Energy Recovery Facility

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1. OVERALL HEALTH CONTEXT

1.1 The Alternatives

Waste management arrangements are intended to separate and collect for recovery as much useful waste material as possible, including recyclable and compostable components. However, Government believes that for the foreseeable future there will always be some residual waste that requires management after levels of recycling and composting have been maximised.

For this material, there are essentially only two treatment options: recovery; and disposal to landfill. In the case of landfill, there is little value recovered. The deposited waste materials will remain in situ for many years, with some materials effectively never degraded and assimilated safely into the environment. Landfill sites generate leachate, which has to be managed long after the site is closed. Landfilled waste also generates landfill gas, a mix of carbon dioxide, methane and various volatile organic compounds. Landfill gas is odorous, hazardous to health and contributes to global warming, with methane being a powerful greenhouse gas. Some landfill gas can be captured and used for heating or to generate electricity, noting that the collection system is not 100% effective. The combustion of landfill gas, whether flaring for controlled disposal, or in an engine for energy recovery produces emissions, particularly of sulphur dioxide and nitrogen dioxide. These emissions are typically unabated.

By contrast, recovery, in a energy recovery facility (ERF) such as that proposed at Portland, serves several purposes and is designed to derive the maximum value from those waste materials that are left after the maximum usable fraction has been removed.

Firstly, heat energy is released which can be used to generate electricity, off-setting fossil fuel generation. Where there are localised users, this heat can also be used for district or industrial heating, off-setting fossil fuel use. The biomass proportion of the residual waste is defined as a renewable fuel meaning that less fossil fuels are needed.

Secondly, combustion renders the waste biologically inert, with the result that no methane is released, and volatile organic compounds and odours are destroyed.

Thirdly, further useful products are recovered in the form of ferrous and non-ferrous metals extracted from bottom ash, and the ash itself which can be used as a secondary aggregate. In both cases, recovery off-sets the consumption of virgin raw materials that would otherwise be needed and reduces the total energy used to create new raw materials.

In the specific case of the Portland ERF, there is also the added benefit that the ERF plant is specifically designed to provide shore to ship power. When ships are in port they must operate their diesel engines to produce power to keep on-board systems operating. In the case of large cruise liners, this energy demand can be substantial. The electricity generated by the ERF plant will be used to provide shore to ship power, where the ships use this electricity instead of running their engines. This means that the ERF will substantially off-set emissions from ships using Portland.

1.2 Regulation of ERF Plants

Burning residual waste produces combustion gases. A modern ERF plant has a combustion chamber that is finely controlled to ensure optimum combustion and destruction of harmful substances. The ERF is fitted with a series of abatement plant to clean the flue gases. The plant is also designed specifically to avoid the creation of dioxins. This requires the rapid cooling of exhaust gases so that there is no time for de novo synthesis to occur. In fact, treatment in the plant is sufficiently effective that there is an overall net reduction in dioxins.

However, no emissions control system can be completely effective and trace amounts of some substances remain in the flue gases that are emitted to air. The flue stack of the plant is designed to ensure that the impacts of these emissions are not significant. Firstly the ERF must use Best Available Technology (BAT) and meet emission limits as set out in the European Best available

technique Reference Notes (Bref Notes). I n December 2019, new emission standards for new and existing plants came into force in the UK. These tighten further already very strict emission limits, and ensure that plants use BAT.

Furthermore, the plant must also be designed so that there are no significant effects when compared to ambient air quality standards. This requires a series of studies specific to the plant that take into account the location of the plant, terrain, meteorology, and plant-specific design parameters including stack height and stack emission parameters. This process is regulated by the Planning Authority (either national or local) and the Environment Agency and is also scrutinised by Public Health England and Natural England.

1.3 National Context

A comprehensive Air Quality Impact Assessment and Human Health Risk Assessment is required in support of both the Planning Permission and the Environmental Permit for the proposed facility. In order to be meaningful, the risks to health due to the emissions from the ERF need to be considered in context. As mentioned above, residual wastes are inevitable and must be managed appropriately, and the ERF offers many advantages over landfilling. In addition, the contribution of the ERF to the exposure of people to potentially harmful substances would be only a very small fraction of their overall exposure. For example, the Environment Agency states that UK ERF plants contribute 0.05% to total UK emissions of fine particulates, and 1.1% to total UK emissions of oxides of nitrogen. This compares to other contributions: to total emissions of fine particles from traffic of 5.4% and from wood burning of 34.3%; and to total emissions of oxides of nitrogen from traffic of 33.5% and wood burning of 0.6%.

The risks associated with emissions from the UK's ERF plants, such as that proposed at Portland, have been discussed by a range of authorities. Public Health England, the Environment Agency for England and the UK Government (through Defra) jointly state: "*modern, well-managed incinerators make only a small contribution to local concentrations of air pollutants… while it is possible that such small additions could have an impact on health, such effects, if they exist, are likely to be very small and not detectable*". They also state "*well run and regulated modern Municipal Waste Incinerators are not a significant risk to public health*".

There are 48 operational ERF plants in the UK. Some of these have been operational since the 1960s and have been continually upgraded to meet new emission standards. Others are very new plants, with the Javelin Park facility on the Gloucester fringe becoming operational in January 2020. These plants have made a strong contribution to reducing the disposal of waste to landfill, in line with the requirements of the Landfill Directive, UK policy and regulation and the recommendations of bodies such as the Committee on Climate Change in its 2019 Net Zero report. As a result, not only have emissions of methane been reduced, but valuable secondary materials recovered, also reducing emissions of greenhouse gases, and a valuable contribution made to the UK's energy and renewable energy needs and targets.

1.4 Particulate Matter

1.4.1 History and Context

Particulate matter is emitted from combustion processes. Most familiar is 'smoke' created when burning, for example from an open fire or a barbeque. Much attention has been paid to the potential health effects of particulate matter. Historically, attention has been paid to 'black smoke', and more recently the focus has been on specific size fractions. In the regulatory context, the focus has been on PM_{10}, which are particles of a diameter of <10µm, and $PM_{2.5}$, which are particles with a diameter <2.5µm. PM_{10} and $PM_{2.5}$ have been of particular interest as they are the size fractions capable of penetrating the upper respiratory tract (PM_{10}), and alveolar level of the lungs ($PM_{2.5}$).

More recently, attention has also turned to the smaller size fractions, commonly termed 'ultra-fine' particulates. These particles in the sub-2.5µm range are often classified into PM_1 or even $PM_{0.1}$. At this size range, the distinction between a 'particle' and a 'molecule' begins to become blurred. Unlike the larger PM_{10} and $PM_{2.5}$, PM_1 and $PM_{0.1}$ can be generated as secondary pollutants in the atmosphere due to the agglomeration of other pollutants. The interest in PM_1 and $PM_{0.1}$ arises from the fact that particles at this size range are capable of passing the air/blood barrier in the lungs and can therefore, potentially, affect more of the body than just the lungs. In addition, there is evidence that these ultrafine particles also affect the wider body by causing inflammation.

1.4.2 Context of ERF emissions

The ERF plant is equipped with bag filters that remove particulate matter from the exhaust gases. These filters have a very high efficiency, removing over 99.9% of all of the particulate matter from the exhaust flow. Furthermore, the filters are also highly efficient in removing very small particles due to 'agglomeration' effects (a useful analogy is 'running blindfold through a forest without running into a tree'). In the national context, ERFs are a very minor source of ultrafine particles.

Table 1.1 sets out the emissions of $PM_{0.1}$, PM_1, $PM_{2.5}$ and PM_{10} from ERF plants, based on the latest 2018 data. In 2018, over 11 million tonnes of wastes were treated by the 42 ERF plants operating in the UK. For comparative purposes, the UK's total emissions from ERF plants are set out along with emissions for some other common activities [1].

Table 1.1 ERF contribution to PM_1 and $PM_{0.1}$

Activity	Emissions (tonnes per year)				Emissions (as percentage of the total)			
	$PM_{0.1}$	**PM_1**	**$PM_{2.5}$**	**PM_{10}**	**$PM_{0.1}$**	**PM_1**	**$PM_{2.5}$**	**PM_{10}**
Total	13047	46889	87247	96474	#	#	#	#
Waste incineration	16	73	84	84	0.12%	0.16%	0.10%	0.09%
Road transport	3129	4798	11983	18586	24%	10%	14%	19%
Domestic combustion	5037	21118	46791	47864	39%	45%	54%	50%
Bonfire night	145	526	1288	1386	1.1%	1.1%	1.5%	1.4%

Of note is that for PM0.1, for example, ERF plants contribute just 0.12% of the total emissions. Bonfire night alone results in emissions of 10 times more PM0.1 than released by all of the ERF plants in a whole year.

[1] National Atmospheric Emissions Inventory (NAEI) accessed August 2020 https://naei.beis.gov.uk/data/data-selector-results?q=135861

2. ASSESSMENT OF HUMAN HEALTH EFFECTS OF SO_2, NO_2, PM_{10} AND $PM_{2.5}$

2.1 Introduction

This report investigates the human health effects resulting from exposure to some of the substances emitted from the proposed ERF and road traffic. It does so by adapting the quantification methodology used by the Department of Health's Committee on the Medical Effect of Air Pollutants (COMEAP) and the Clean Air for Europe (CAFE) programme. These methods are as set out in the 1998 COMEAP report [1] and the CAFE report [2]. The assessment is based upon health response data from the 2009 COMEAP report[3] . Exposure to increased concentrations of pollutants such as particulate matter (PM), nitrogen dioxide (NO_2) and sulphur dioxide (SO_2) is associated with effects on the human body including the respiratory system, brain and cardiovascular system, leading to increased morbidity and changes in mortality through mechanisms that are not yet fully understood.

It is likely that air pollution affects human health both in the short term and the long term. Short term effects are probably caused by air pollution having a marginal effect on an individual who is already vulnerable, either transiently or permanently. Long term effects may be due to the marginal effect of air pollution in contributing to the progression of chronic diseases that have other causes.

The methods developed by COMEAP and CAFE can be used to predict the health effects associated with developments such as this facility which will result in increased exposure to air pollutants.

2.2 Methodology

2.2.1 Background

Concentration-response functions for the effect of air pollution on health have been proposed based on a review of the available literature. The methodology used in this assessment combines the use of an exposure-response coefficient with details of the specific population affected and the predicted impact from the emissions of the pollution source. The approach to quantifying acute health effects for those pollutants where epidemiology has identified an association is encapsulated by the following linear equation:

$$\Delta E = \beta \times \Delta C \times P \times E,$$

where: $(\Delta)E$ = (change in) background rate of events;
β = exposure-response coefficient;
ΔC = change in concentration of pollutant;
P = population exposed.

2.3 Approach

The exposure-response coefficients used in this assessment are based upon data published by COMEAP. In a series of reports, COMEAP has drawn together a wide range of evidence from which to derive these factors. The epidemiological evidence from which these factors are derived is garnered from a large number of long term studies. As such, older data remain valid and informative for a long period of time and are rarely updated. The COMEAP reports reflect this, in that two reports from 2006 and 2009 contain the majority of the factors used, and subsequent reports have focused on specific topics and present factors only for specific issues.

[1] Committee on the Medical Effects of Air Pollutants (COMEAP) (1998) Quantification of the Effects of Air Pollution on Health in the United Kingdom Department of Health, The Stationery Office, London.

[2] AEA Technology (2005) Methodology for the Cost Benefit Analysis for CAFE. Volume 2: Health Impact Assessment Available at http://europa.eu.int/comm/environment/air/cafe/

[3] COMEAP (2009) Long Term Exposure to Air Pollution: Effect on Mortality.

PM_{10} and $PM_{2.5}$

Health effects associated with PM_{10} and considered in this report include:

- Cardiovascular mortality;
- Cardiovascular admissions;
- Cardiac admissions;
- Ischaemic heart disease admissions;
- Dysrhythmias;
- Heart failure admissions;
- Cerebrovascular admissions; and
- Mortality.

The linear equation in Section 2.2 is used for all of the health effects with the exception of mortality. For mortality, the CAFE methodology adopts the relationship between mortality and long-term exposure to fine particulate matter ($PM_{2.5}$) arising from a cohort study by the American Cancer Society [1]. It also takes the view that the results should be expressed in terms of life years lost, rather than numbers of deaths. This represents the current consensus view of the subject and is also consistent with the view of COMEAP, as set out in its report on the quantification of the long term effects on mortality [2].

In adopting this approach, a different method is required to the equation outlined above for acute effects that instead uses 'life tables'. Miller and Hurley [3] recognise that quantitative health impact assessments of chronic mortality, where the impacts are expected to be observed over a number of years, are complicated by the link between death rates and surviving populations. They have therefore developed a series of spreadsheets to predict the change in mortality based on the life table approach. A similar approach has been adopted in this assessment.

The calculation is carried out by determining the population affected by emissions from the ERF, and based on the life expectancy of men and women, calculating a baseline life expectancy for the population. For every microgram of $PM_{2.5}$ impact as a result of emissions from ERF, there is an associated risk that it will cause a decrease in life expectancy, or loss of life. Thus, the total emissions of $PM_{2.5}$ over the surrounding area of the ERF can be used to calculate what the estimated life years lost will be in terms of the total population exposed.

In June 2009, COMEAP published a second report on the 'Long-term Exposure to Air Pollution: Effect on Mortality'. The updated report summarises the new findings of a significant amount of research that has been undertaken since the publication of the 2001 report. It recommends coefficients which, when used in conjunction with methods developed for the Department of Health and the European Commission by the Institute of Occupational Medicine, will allow the calculation of the potential impact on mortality and life expectancy of specified reductions in concentrations of air pollutants. Although the coefficients have not changed since the previous 2001 report [4], the evidence base regarding the effects of long-term exposure to air pollutants has strengthened since it was published.

The dispersion model outputs for particulate matter are treated as being either PM_{10} or $PM_{2.5}$. In practice, almost all of the PM emitted will be in the size fraction 2.5 µm and less, because the fabric filter used will remove almost all of the particles with a larger diameter, whilst being least efficient at

[1] Pope CA, Burnett RT, Thun MJ, Calle EE, Kreswki D, Ito K, Thurston GD (2002) Lung cancer, cardiopulmonary mortality and long-term exposure to fine particulate pollution. Journal of the American Medical Association 287 1132-1141.

[2] COMEAP (2007) Long term Exposure to Air Pollution - Effects on Mortality. Draft report issued for comment July 2007.

[3] Miller B, and Hurley J: Life table methods for quantitative impact assessments in chronic mortality. Journal of Epidemiology and Community Health.2003; 57: 200-206.

[4] http://www.advisorybodies.doh.gov.uk/comeap/finallongtermeffectsmort2009.htm

around 1 µm. Incidentally, particles of size 0.1 µm and less will be very efficiently removed by the filter through inertial impaction processes.

Nitrogen Dioxide

Health effects associated with nitrogen dioxide and considered in this report include:

- Cardiovascular mortality;
- Cardiac admissions;
- Ischaemic heart disease admissions;
- Heart failure admissions;
- Cerebrovascular admissions; and
- Mortality.

The implications of exposure to NO_2 for respiratory hospital admissions can be considered through the use of the relationship cited by COMEAP (2006), which it took as a 0.038% increase in the rate of the health effect for every 1 µg m^{-3} rise in NO_2 concentrations.

Acute mortality and respiratory hospital admissions from NO_2 should be considered as an alternative to those data used for particulate matter and not in addition. This is because NO_2 may be acting as a marker for a particulate matter effect. Indeed, NO_2 concentrations may be a better marker for locally-emitted particulate matter and its association with health impact in the original epidemiological studies than are the actual PM_{10} concentrations observed, since the latter consist partly of the regional contribution. Likewise mortality and respiratory hospital admissions associated with SO_2 should not be added, as there may be some synergistic effects, ie the observed associations are not independent of each other.

Sulphur Dioxide

Health effects associated with sulphur dioxide and considered in this report include:

- Cardiovascular mortality;
- Cardiovascular admissions;
- Cardiac admissions;
- Ischaemic heart disease admissions;
- Heart failure admissions;
- Cerebrovascular admissions; and
- Mortality.

The implications of exposure to SO_2 could be considered through the use of the relationship used by COMEAP to estimate respiratory hospital admissions, which it took as a 0.05% increase in the rate of respiratory hospital admissions for every 1 µg m^{-3} rise in SO_2 concentrations, and for mortality a 0.06% increase using the linear equation presented previously.

2.3.2 Summary of Concentration-Response Coefficients

Coefficients for health outcomes used in this study and applied to the increased exposure to air pollution are shown in Table 2.1.

Table 2.1 Increases in Health Outcomes from Exposure to an Additional 1 µg m-3

Pollutant	Outcome	Factor used in assessment	Source
PM_{10}	All Mortality	0.0015	COMEAP (2018)
PM_{10}	Cardiovascular mortality	0.0009	COMEAP (2006)
PM_{10}	Cardiovascular admissions	0.0003	COMEAP (2006)
PM_{10}	Cardiac admissions	0.0009	COMEAP (2006)
PM_{10}	Ischaemic heart disease admissions	0.0008	COMEAP (2006)
PM_{10}	Dysrhythmias	0.0008	COMEAP (2006)
PM_{10}	Heart failure admissions	0.0014	COMEAP (2006)
PM_{10}	Cerebrovascular admissions	0.0004	COMEAP (2006)
$PM_{2.5}$	All Mortality	0.006	COMEAP (2018)
$PM_{2.5}$	Cardiopulmonary mortality	0.009	COMEAP (2009)
$PM_{2.5}$	Lung cancer mortality	0.008	COMEAP (2009)
$PM_{2.5}$	Cardiovascular mortality	0.0014	COMEAP (2006)
NO_2	All Mortality	0.00095	COMEAP (2018)
NO_2	Cardiovascular mortality	0.001	COMEAP (2006)
NO_2	Cardiac admissions	0.0013	COMEAP (2006)
NO_2	Ischaemic heart disease admissions	0.0006	COMEAP (2006)
NO_2	Heart failure admissions	0.0013	COMEAP (2006)
NO_2	Cerebrovascular admissions	0.0004	COMEAP (2006)
SO_2	Cardiovascular mortality	0.0008	COMEAP (2006)
SO_2	Cardiovascular admissions	0.0006	COMEAP (2006)
SO_2	Cardiac admissions	0.0024	COMEAP (2006)
SO_2	Ischaemic heart disease admissions	0.0012	COMEAP (2006)
SO_2	Heart failure admissions	0.0009	COMEAP (2006)
SO_2	Cerebrovascular admissions	0.0003	COMEAP (2006)

Sources: COMEAP (2018) Association of long term average concentrations of nitrogen dioxide with mortality; COMEAP (2016) Long term exposure to air pollution and chronic bronchitis; COMEAP (2009) Long-Term Exposure to Air Pollution: Effect on Mortality

2.3.3 COMEAP (2006) Cardiovascular Disease and Air Pollution Approach

Input data and their application

The essential data inputs for air pollution and health effects are:

- Dispersion modelling outputs from the modelling of the ERF and additional road traffic for PM_{10}, SO_2 and NO_2, expressed as annual mean ground level average concentrations (µg m^{-3}) in a spatial output for use with the GIS software ArcGIS;
- Population data, at the 'super output area level', based on the 2011 census; and
- Background rates of all relevant health outcomes (national and local).

The exposed population is defined by the boundaries of the dispersion modelling. Those who fall outside of the dispersion model domain are considered to be unexposed although in reality there will be a gradient of exposure down to zero.

The numbers of exposed people were counted within 10 exposure 'bands,' using GIS software.

The number of people at each exposure level is determined using population density data at the super output area level. This method assumes for pragmatic reasons that there is an equal distribution of people within each super output area and the number of people in each area determined on a pro rata basis.

Once the number of exposed people is known, it is possible to calculate the health effect from exposure to the additional pollutants arising from the operation of the ERF.

Box 2.1 Ship emissions

One of the key reasons for the siting of the proposed ERF is the provision of shore to ship power for vessels in the Portland harbour. Currently, it is not possible to provide power due to limitations in the capacity of the transmission network to Portland. As a result, ships in the harbour use their own engines to generate power. As this power is based on the use of the ships' diesel engines, emissions are inherently high and are not abated. The provision of shore to ship power will greatly reduce the emissions from ships 'hotelling' in the harbour, particularly cruise ships which require a significant amount of power while docked .

This assessment focusses only on the increases in exposure due to emissions from the ERF and road traffic, and does not consider the positive impacts on air quality and health due to the reduction in emissions from vessels in port. Emissions from ships delivering RDF to the proposed development have not been modelled because of the negligible number of vessel movements, the fact that impacts would be limited to the short period they would be in the dock while material was being unloaded and the small amount of power needed to maintain supply to the ship during berthing.

Outputs

Results are expressed as numerical estimates for the morbidity outcomes described above over a 30 year period (estimated life of plant) and also in life years lost.

2.4 The Context

The background statistics that were used to calculate the results were based on national data from various sources. National statistics for disease rates and life expectancy were used for this assessment and are presented in Table 2.2. The diseases assessed are those for which there are risk factors and baseline data available.

Table 2.2 Background Rates of Disease

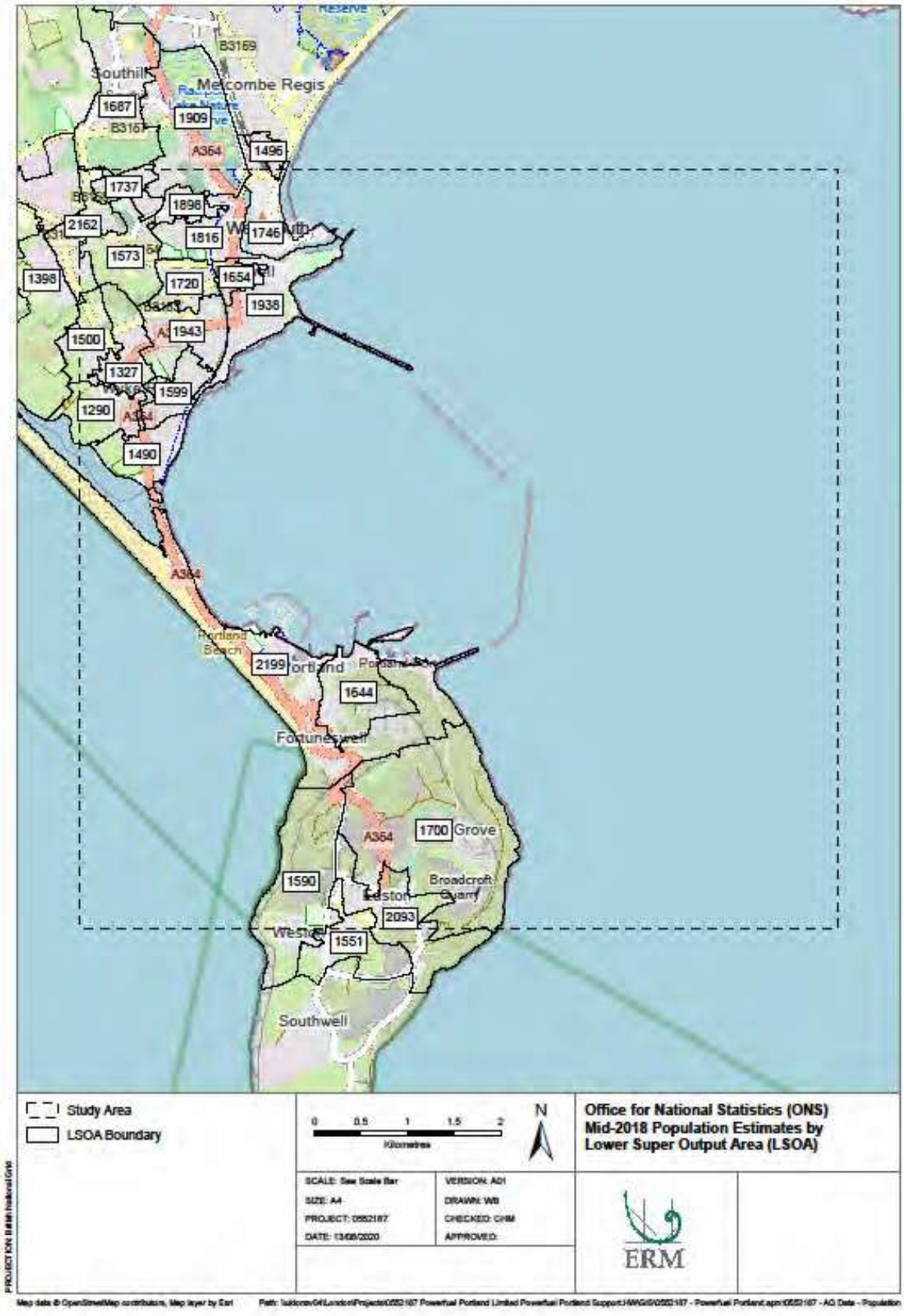
Outcome	Description	Factor
All mortality	2018 crude deaths/1000 population for Dorset	12.5
Lung cancer mortality	Lung cancer mortality	0.547
Cardiovascular mortality	Under 75 mortality rate for all CV disease (2016-2018)	0.56
Cardiovascular admissions	Prevalence of cardiovascular disease (CVD), coronary heart disease (CHD), and stroke (CBVD) by nation and region, United Kingdom 2017	61.62

Outcome	Description	Factor
Ischaemic heart disease admissions	Prevalence of cardiovascular disease (CVD), coronary heart disease (CHD), and stroke (CBVD) by nation and region, United Kingdom 2017	18.15
Heart failure admissions	Total number of in-patient incidents in 2017/2018 = 208757, population 66.27 million	3.150098

2.5 Results

The study area used has a total population of 32,028. The distribution of the population is shown in Figure 2.1. The Air Quality Impact assessment identified the impacts of the emissions from the ERF and from the additional HGV traffic generated by the operation of the ERF plant. From these results, contour plots are generated and overlain on the population data. From these data, the additional exposure of the population in the study area is calculated. The exposure data is then combined with the number of baseline health outcomes and the risk factors for each pollutant used to calculate the additional health outcomes as a result of the operation of the ERF.

Figure 2.1 Population distribution



2.5.1 Additive Effects

The results for each pollutant are presented separately because of the high likelihood that the health effects estimated for each pollutant are not independent of each other. Adding the health effects together will result in an overestimate of the any health effects.

2.5.2 Years of life lost through exposure to $PM_{2.5}$

Application of the method results in an estimate of 0.64 years of life lost per year, distributed across the whole of the exposed population of 32,028. The measure of life years lost would not be equally distributed throughout the exposed population. Statistically, those in the highest exposure group would be most susceptible to a reduction in life years. However, leaving this qualification aside, the result averaged over the exposed population gives a reduction of approximately 10 minutes per person per year, or 5 hours if continually exposed throughout the 30 year lifetime of the plant.

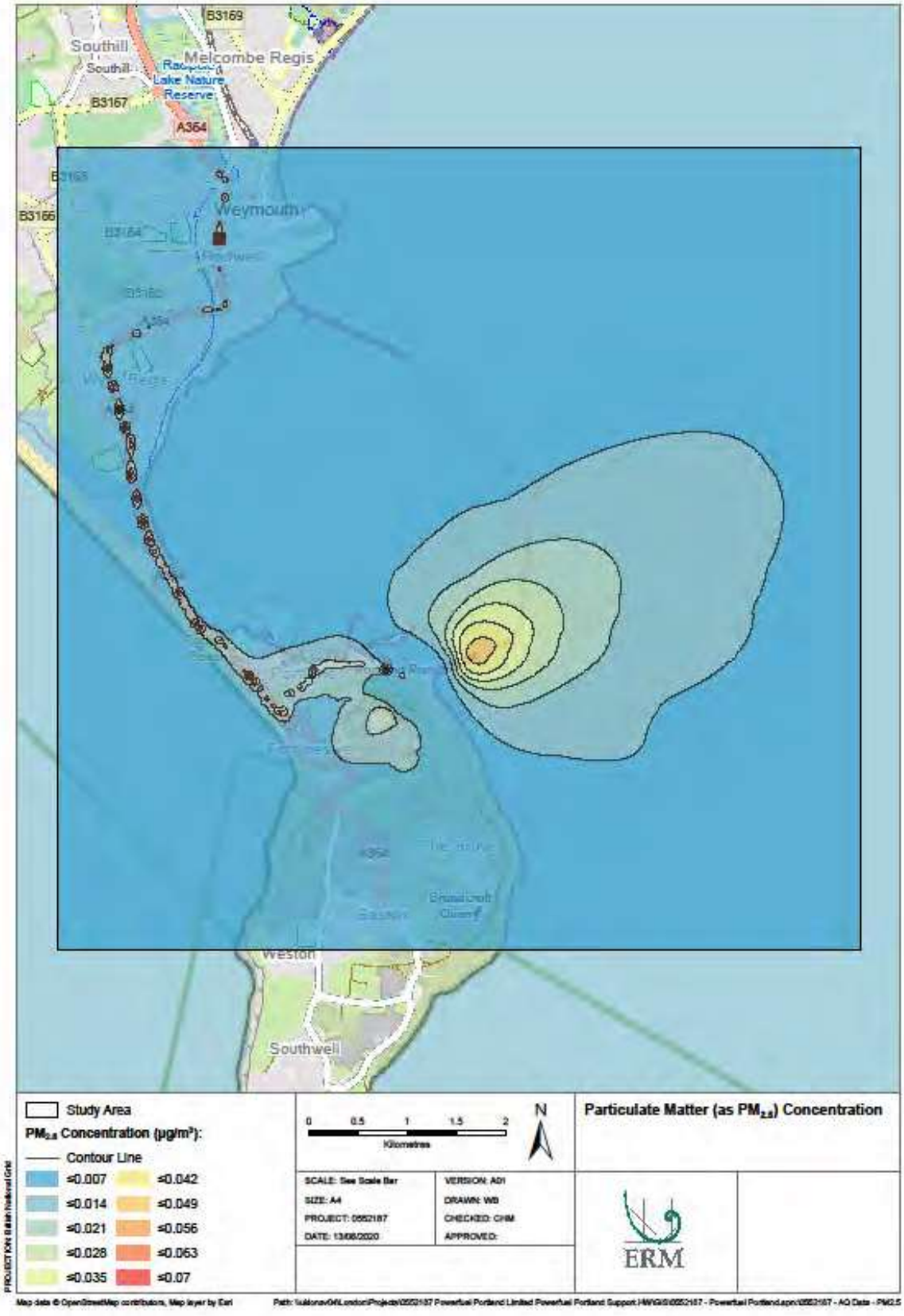
To put this figure into context, it can be compared with the reduction in life expectancy currently experienced as a result of existing air pollution. Public Health England (2014) calculate that 327 years of life are lost per year in the total population of Weymouth and Portland due to existing air quality[1]. For further context, people who have regularly smoked throughout their adult life lose approximately 4 years of life, compared to people who have never smoked[2].

Figure 2.2 shows the impact contour plots of PM2.5 combining plant emissions and traffic emissions.

[1] Public Health England (2014) Estimating Local Mortality Burdens Associated with Particulate Air Pollution https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/332854/PHE_CRCE_010.pdf

[2] ScienceDaily (August 31, 2013), Smokers Who Survive To 70 Still Lose Four Years Of Life, Citing Article In The European Society Of Cardiology Journal

Figure 2.2 PM2.5



2.5.3 Particulate Matter (PM_{10})

Figure 2.3 shows the impact contour plots of PM_{10} combining plant emissions and traffic emissions.

Figure 2.3 PM_{10}

Table 2.3 Estimate of health effects from an increased exposure to PM_{10}

Outcome	Per annum	Per 30 years of operation	Number of years operation for 1 additional case
All mortality	0.0041	0.12	246
Cardiovascular mortality	0.00011	0.0033	9136
Cardiovascular admissions	0.0040	0.12	249
Ischaemic heart disease admissions	0.0032	0.095	317
Heart failure admissions	0.00096	0.029	1044

The increased exposure to PM_{10} will have an insignificant effect on the health of the local population. During the estimated 30 year operating period, there will not be expected to be a single additional case for any of the health indicators. Indeed, the ERF would have to operate for 246 years to generate sufficient pollution for one additional mortality case to arise.

To put these figures into context, for example, there are 18 cases of cardiovascular mortality in the Study Area each year compared to an additional 0.00011 cases due to the operation of the ERF.

2.5.4 Nitrogen Dioxide (NO_2)

Figure 2.4 shows the impact contour plots of NO_2 combining plant emissions and traffic emissions.

Figure 2.4 NO_2

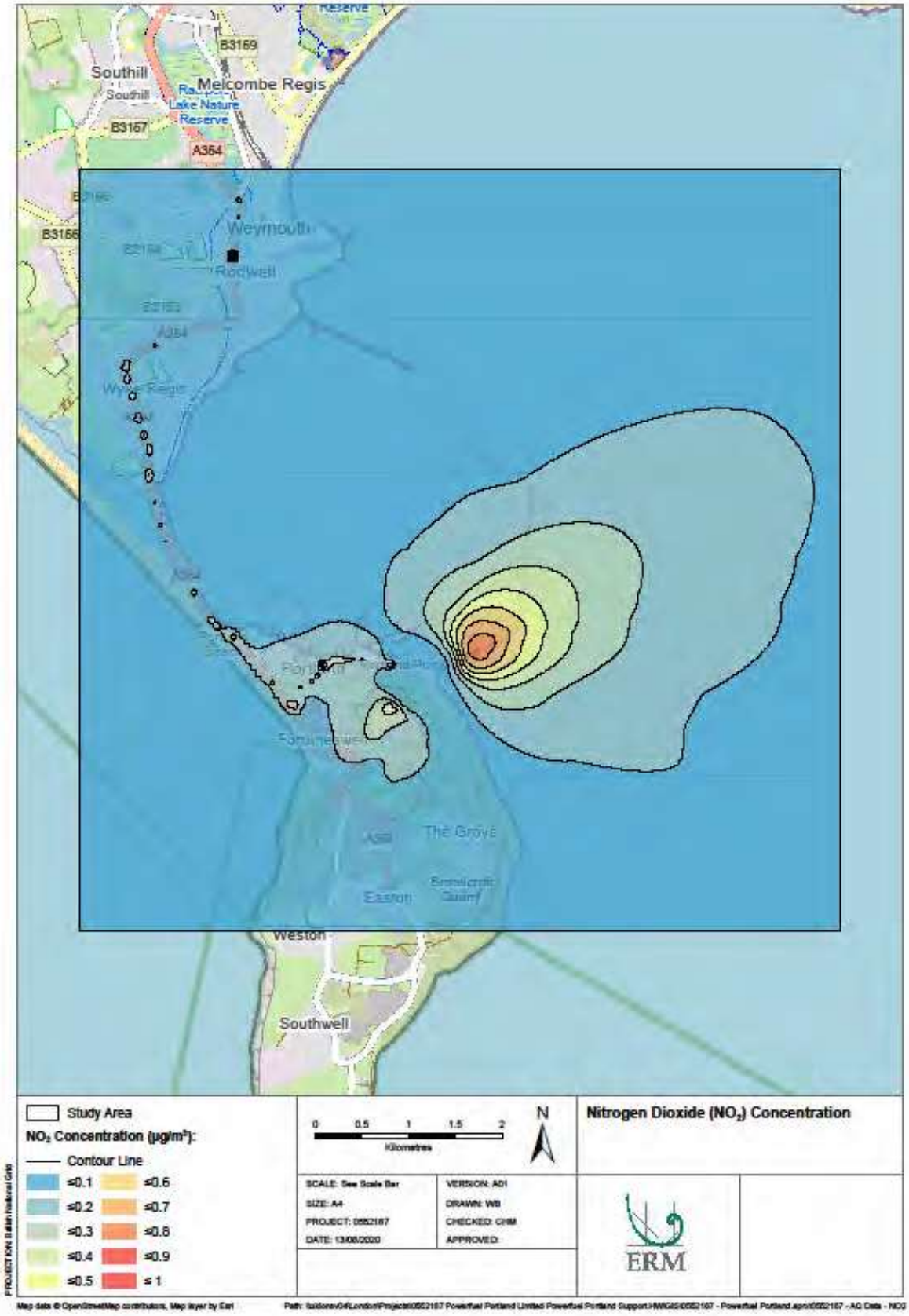


Table 2.4 Estimate of health effects from an increased exposure to NO_2

Outcome	Per annum	Per 30 years of operation	Number of years operation for 1 additional case
All Mortality	0.022	0.67	45
Cardiovascular mortality	0.0011	0.03	948
Ischaemic heart disease admissions	0.021	0.62	49
Heart failure admissions	0.0077	0.23	130
Cerebrovascular admissions	0.0062	0.19	160

To put these figures into context, they can be compared to the total number of Ischaemic Heart Disease (Coronary Heart Disease) primary diagnoses. In the Study Area, there are 581 cases of Ischaemic Heart Disease each year, compared to an additional 0.021 cases due to the operation of the ERF.

2.5.5 Sulphur Dioxide (SO_2)

Figure 2.5 shows the impact contour plots of SO_2 combining plant emissions and traffic emissions.

Figure 2.5 SO_2

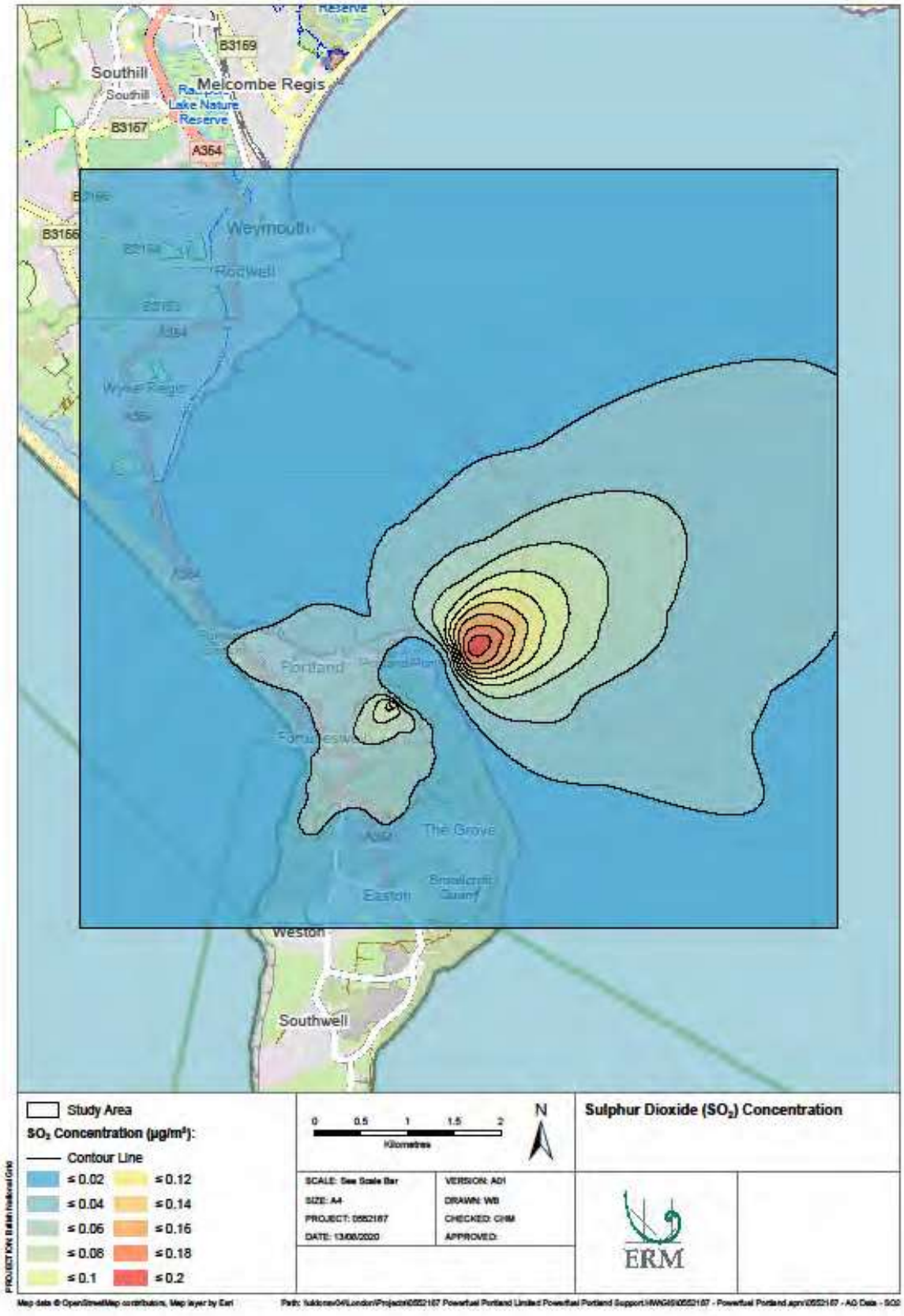


Table 2.5 Estimate of health effects from an increased exposure to SO_2

Outcome	Per annum	Per 30 years of operation	Number of years operation for 1 additional case
Cardiovascular mortality	0.00018	0.0053	5696
Cardiovascular admissions	0.014	0.43	69
Ischeamic heart disease admissions	0.0085	0.256	117
Heart failure admissions	0.0011	0.033	900
Cerebrovascular admissions	0.00097	0.029	1027

The increased exposure to SO_2 will have an insignificant effect on the health of the local population. During the estimated 30 year operating period, there will not be an additional case for any of the health outcomes considered. Again, to put these figures into context, there are 581 cases of Ischaemic Heart Disease in the Study Area each year compared to an additional 0.0085 cases due to the operation of the ERF.

2.6 Conclusions

The health effects associated with emissions of NO_2, SO_2, PM_{10} and $PM_{2.5}$ from the ERF are shown to be very small and could reasonably be described as negligible, especially in comparison to the health effects associated with the existing exposure to atmospheric pollutants and the existing background events for the effects considered.

Furthermore, these impacts are considered only in the context of the increase in $PM_{2.5}$, PM_{10}, NO_2 and SO_2 arising from the operation of the ERF and associated HGV traffic. What is not considered here is the off-set that will be achieved with the provision of shore to ship power provision in Portland. The update of shore to ship power will greatly reduce the emissions of $PM_{2.5}$, PM_{10}, NO_2 and SO_2 arising from shipping emissions, as ships will no longer need continually to run engines to provide power.

Moreover, it is important to recognise that the ERF is treating and disposing of large quantity of waste which must be dealt with by some means. This would very likely be landfill, which is also associated with emissions to air and road traffic. Therefore, the assessment of health effects does not take place against a ‘zero effect’ alternative. All options have some implications for health.

3. LIFETIME HEALTH RISKS

3.1 Scope of the Assessment

The emissions from the proposed ERF plant will contain a number of substances that cannot be evaluated in terms of their effects on human health simply by reference to ambient air quality standards. Health effects occur through exposure routes other than purely inhalation and are cumulative over a lifetime. As such, an assessment needs to be made of the overall human exposure to the substances by the local population and then the risk that this exposure causes.

The assessment presented here considers the impact of certain substances released by the ERF plant on the health of the local population. These substances are those that are 'persistent' in the environment and have several pathways from the point of release to the human receptor. These are generically referred to as 'Contaminants of Potential Concern' (COPCs). The COPCs of interest are dioxins/furans and some metals.

The exposure scenarios used here represent a highly conservative situation in which all exposure assumptions are chosen to represent a worst case and should be treated as an extreme view of the risks to health. The possibility of all high end exposure assumptions accumulating in one individual is, for practical purposes, never realised. Therefore, intakes presented here should be regarded as an extreme upper estimate of the actual exposure that would be experienced by the real population in the locality.

3.2 Approach to the Assessment

The risk assessment process for dioxins/furans and metals is based on the application of the US EPA Human Health Risk Assessment Protocol (HHRAP) 1. This protocol has been assembled into a commercially available model developed by Lakes Environmental, Industrial Risk Assessment Program (IRAP-h, Version 5.1).

The approach seeks to quantify the *hazard* faced by the receptor, the *exposure* of the receptor to the COPC identified as being a potential hazard and then to assess the *risk* of the exposure, as follows.

- **Quantification of the exposure**: an exposure evaluation determines the dose and intake of key indicator chemicals for an exposed person. The dose is defined as the amount of a substance contacting body boundaries (in the case of inhalation, the lungs) and intake is the amount of the substance absorbed into the body. The dose is therefore dependant on:
  - Location of the exposed individual and duration of exposure;
  - Exposure rate;
  - Emission rate from the source.
- **Risk characterisation**: following the above steps, the risk is characterised by examining the toxicity of the COPCs to which the individual has been exposed, and evaluating the significance of the calculated dose in the context of probabilistic risk.

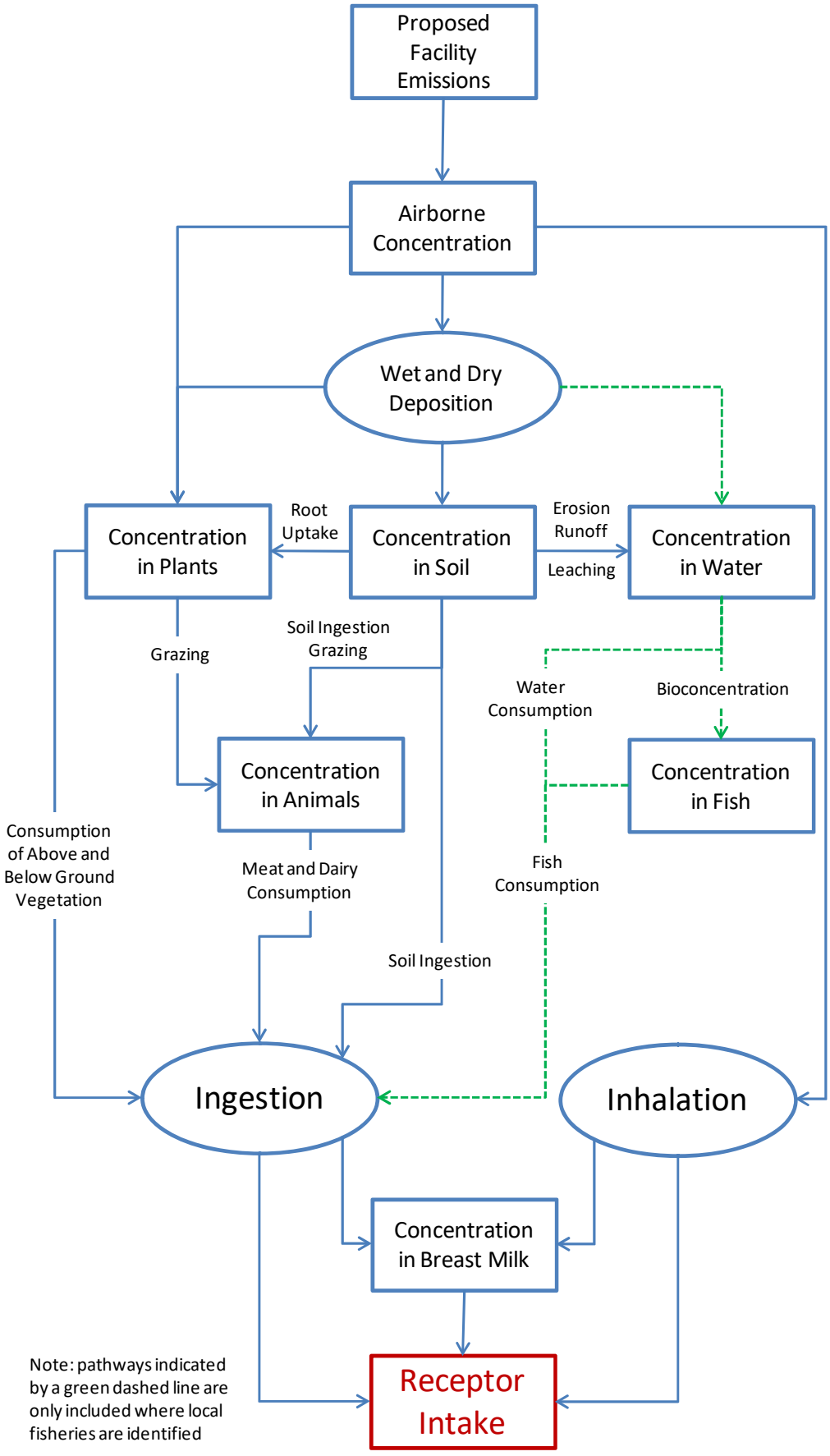
The risk of developing cancer due to exposure to the COPCs is then calculated across the lifetime of an exposed person.

3.2.1 Potential Exposure Pathways

All of the possible exposure pathways included in the IRAP model are shown in Figure 3.1. However, in this case several of these pathways are not applicable.

1 US EPA Office of Solid Waste (September 2005) Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities

Figure 3.1 All Possible Exposure Pathways for Receptors



There are two primary exposure 'routes' where humans may come into contact with COCPs: direct inhalation; and indirectly through ingestion of vegetation, and animals and animal products that become contaminated through the food chain. Given the local context, two exposure scenarios have been assessed for local residents, as follows.

- **Scenario 1** is a person who lives within the study area, and undertakes recreational activities such as gardening. This means that exposure is via inhalation, dermal contact with soil and some incidental ingestion of soil. However, this person does not cultivate food at home, and does not consume locally grown food, for example fruit and vegetables, eggs, chickens or meat.
- **Scenario 2** is a person who lives within the study area, and undertakes recreational activities such as gardening. This person does cultivate food at home, and does consume locally grown food, including fruit and vegetables, eggs and chickens. However, this person does not consume locally farmed larger animals such as pigs or cattle. This means that exposure is via inhalation, dermal contact with soil, incidental ingestion of soil and via intake through food grown at the property.

In scenario 2 the total intake will be greater, as this person is also exposed via the food chain due to consuming locally grown produce.

The following exposures are assumed to be negligible:

- Dermal contact with soil, given the sporadic nature of exposure and the very low dermal uptake rate;
- Contact with contaminated water when swimming and through consumption of locally caught fish due to the sporadic nature of exposure, and the fact that in the marine environment sea water is continually circulated away from the port so accumulation does not occur;
- Drinking water, as all properties are assumed to be on mains water or drawn from a borehole (contamination pathway would only be via surface water, which does not occur); and
- There is no significant livestock rearing in the study area, and therefore consumption of locally grown beef and pork is not considered.

3.2.2 Compounds of Potential Concern (COPCs)

The COCPs that have been included for this assessment are those that are permitted emissions and which are included in the EPA HHRAP COPC database for the assessment of long term health effects. Therefore, the following have been considered as COPCs for the proposed ERF:

- Dioxins and Furans (note that the worst case assumption is made that all emissions are as the most hazardous TCDD congener);
- Antimony (Sb);
- Arsenic (As);
- Cadmium (Cd);
- Chromium (Cr), trivalent and hexavalent;
- Lead (Pb);
- Mercury (Hg);
- Nickel (Ni); and
- Thallium (Tl).

3.2.3 Emission Concentrations for the COPCs

The emission concentrations and impacts for dioxins/furans and metals are reported in the air quality impact assessment (Technical Appendix D).

Box 3.1 Metals emissions

In Technical Appendix D an explanation is provided on the method for deriving emission concentrations for metals, using the methodology set out by the Environment Agency. In Technical Appendix D the 'maximum' emissions have been used in the assessment. However, in this assessment the 'mean' emissions are used to calculate the metals emissions.

This approach is appropriate in order to accurately reflect the long term assessment scenarios and avoid overstating impacts through the combination of multiple worst case assumptions.

Table 3.1 Emission Rates Used in the IRAP Model

COCP	Emission (g/s)
Dioxins	2.34×10^{-9}
Antimony	1.43×10^{-3}
Arsenic	4.69×10^{-4}
Cadmium	7.81×10^{-4}
Chromium III	1.07×10^{-3}
Chromium VI	1.31×10^{-5}
Lead	2.54×10^{-3}
Mercury	7.81×10^{-4}
Nickel	7.99×10^{-4}
Thallium	7.81×10^{-4}

In terms of mercury, the worst case assumption is made that there are no losses to the global cycle, and all mercury is available for deposition from the vapour phase.

The general term dioxins denotes a family of compounds, with each compound composed of two benzene rings interconnected with two oxygen atoms. There are 75 individual dioxins, with each distinguished by the position of chlorine or other halogen atoms positioned on the benzene rings. Furans are similar in structure to dioxins, but have a carbon bond instead of one of the two oxygen atoms connecting the two benzene rings. There are 135 individual furan compounds. Each individual furan or dioxin compound is referred to as a congener and each has a different toxicity and physical properties with regard to its atmospheric behaviour. In this case, the assumption is made that all dioxins are emitted as 2,3,7,8 TCDD, the most hazardous cogener. This represents the worst case approach. This approach was used in this case as uptake into the food chain, and accumulation in larger farmed animals is not a consideration and this exposure rate dominates exposure.

Deposition to soils for the IRAP model has been calculated from the air quality modelling results. This calculation requires particle size and deposition rate. Particle size distribution was derived from Jones and Harrison (2016) [1], and identified particle mode at 0.1µm and deposition velocity was derived from Gronholm et al. (2007) [2], with a deposition velocity of 0.4cm/s.

3.2.4 Input Parameters for the IRAP Model

As discussed in Section 3.2.1, two exposure scenarios have been considered. These are essentially the same, with the exception that one scenario includes consumption of locally grown vegetables, poultry and eggs.

The receptor types can also be divided into adults and children. Children are important receptors because they tend to ingest soil and dusts directly and have lower body weights, so that the effect of the same dose is greater in the child than in the adult. However, a child's exposure is less significant for cancer outcomes given the shorter exposure time in childhood compared to whole lifetime exposure.

The IRAP model contains a database of physical and chemical parameters for each of 206 COPCs. This database is based on default values provided by the HHRAP and all default values have been used for this assessment.

3.2.5 Site and Site-Specific Parameters

The IRAP health risk assessment model requires information relating to the location and its surroundings. The parameters required include the following.

- The fraction of animal feed (grain and forage for poultry) grown on contaminated soils and quantity of animal feed and soil consumed by poultry is considered.
- The interception fraction for above ground vegetation, forage and silage and length of vegetation exposure to deposition. The yield/standing crop biomass is also required.
- Input data for assessing the risks associated with exposure to breast milk, including:
  - body weight of infant;
  - exposure duration;
  - proportion of ingested COPC stored in fat;
  - proportion of mother's weight that is fat;
  - fraction of fat in breast milk;
  - fraction of ingested contaminant that is absorbed; and
  - half-life of dioxins in adults and ingestion rate of breast milk.
- Other physical parameters (e.g. soil dry bulk density, density of air, soil mixing zone depth).

For all of these parameters, the IRAP/EPA HHRAP default values have been used. Other site-specific parameters are also required which are not provided by the IRAP model. These parameters were specified for the proposed ERF plant location as follows:

- Annual average evapotranspiration rate of 55 cm a^{-1} (assumed to be 70% of total precipitation);
- Annual average precipitation of 78.5 cm a^{-1} (based on 2004 meteorological data);
- Annual average irrigation of 0 cm a^{-1};

[1] Jones A. Harrison R. (2016) Emission of ultrafine particles from the incineration of municipal solid waste: A review Atmospheric Environment Vol. 140

[2] Gronholm T. Aalto P, Hiltunen V et al (2007) Measurement of aerosol particle dry deposition velocity using the relaxed eddy accumulation technique Tellus Vol 59, Issue 3

- Annual average runoff of 7.8 cm a^{-1} (assumed to be 10% of total precipitation); and
- A time period over which deposition occurs of 30 years.

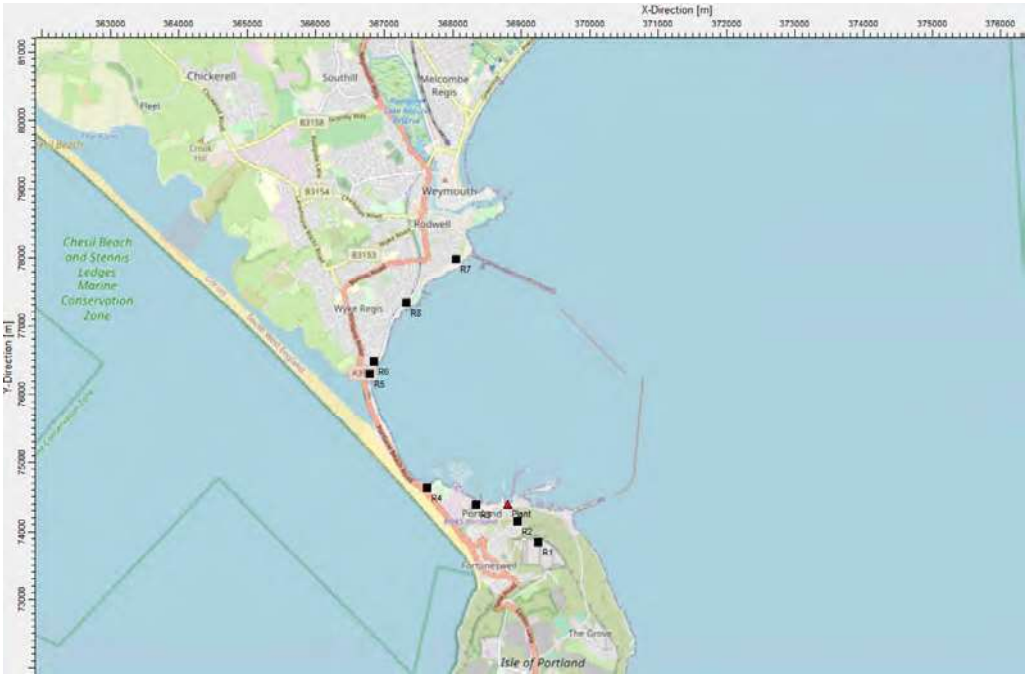
3.2.6 Receptors

In addition to the two exposure scenarios described, eight representative receptor locations have been identified. These are set out in Table 3.2 and Figure 3.2.

Table 3.2 Receptors

ID	Location	X	Y	Notes
R1	Fortuneswell, Portland	369250	73840	Highest terrestrial value anywhere receptor present
R2	East Weare Road, Portland	368923	74138	
R3	Castletown, Portland	368374	74358	
R4	Property on Hamm Beach Road, Portland	367638	74598	
R5	Smallmouth Close, Weymouth	366775	76295	
R6	Dowman Place, Weymouth	366853	76462	
R7	Redcliffe View, Rodwell	368089	77993	
R8	Old Castle Road, Weymouth	367310	77299	

Figure 3.2 Receptors



3.2.7 Assessment of Non-carcinogenic and Carcinogenic Risk

Non-carcinogenic Risk

The non-carcinogenic effect of the emissions on human health can be assessed in terms of the Hazard Quotient (HQ). For ingestion, the HQ is calculated as the Average Daily Dose (ADD) divided by the reference dose (RfD). For example, the HQ for ingestion exposure for cadmium (Cd) is calculated as follows:

$$HQ_{Ing,\,Cd} = \frac{ADD_{Ing,\,Cd}}{RfD_{Ing,\,Cd}}$$

Where:

$$ADD_{Ing,\,Cd} = \frac{I_{Ing,\,Cd} \bullet ED \bullet EF}{AT \bullet 365}$$

Where: $ADD_{Ing,\,Cd}$ = ingestion dose for cadmium; ED is the exposure duration (dependent on the receptor type); EF is the exposure frequency (350 days per year); and AT is the averaging time (equal to ED for non-carcinogenic effects and 70 years for carcinogenic risks).

For inhalation, the HQ is calculated as the exposure concentration divided by the reference concentration (RfC). For example, the HQ for inhalation exposure for cadmium (Cd) is calculated as follows:

$$HQ_{Inh,\,Cd} = \frac{EC_{Cd} * 0.001}{RfC_{Inh,\,Cd}}$$

Where:

$$EC_{Cd} = \frac{C_a \bullet ED \bullet EF}{AT \bullet 365}$$

Where: EC_{Cd} is the exposure concentration (µg m 3), $RfC_{Inh,\,Cd}$ is the reference concentration for cadmium (mg m^3) and Ca is the concentration of cadmium in air.

The Reference Dose and Reference Concentration for each COPC and exposure pathway is provided in Section 4.7. The RfDs and RfCs are set conservatively, that is they are protective of health and doses at or greater than the RfD or RfC indicate the potential for effect, rather than clear and certain indication of an effect. For example, should the maximum daily intake for the new source, in this case the proposed ERF plant, be equal to the RfD, then the HQ would be equal to 1.0 and this would indicate the potential for a health effect. On the other hand, a hazard quotient of less than unity (1.0) implies that such an exposure would not create an adverse non-carcinogenic health effect.

The Hazard Index (HI) is the sum of the individual COPC/pathway HQs and assumes that there are no synergistic or antagonist health effects arising from the release. The smaller the HI, the less risk to human health is implied.

The risk of interest in this context is the extra lifetime risk associated with the total dose resulting from exposure to the proposed ERF plant emissions. For each COPC, the US EPA has calculated a carcinogenic slope factor (CSF). These are calculated for ingestion exposure whereas for inhalation exposure, a unit risk factor (URF) has been adopted. Where the CSF or URF is zero, this indicates that the COPC is non-carcinogenic via that exposure route. The IRAP model uses these values to calculate a cancer risk for each pollutant and for each pathway for exposure, so that the results can be expressed in a high degree of detail.

The risk associated with the ingestion exposure (food, water and soil) of cadmium is calculated as follows:

$$Risk_{Ing,\,Cd} = ADD_{Ing,\,Cd} \bullet CSF_{Ing,\,Cd}$$

Where $ADD_{Ing, Cd}$ is the sum of the average daily dose from all ingestion exposure routes.

The risk associated with the inhalation of cadmium is calculated as follows:

$$Risk_{Inh, Cd} = EC_{Cd} \bullet URF_{Inh, Cd}$$

3.2.8 Defining Significance

In order to quantify the risks, the following significance thresholds are used:

- For non-carcinogenic risks the threshold is 1.0. Where a value less than 1.0 is predicted, then health risk is insignificant.
- For the purposes of this study, guidelines on cancer risk from the World Health Organisation have been used. The WHO sets two thresholds:
  - A risk of 1 in 100,000 lifetime risk is considered 'maximum tolerable risk'; and
  - A risk of 1 in 1,000,000 lifetime risk is considered 'acceptable risk' at which no further improvements to safety need to be made.

3.3 Assessment of Non-Carcinogenic Effects

3.3.1 Summary of Non carcinogenic Effects

The Hazard Index (HI) calculated by IRAP for emissions from the ERF plant for each of the nine receptors (adult and child) is presented in Table 3.3.

Table 3.3 Non-Cancer Risk

Scenario	Receptor	Exposures	R1	R2	R3	R4	R5	R6	R7	R8
Significance Threshold						**1.0**				
1	Adult	No home grown produce, assume no soil ingestion	$5.14X10^{-4}$	$1.71X10^{-4}$	$2.02X10^{-4}$	$1.20X10^{-4}$	$4.35X10^{-5}$	$4.09X10^{-5}$	$3.58X10^{-5}$	$4.35X10^{-5}$
1	Child	No home grown produce, assume some soil ingestion	$8.81X10^{-4}$	$2.94X10^{-4}$	$3.46X10^{-4}$	$2.05X10^{-4}$	$5.51X10^{-5}$	$7.09X10^{-5}$	$3.58X10^{-5}$	$7.52X10^{-5}$
2	Adult	Home grown produce – fruit and vegetables, chicken, eggs (no home grown milk, beef or pork)	$1.13X10^{-2}$	$3.79X10^{-3}$	$4.64X10^{-3}$	$2.63X10^{-3}$	$9.77X10^{-4}$	$9.27X10^{-4}$	$8.03X10^{-4}$	$9.81X10^{-4}$
2	Child	Home grown produce – fruit and vegetables, chicken, eggs (no home grown milk, beef or pork)	$2.67X10^{-2}$	$8.97X10^{-3}$	$1.05X10^{-2}$	$6.22X10^{-3}$	$2.31X10^{-3}$	$2.19X10^{-3}$	$1.89X10^{-3}$	$2.31X10^{-3}$

The HIs are substantially below the significance threshold of 1.0 and therefore the conclusion is reached that there will not be significant effects at any receptors.

3.4 Assessment of Carcinogenic Effects

The total lifetime cancer risk calculated by IRAP for emissions from the ERF plant for each of the receptors is presented in Table 3.4.

Table 3.4 Results – Cancer Risk

Scenario	Receptor	Exposures	R1	R2	R3	R4	R5	R6	R7	R8
Significance Threshold		1.0X10-6 (1 in 1,000,000 lifetime risk)								
1	Adult	No home grown produce, assume no soil ingestion	$3.11X10^{-8}$	$1.03X10^{-8}$	$1.22X10^{-8}$	$7.29X10^{-9}$	$2.63X10^{-9}$	$2.48X10^{-9}$	$2.17X10^{-9}$	$2.63X10^{-9}$
1	Child	No home grown produce, assume some soil ingestion	$6.26X10^{-9}$	$2.08X10^{-9}$	$2.46X10^{-9}$	$1.46X10^{-9}$	$5.29E^{-10}$	$4.98E^{-10}$	$4.36E^{-10}$	$5.29E^{-10}$
2	Adult	Home grown produce – fruit and vegetables, chicken, eggs (no home grown milk, beef or pork)	$4.84X10^{-8}$	$1.61X10^{-8}$	$1.64X10^{-8}$	$1.13X10^{-8}$	$4.11X10^{-9}$	$3.87X10^{-9}$	$3.38X10^{-9}$	$4.11X10^{-9}$
2	Child	Home grown produce – fruit and vegetables, chicken, eggs (no home grown milk, beef or pork)	$8.71X10^{-9}$	$2.90X10^{-9}$	$2.82X10^{-9}$	$2.03X10^{-9}$	$7.41E^{-10}$	$6.99E^{-10}$	$6.10E^{-10}$	$7.42E^{-10}$

The risk of cancer in all receptors are substantially below the 1 in 1 million threshold of significance set by the WHO. On this basis, there is negligible risk of cancer due to emissions form the ERF plant.

3.5 Summary and Conclusions

The assessment considered the potential impacts of emissions on human health. Eight representative receptor locations were identified in Portland and Weymouth, including the location where the highest impacts on a terrestrial location are predicted to arise.

Two exposure scenarios were considered. One considered a resident living close to the plant, and not growing any food at home. A second scenario was also considered where the resident is eating fruit, vegetables, chicken and eggs reared on their property. Consideration was also made of the relative difference in exposure of adults and children.

The assessment is worst case, insomuch as the assumption is made that the residents are exposed for 350 days per year, for a 70 year lifetime. However, in order to reflect a more realistic case, the 'average' emissions of metals is used rather than the 'maximum' that was used in the air quality impact assessment.

The assessment concluded that the risk to health due to emissions from the ERF plant are negligible, in terms of both carcinogenic and non-carcinogenic risks.

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