

## **CERC/Proof /Bridges**

**TOWN & COUNTRY PLANNING (INQUIRIES PROCEDURE) (ENGLAND)  
RULES 2000**

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**Land at Rostowrack Farm, Wheal Remfry, Goonvean and Parkandillick Dryers, St  
Dennis, St Austell**

## **Health Effects of Atmospheric Emissions - Proof of Evidence of Professor Jim Bridges**

**RESEARCH FOR SUSTAINABILITY,  
GUILDFORD, SURREY.**

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## **0.1 PURPOSE OF THIS PROOF OF EVIDENCE**

I am Professor Jim Bridges, Emeritus Professor of Toxicology and Environmental Health at the University of Surrey and Chair of the EU Scientific Advisory Committee on Emerging and Newly Identified Health Risks. For the past thirty years I have been involved in the assessment of health risks from different chemicals and processes (For short CV see appendix 1)

The principal purpose of this Proof is to carry out an assessment of the risk to the local community from chemicals that may be emitted to air from the stack of the proposed plant. The report relies on both the published scientific literature and on the air quality section of the environmental statement (ES). The approach used has been to:

- To briefly summarise the information on the relationship between air quality and health taking into account sources of air pollution in Cornwall (Appendix 1 and Chapter 1)
- To Identify the chemicals of interest that may be emitted from the stack and to set out the most appropriate methodology for the assessment of the possible health impact of each chemical on the health of the local population (Chapter 2, section1)
- To evaluate the impact of each chemical in turn by comparing conservative estimates of maximum ground level concentrations against relevant standards or guidelines and through consideration of their contribution to existing background concentrations. (Chapter 2 section 2)
- To consider any consequences in health terms from simultaneous exposure to these chemicals of interest (Chapter 2 section3)

- To consider whether there could be any human health implications through the uptake of any of these chemicals of interest in food animals and plants (Chapter 2 section 4)
- To compare the above conclusions with those of the published literature on measurements of the same chemicals of interest around comparable waste to energy/incinerator plants operating in various countries (Chapter 3, section1 and appendix 3 )
- To review the published literature that considers the health implications of living in the neighbourhood of a modern waste to energy/incinerator plant (Chapter 3 section2 and Appendix 4).
- To compare the above findings with the views of expert committees and government reports that address the relationship between incinerator emissions and local community health. (Chapter 3, Section 3).
- To summarise the relevant aspects of some of the more recent reports of planning inspectors who have reviewed the evidence for specific waste to energy plant proposals (Chapter 3, Section 4)

I confirm that the evidence which I have prepared for this appeal Inquiry ( reference APP/DO840/A/09/211307) in this proof of evidence is true and has been prepared and is given in accordance with the guidance of my professional institution and I confirm that the opinions expressed are my true and professional opinions.

## **CHAPTER 1: AIR QUALITY AND HEALTH CONSIDERATIONS**

### **1.1 LOCAL CONCERNS**

1.1 It is appropriate when conducting a risk assessment on a particular facility or proposed facility to note the nature of public concerns and to address them where possible in the assessment. The rule 6 statements have identified several concerns most of which are of a general nature. The St Dennis Anti-incinerator Group (STIG) has expressed concerns regarding the emission levels and health impacts of dioxins, small particles and other pollutants which they do not name and how these might combine with clay dust. An identically worded statement is made by St Dennis Parish Council. Both also have identical statement on the anxiety of the local community and perception of harm to health.

The Cornwall Sustainable Waste Network (CSWN) also raises the issue of dioxins and its concerns on the possible impact on the food industry and associated sectors and of perceptions of adverse impacts on the quality and purity of food.

1.2 Individual local residents have also identified dioxins and/or particulate matter as of concern. Several state that there is a high incidence of ill health in the area and that the proposed plant might exacerbate this situation. Other refer to statements by Mr Ryan or Dr Van Steenis suggesting that there are health problems around incinerators in the UK that are currently in operation .

1.3 These concerns are addressed in this Proof as follows:

- dioxins and particulate matter are specifically addressed (along with many other chemicals both in the main proof and in the appendices)
- the local situation in terms of air pollutants and health is reviewed briefly (appendix 9).
- the issue of possible food contamination is discussed (section 2.5)
- the published literature on contamination around incinerators is reviewed (section 4.1 and appendices 6 and 7).

- Findings of research on the relationship between actual risk and perceived risk are discussed (appendix 8).

## **1.2 AIR QUALITY AND PUBLIC HEALTH**

1.4 Public health specialists in Europe have recommended that the priority chemicals for assessment are particulate matter (PM)(both as PM<sub>10</sub>, and PM<sub>2.5</sub>), nitrogen dioxide (NO<sub>2</sub>) and ozone (O<sub>3</sub>) .

1.5 In regard to the health of the population in Cornwall, The NHS Report (2008) identified the incidence of respiratory diseases in Cornwall overall as slightly above the national average (1.5 compared to 1.43), This difference is probably not significant. However in some areas of Cornwall the incidence was found to be rather greater (1.9). This difference is probably attributable primarily to the fact that these areas tend to be socio-economically disadvantaged. It is very difficult from the data available to identify whether this increased incidence of respiratory disease also has some association with quality. Nonetheless it is appropriate to identify some of the sources of air pollution in Cornwall (see appendix 9).

## **1.3 MAIN CONTRIBUTORS TO CHEMICALS IN AIR**

1.6 Burning of fossil fuels and an ever increasing number of motor vehicles continue to influence ambient air quality in the EU in general and in the UK in particular. Primary man made sources of air pollution include diesel and petrol driven engines, mineral excavation, and electricity generation (see appendix 9)

1.7 Over 900 different chemicals have been found in indoor air (SCALE, 2004). It is generally assumed that most individuals spend 8 hours at work and 10-12 hours at home every day, which makes the total indoor exposure around 20 -22 hours. Consequently a particular concern for health is indoor air quality.

## **1.4 CONCLUSIONS**

1.8 In Cornwall as in other counties of England and Wales the air contains many chemicals of natural origin and as a result of human activities. In terms of human sources affecting air quality and the levels of individual chemicals in the air Cornwall appears to be very largely similar to many other counties and indeed many other developed countries. The key consideration from a health risk assessment viewpoint is will the proposed CERC have a significant impact on the local air quality. This assessment is the carried out in the subsequent chapters of this proof of evidence and in the appendices.

## **CHAPTER 2 RISK ASSESSMENT OF INDIVIDUAL CHEMICALS OF INTEREST**

### **2.1 PRINCIPLES OF RISK ASSESSMENT**

2.1 It is crucial to any consideration of health risk to the local population, from the operation of a proposed plant such as the CERC, to recognize that there is a great difference between a *hazard* and a *risk*. 'Hazard' describes the intrinsic adverse properties of the chemical, biological or physical agent of interest.

2.2 Commonly, in order for the adverse effects to be realized a high level and /or extended duration of exposure is required. Every chemical, biological or physical agent has hazardous properties (regardless of whether they are 'natural' or 'man-made'). However, these properties will differ from one another both in their nature and in their potency (i.e. the exposure conditions required to produce an adverse effect(s) between chemicals).

2.3 Risk can be defined as the chance, in quantitative terms, of a defined hazard occurring. The 'risk' to human health defines the nature and severity of the effects due to exposure to the chemical, biological or physical agent under a particular set(s) of exposure conditions. Thus, to determine whether there is a risk from exposure to substances emitted from the proposed plant (and if so, the nature and magnitude of the risk) consideration of the likely exposure conditions (in particular the ground level concentrations in areas where members of the local populations reside) is essential.

2.4 Although the distinction between hazard and risk is well understood by public health experts the two terms are often confused in the media and by politicians and lobbyists. An exposure to a particular chemical can be considered as a

hazard but depending on the exposure conditions, the risk may be insignificant (i.e. close to zero) or significant or substantial. To determine the level of risk requires good information on the actual/likely exposure situation.

2.5 To assess the risk to human health from chemicals emitted from the proposed CERC the following is required :

- To select the chemicals that are most likely to be of concern to health
- To measure or calculate the level, duration and possible routes of exposure to each chemical of interest (*exposure assessment*)
- To identify and understand hazardous properties of the chemicals of interest (*hazard assessment*)
- To assess the potential risk (*safety margins and/or life time risk*).

### **2.1.2 Selection of the chemicals of interest**

2.6 This Proof of Evidence is concerned with the potential health risks to the local population from the chemicals that are likely to be released in the air (termed the 'chemicals of interest' subsequently) as a result of the operation of the proposed CERC. The chemicals selected either have statutory limits on their emission levels and/or have full or draft ambient air standards.

### **2.1.3 Exposure assessment**

2.7 This Proof of Evidence is based on the air quality section of the Environmental Statement:

- the air quality modelling work and environmental measurements provided in the air quality report
- Use of COMEAP ( the UK Committee on Medical Episode of Air Pollution) calculation based on the estimated exposure to sulphur dioxide, nitrogen dioxide and particulate matter
- the estimates of ingestion and inhalation of persistent chemicals, such as

dioxins and metals.

#### **2.1.4 Hazard assessment**

2.8 The purpose of the hazard assessment is to identify:

- \* all the hazardous properties of the chemical of interest that may be relevant from a public health viewpoint
- \* a relevant standard or guideline, that has an appropriate specified duration, and is sufficiently strict to ensure that each hazardous property is suitably covered by it. If such a standard cannot be identified then it is necessary to derive one from the toxicological and epidemiological data base on that chemical.

#### **2.1.5 Risk assessment**

2.9 A safety margin may be calculated once the standard/guideline value has been selected and a conservative exposure level estimated by dividing the standard by the exposure level (see appendix 3).

2.10 In this Proof of Evidence a tiered approach devised by the author has been used. In tier 1 to assess the safety margin(s) the worst case scenario is adopted, in which a maximum hypothetical exposure and the strictest standard are selected as the basis for calculation of safety margins. For short-term exposure, if the safety margin are greater than 10, no further assessment is required (See Appendix 4). For long-term exposure, if the safety margin is greater than 100, no further assessment is required.

## **2.2 SHORT -TERM AND LONG -TERM EFFECTS OF THE CHEMICALS OF INTEREST.**

2.11. For some chemicals it is the effects that follow shortly after a peak of exposure that need to be given particular consideration from a risk assessment perspective. These chemicals are usually referred to as acutely toxic(short-term). Chemicals that cause irritation to the eyes, nose, throat and/or lungs are in this

category.

2.12. For many other chemicals only at very high exposure levels will acute effects occur. Such exposure levels are only likely to be achieved under extreme exposure conditions. However, if exposure to them occurs frequently then over time adverse effects may arise. These chemicals are often referred to as chronically toxic(long-term).

2.13 In the following parts of this chapter those chemicals of interest whose principal effects are acute are addressed first followed by those that exert principally chronic adverse effects.

## **2.3 CHEMICALS OF INTEREST WITH PRINCIPALLY SHORT-TERM EFFECTS**

A number of chemicals fall into this category including a number of criteria pollutants. At high exposure levels they tend to be irritants to the mucous membranes.

### **2.3.1 HYDROGEN CHLORIDE (HCl) AND HYDROGEN FLUORIDE (HF)**

#### *Sources of exposure*

2.14 Both hydrogen chloride and hydrogen fluoride, in the presence of moisture are acidic. Hydrogen fluoride is the stronger acid. In contact with food these acids are rapidly neutralised. Thus, the only relevant routes of environmental exposure from a health risk viewpoint, to these two chemicals are inhalation and dermal contact.

2.15 The inhalation route is, by far, the most important one. Current levels of these two acids in ambient air in the UK are not considered to be a public health

concern, therefore no routine measurements are made of their levels in urban environments. However, from the data that is available from ERM it appears that the area around the proposed CERC has average levels of hydrogen chloride for an urban environment.

#### *Summary of effects*

2.16 The main adverse effect is inflammation of mucous membranes. Due their acidic properties, if present in air at sufficient concentrations these two chemicals will cause acute irritation to the eyes nose, throat and respiratory tract (lungs). For each of these effects a threshold can be identified below which adverse effects are unlikely to occur.

#### *Basis for the standards*

2.17 The UK Environment Agency has identified a short term EAL of  $800\mu\text{g}/\text{m}^3$  for hydrogen chloride and  $250\mu\text{g}/\text{m}^3$  for hydrogen fluoride based on their irritant properties. The Expert Panel on Air Quality Standards (EPAQS) has recommended slightly stricter short- term EAL of  $750\mu\text{g}/\text{m}^3$  for hydrogen chloride and  $160\mu\text{g}/\text{m}^3$  for hydrogen fluoride. An annual standard has been set for HCl of  $20\mu\text{g}/\text{m}^3$  but no long term standard has been set for HF.

#### *Tier 1 assessment*

2.18 For the proposed CERC alone.

The modelled hourly mean value for hydrogen chloride (HCl) is  $0.5\mu\text{g}/\text{m}^3$  while the value for hydrogen fluoride (HF) is  $0.05\mu\text{g}/\text{m}^3$ . Using the stricter EPAQS value, the calculated safety factors for short -term exposure to hydrogen chloride and hydrogen fluoride respectively of 1500 and 3200. Since the benchmark for further consideration is a factor of 10 (See appendix 2) , it is evident that a tier 2 assessment is not needed for either hydrogen chloride or hydrogen fluoride. Estimated annual exposure to HCl has been modelled as  $0.0087.\mu\text{g}/\text{m}^3$ .

This compared with the standard gives a safety value of 2299. This is very much larger than the bench mark value of 100 for longer term exposures. Thus a tier 2 assessment is not needed.

### 2.19 Plus back ground values

Short- term background values for HCl are 3.4µg /m<sup>3</sup> and for HF are 2.0 µg /m<sup>3</sup>. The long term background level for HCl is 1.7ug/m<sup>3</sup>. Assuming that the contributions are additive, the total short term levels would be 3.9.ug/m<sup>3</sup> and 2.05ug/m<sup>3</sup> respectively. These levels are very much below the air quality standards (i.e. 0.52% and 1.28% of the respective standards).

#### *Summary of the risks from inhalation of HCl and HF*

2.20 (i) size of the safety margin. The safety margins for acute exposure are extremely large.

(ii) Impact on compliance with air quality standards. The proposed CERC will not have any significant impact on the compliance with the air quality standards. The NSAQ classification for both the impacts of HCl and HF would be neutral. It can be concluded that the health risk from the emission of hydrogen chloride and hydrogen fluoride from the proposed CERC will not constitute a significant health risk to the local population.

### **2.3.2 NITROGEN DIOXIDE**

#### *Sources of exposure*

2.21 Nitrogen dioxide is derived from nitric oxide, which is a product of combustion. In the UK as a whole around 50% of nitrogen oxides in ambient air are produced by road transport (see Chapter 1). The WHO Air Quality Guidelines (2000) identified that in cities, ambient levels of NO<sub>2</sub> can range from annual means of 10-50 parts per billion (ppb) (20-90 µg/m<sup>3</sup>). Hourly, the ranges can vary from 40 to 540 ppb (75-1010 µg/m<sup>3</sup>). Levels in many indoor premises tend

to be higher. If gas appliances are poorly vented, average values over a number of hours can reach 100 ppb with peak 1 hour values up to 1000 ppb.

#### *Summary of effects*

2.22 The principal target organ for nitrogen dioxide is the respiratory system (lungs) although irritation to other mucus membranes can also occur. In animal experiments, 1 to 6 months exposure at concentrations of 560-960  $\mu\text{g}/\text{m}^3$  is required to produce changes in lung structure, lung metabolism and lung defences against infection. These studies also indicate that high peak concentrations contribute more to the toxicity of  $\text{NO}_2$  than does an extended duration of exposure. Some individuals in the human population may, however, have a greater sensitivity to  $\text{NO}_2$  than the findings from these animal studies would imply.

#### *Basis for the standards*

2.23 The current UK standard is based on experimental exposure studies in humans, which have shown stimulation of inflammatory markers and heightened susceptibility to allergens. The current 200  $\mu\text{g}/\text{m}^3$  hourly value was set to protect particularly sensitive individuals "for acute exposures only very high concentrations ( $\sim 1000$  ppb) affect healthy people" (WHO, 2000). The EU Daughter Directive ambient air value for 2010 is 200  $\mu\text{g}/\text{m}^3$  hourly mean with a maximum of 18 permitted exceedences per year. The annual mean value is 40  $\mu\text{g}/\text{m}^3$  (21 ppb). This is based on World Health Organisation proposal (2000) of an annual average guideline of 21 – 26 ppb (40-50  $\mu\text{g}/\text{m}^3$ ). The WHO Report (2000) emphasised that while there is "no particular study or set of studies that support a numerical value" "the data base clearly indicates the need to protect the public from chronic exposures". The USEPA, (1997) has taken a very different approach to the one used by WHO. It did not set a short term value for  $\text{NO}_2$ , but using the same data as that available to UK and WHO experts sets an annual

mean which is far less stringent of 53 ppb ( $100 \mu\text{g}/\text{m}^3$ ). These differences between the UK / EU and the USA do not indicate discrepancies in the interpretation but rather reflect the differences in the degree of caution built into the standards used to protect public health.

2.24 The Department of Health has four descriptors of air quality for  $\text{NO}_2$ , i.e.:

Very good	0-49 ppb
Good	50-99 ppb
Poor	100-299 ppb
Very poor	$\geq 300$ ppb

The Department of Health states that “even when air quality is described as “very poor” very few health effects are likely to occur as a result of exposure to  $\text{NO}_2$  alone at the levels to be expected in the UK. However, mainly because urban pollution is a mix of components, levels of  $\text{NO}_2$  in excess of 300 ppb merit the issuing of clear advice to those suffering from respiratory disorders, e.g.: asthma. As a cautionary approach a general warning should be provided should  $\text{NO}_2$  levels rise above 600 ppb” (DEFRA, 2004).

#### *Tier 1 assessment*

2.25 For the proposed CERC alone.

The calculated maximum increment of  $\text{NO}_2$  on short term ground level concentrations is  $6-6\mu\text{g}/\text{m}^3$  over 1 hour. The standard as indicated above is  $200\mu\text{g}/\text{m}^3$ . This gives a safety factor of 30.3. This is considerably greater than the benchmark of 10 for acute exposure and therefore a Tier 2 assessment is not required. The annual average contribution of  $\text{NO}_2$  from the proposed CERC will be  $0.13\mu\text{g}/\text{m}^3$ , giving a safety factor of 308. As this value is considerably greater No further evaluation is required.

Plus background values

2.26 The background 1 hour levels of NO<sub>2</sub> is 15µg/m<sup>3</sup>. If this is added to the maximum modelled emissions from the proposed plant, a level of 21.6µg/m<sup>3</sup> would arise. Thus the total would be 10.8% of the standard. Thus a substantial safety margin will remain. The background annual value for existing air quality is 7.5ug/m<sup>3</sup>. Thus, the total would be 7.63% of the standard i.e. considerably below the standard of 40.

2.27 COMEAP Assessment.

An alternative way of assessing the risk from nitrogen dioxide is to use of COMEAP(Committee on Medical Episodes of Air Pollution) methodology. It is not certain whether the basis for their calculations is exclusive to nitrogen dioxide or whether there is contribution from sulphur dioxide and particulate matter as well. The analysis by ERM of the health impacts of nitrogen dioxide on the local population may be summarised as follows:

*Table 2.1 Estimated increased incidence of adverse effects due to Nitrogen dioxide emissions from the CERC*

Effect on health	Background Incidence of the effect in the population per annum	Extra cases per million per annum of the population due to the operation of the CERC	Estimated total Number of the population affected throughout the lifetime of the proposed plant+
Deaths brought forward	7690	0.0041	0.123

Respiratory hospital admissions	7800	0.0045	0.136
Cardiovascular admissions	14000	0.0277	0.833

*+ based on the plant operating for 30 years.*

These estimates are conservative ones. It is evident that there will be no significant impact of the nitrogen dioxide emissions from the proposed plant on the local population.

*Summary of the risks from the inhalation of nitrogen dioxide*

2.28 (i) size of the safety margin. The safety margin for acute exposure is substantial and bearing in mind that it is calculated from conservative air quality modelling indicates that the impact on the health of the local population will be negligible.

(ii) Impact on compliance. The proposed plant will not have any significant impact on the compliance with the air quality standards. The NSAQ classification would be neutral.

(iii) analysis using COMEAP methodology indicates insignificant effects.

It can be concluded that in health terms. This indicates that the contribution of the short-term and long-term ground level concentrations of NO<sub>2</sub> from the operation of the proposed CERC is not significant.

**2.3.3 SULPHUR DIOXIDE**

*Sources of exposure*

2.29 The main sources of sulphur dioxide are combustion processes including transport. Since the 1980's sulphur dioxide emissions have been reduced

considerably in Europe and the UK. However, in particular areas there may be specific sources of sulphur dioxide, such as brick works (WHO, 2000).

#### *Summary of effects*

2.30 Short-term levels of sulphur dioxide can give rise to health effects such as shortness of breath, wheezing and an increase in airways resistance. These effects can be enhanced by physical exercise. Individuals with respiratory allergies, such as asthma and hay fever, tend to be more susceptible to the effects of sulphur dioxide.

#### *Basis for the standards*

2.31 The short-term standard is based on "the minimum concentrations associated with adverse effects in the most extreme circumstances" (WHO, 1999) i.e.: in asthmatic patients.

-The 15 minutes mean is  $266 \mu\text{g}/\text{m}^3$  – not to be exceeded more than 35 times a year.

- The one hour mean is  $350 \mu\text{g}/\text{m}^3$  – not to be exceeded more than 24 times a year.

- The daily mean of  $125 \mu\text{g}/\text{m}^3$  - not to be exceeded more than 3 times a year.

An annual value of  $50\mu\text{g}/\text{m}^3$  although the effects of sulphur dioxide are considered to be primarily acute.

#### *Tier 1 assessment*

#### 2.32 Proposed CERC alone.

The modelled 15 minute value of  $2.04\mu\text{g}/\text{m}^3$  may be compared against the stringent UK 15 minute value of  $266\mu\text{g}/\text{m}^3$ . This gives a calculated safety factor of 130. The modelled 1 hour value of  $1.57\mu\text{g}/\text{m}^3$  giving a safety factor of 233. The modelled 24 hour value of  $0.41\mu\text{g}/\text{m}^3$  giving a safety factor of 305. In each case the safety factor is considerably larger than the acute bench mark figure of 10

consequently a tier 2 assessment is not needed. The annual value of 0.017 ug/m<sup>3</sup> which gives a safety factor of 2941. Using the appropriate benchmark value of 100 a tier 2 assessment is not required.

Plus background values.

2.33 The short term background air quality value is 18ug/m<sup>3</sup> giving a total of 19.57 ug/m<sup>3</sup> assuming levels will be additive. This is well below the relevant standard (5.6% ).The annual mean background exposure is 8.8ug/m<sup>3</sup> giving a total of 8.8ug/m<sup>3</sup> . This is 17.6% of the relevant standard.

COMEAP Assessment

2.34 The table below summarises the health impacts on the local population derived from the calculations carried out by ERM based on the COMEAP (Committee on Medical Episodes of Air Pollution) calculation method:

*Table 2.2 Estimated increased incidence of adverse effects due to sulphur dioxide emissions from the CERC*

Effect on health	Incidence of the effect in the population per annum	Extra cases per million of the population per annum due to the CERC	Estimated total number of the population affected throughout the lifetime of the proposed plant+
Non traumatic deaths brought forward	7690	0.0008	0.024
Respiratory hospital	7800	0.0010	0.03

admissions			
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+ based on the plant operating for 30 years.

*Summary of the risks from inhalation of SO2*

2.35 (i) size of the safety margin. The safety margin for both acute and long term exposure is substantial.

(ii) impact on compliance. The proposed CERC will not have any significant impact on the compliance with the air quality standards. The NSAQ classification would be neutral.

(iii) Analysis using COMEAP methodology indicates no significant impact in terms of either morbidity or mortality.

2.36 It can be concluded that the emission of sulphur dioxide from the proposed plant will not constitute a health risk to the local population. In health terms the contribution of the short-term and long-term ground level concentrations of sulphur dioxide from the operation of the proposed plant is not significant.

**2.3 4 CARBON MONOXIDE (CO)**

*Principal sources of exposure*

2.37 Carbon monoxide is a gas produced by incomplete combustion. It is generated by motor vehicles and a variety of industrial and domestic combustion processes. It is also a significant component of cigarette smoke. It is a natural chemical as it is produced in small amounts by the tissues in the human body. In respect of emissions from the proposed CERC the only relevant route of exposure is by inhalation. Typical annual average concentrations of carbon monoxide in the UK, except close to busy roads, are about 1ppm (i.e.

1.45mg/m<sup>3</sup>). During significant air pollution incidents the level of CO can exceed 15ppm. This can also occur inside vehicles in heavy traffic.

#### *Summary of effects*

2.38 Carbon monoxide is rapidly absorbed through the lung. It binds strongly to haemoglobin present in red blood cells to form carboxyhaemoglobin, and to other haemoproteins in the body. Haemoglobin is the principal means by which oxygen is transported in the body. Carbon monoxide binding interferes with this essential oxygen transport function. If a sufficient proportion of the haemoglobin/haemoproteins are bound to carbon monoxide the body will suffer from oxygen deficiency. Other haemoproteins play an important role in energy generation in the body. Should the binding persist acute adverse effects can occur to the brain, cardiovascular system, exercising muscle and developing foetus (WHO, 2000).

2.39 For each of the above effects there is a threshold exposure level for the adverse effects of carbon monoxide indeed it is possible that very low levels may be beneficial. Healthy, non-smoking individuals typically have carboxyhaemoglobin levels of between 0.5 and 1% whereas heavy smokers may have levels of 5-10%. There is a very extensive literature on CO toxicity. Carbon monoxide causes around 60 deaths a year in the UK and some 500 admissions to hospital. Most arise due to accidental fires or faulty domestic heating/inadequate ventilation. In the past CO was used for suicides.

2.40 Individuals with heart/coronary conditions represent a potentially at risk group. Others include individuals with chronic lung disease and the developing foetus. There remains uncertainty as to whether long-term exposure to carbon monoxide increases the likelihood of developing cardiac disease. At present there

is no evidence to confirm this and the focus is therefore on acute exposure levels.

#### *Basis for the standard*

2.41 The ambient air standards are based on the levels and duration of exposure necessary to cause 2% of carboxyhaemoglobin in the blood of the population. This level is on the borders of causing a physiological effect in sensitive individuals, thus it is not a true threshold. The relevant standards are derived directly from WHO guidelines (2000):

- 100mg/m<sup>3</sup> for 15minutes
- 30mg/m<sup>3</sup> for 1 hr
- 10mg/m<sup>3</sup> for 8 hrs.

The standard is thus set on a minor effect.

#### *Tier 1 assessment*

##### 2.42 Proposed CERC alone.

The modelled maximum 8hr value as a running mean for carbon monoxide is 1.6ug/m<sup>3</sup>. The corresponding standard is 10,000ug/m<sup>3</sup>. This gives a safety value of 6250. As the effect considered is an acute one, the bench mark for a tier 2 assessment is a safety margin of less than 10. It is evident that a tier 2 assessment is not needed for carbon monoxide.

##### Plus background values

2.43 If we consider the proposed CERC plus existing air quality: the current air quality value is 192µg/m<sup>3</sup>. Thus the air quality levels assuming that the contribution from the proposed plant is additive would be up to 193.6ug/m<sup>3</sup>. This is 0.019% of the relevant standard.

#### *Summary of the risks from inhalation of carbon monoxide*

2.44 (i) size of the safety margin. The safety margin is extremely large  
(ii) impact on compliance. The proposed plant will not have any significant impact on the compliance with the air quality standards for carbon monoxide. The NSAQ classification would be neutral. It can be concluded that the emission of carbon monoxide from the proposed plant will not constitute a health risk to the local population.

### **2.3.5 PARTICULATE MATTER**

#### *Sources of exposure*

2.45 PM<sub>10</sub> is defined as suspended particulate matter (SPM) with a median diameter of less than 10µM. It comes from a very wide variety of sources and is chemically heterogeneous. Thus particles may incorporate metals and other inorganic substances such as salt, carbon, hydrocarbons, dioxins and/or polycyclic hydrocarbons. PM10 measurements include both particles classified as PM2.5 and ultra-fine particles (also called nanoparticles). There is an ongoing debate on the size and nature of the particles that should be monitored for public health protection purposes. However, it is PM<sub>10</sub> which are measured because the equipment is widely available and because the present standards set by the Europe Union refer to PM<sub>10</sub>. At present our understanding of the relationship between airborne particle levels and health impacts is based on PM<sub>10</sub>.

2.46 In terms of ambient air, the amount of ultra-fine particles is rather similar in urban and rural areas, with as much as 10<sup>6</sup> to 10<sup>8</sup> nanoparticles per litre of air depending on conditions. In rural areas these particles mostly originate from the oxidation of volatile compounds of biogenic or anthropogenic origin (for example, fuel burning). In urban areas, the primary sources of these particles are diesel engines or cars with defective or cold catalytic converters. Photo-oxidation processes also lead to significant numbers of ultra-fine (nanoparticles) in urban areas, which are generated as by products of combustion and other industrial

processes. Epidemiology findings are used to calculate the potential health effects (short-term) increases in the ground levels of PM<sub>10</sub> for the local population as a whole. The total levels of exposure to particulates' in ambient air' is tending to fall over time as tighter regulations take force.

2.47 However, since most people spend the majority of their time indoors particles in indoor air represent a very important source of their exposure. Important sources of fine particulate matter indoors include: gas and electricity cooking (Dennekamp *et al* 2001(a), Mitsakou *et al* 2007), domestic heating (particularly from wood fires), use of candles and passive tobacco smoke. Dennekamp *et al* (2001,a) examined the impact of cooking on airborne levels of ultra-fine particles (UFP). The levels generated by gas and electric cooking appliances were found to be similar to those measured outdoors during incidents of very high pollution (i.e.: increased levels of > 20 µg/m<sup>3</sup> or more). In a survey of around 500 offices, levels of respirable particles (which roughly approximate to PM<sub>10</sub>) ranged from 28-133 µg/m<sup>3</sup>. The air in a normal room in domestic premises may contain 10,000 to 20,000 nanoparticles per cm<sup>-3</sup>

#### *Summary of effects.*

2.48 The adverse effects of high level exposure to PM<sub>10</sub> can be summarised as follows:

- \* Respiratory disease ( possibly even lung cancer );
- \* Cardio-vascular system effects;
- \* Possible adverse reproductive effects.

#### *Basis for the standards*

2.49 A 24 hour value of 50 µg/m<sup>3</sup> has been adopted for PM<sub>10</sub> by the EU with an annual value of 40 µg/m<sup>3</sup>. However, WHO (1999) set no specific guidelines for either short- term or long- term exposure to PM<sub>10</sub> because" no obvious

exposure concentration and duration could be judged a threshold". There is an ongoing debate about the regulatory value of setting specific standards for smaller particle sizes.

*Tier 1 assessment –*

Proposed CERC alone

2.50 Based on the consideration of the HMEI the calculated maximum increment of PM<sub>10</sub> on short term (24 hour) ground level concentrations is 0.035 µg/m<sup>3</sup>. In relationship to the standard, this is a very small contribution, giving a calculated safety factor of 1429. The calculated annual contribution of the plant to the background levels of PM<sub>10</sub> is 0.0087µg/m<sup>3</sup> (calculated safety factor 4598). On the basis of these large safety factors there is no need for Tier 2 assessment.

Plus background levels.

2.51 The existing 24- hour air quality value is 41ug/m<sup>3</sup>. As a worst case the assumption is made that the contribution from the CERC will be additive. Under this scenario the total levels of PM10 in ambient air would be 41.04ug/m<sup>3</sup>. This is a very small change from the existing levels and substantially below the percentile standard of 50 (82% of the standard). The annual background air quality value 23ug/m<sup>3</sup> which if added to the contribution from the plant alone would give a value of 23.009ug/m<sup>3</sup> (57.5.% Of the standard). Thus there will be no significant impact on the air quality standard and can be classified in NAQS terms as neutral.

2.52 COMEAP assessment

The estimates in the Air quality part of the ES (given in their appendix E) may be summarised as follows:

*Table 2.3 Estimated increased incidence of adverse effects due to the emission of particulates from the CERC*

Effect on health	Incidence of the effect in the population per annum	Extra cases per million of the population per annum due to the CERC	Estimated total number of the population affected throughout the lifetime of the proposed plant+
Chronic bronchitis	7690	0.0058	0.174
Respiratory hospital admissions	7800	0.0009	0.028
Cardiovascular admissions	14000	0.0009	0.028
GP consultations for asthma	64130	0.0166	0.499

*+ based on the plant operating for 30 years.*

2.53 These estimates are conservative ones. It is evident that there will be no significant impact on the health of the local population from exposure to particulate matter.

*2.54 Summary of the risks from the inhalation of PM10*

(i) size of the safety margin. The safety margin for acute exposure is very substantial

.(ii) impact on compliance. The proposed CERC will not have any significant impact on the compliance with the air quality standards. The NSAQ classification would be neutral.

(iii) Analysis using COMEAP methodology. This indicates insignificant effects on the health of the local community.

It can be concluded that the emission of particulate matter from the proposed CERC will not constitute a health risk to the local population.

## **2.4 CHEMICALS OF INTEREST WITH PRINCIPALLY LONG -TERM EFFECTS**

Chemicals of interest in this category are: various metals, dioxins, polycyclic aromatic hydrocarbons and volatile organic chemicals such as benzene. There is no common long term effect of these chemicals.

### **2.4.1 METALS**

2.55 An important determinant of the toxic behaviour is its biological half-life which is a measure of its persistence in the body, manganese, cobalt, chromium, iron, calcium, selenium. Others exert at least part of their toxic effects by competing with these metals. The principal cause of toxicity of metals is the formation of a bond between the ionic form of the metal and an enzyme(s) or other essential macromolecule(s) or cell or organelle membrane. A number of metals are essential for human health, for example, sodium, potassium, magnesium by its speciation (physical and chemical form). Each metal has its own characteristic toxicity profile. Therefore the toxic properties of each are addressed briefly in a subsequent section of this report.

2.56 Several metals have been found to be carcinogenic as a result of investigations among workers in the metal industries these include: arsenic,

cadmium, chromium VI, nickel, beryllium and possibly lead (see IARC monographs). Caution is needed in the extrapolation of such findings to other exposure situations because often the speciation is not well defined and the dose response relationship is unclear. It appears to be the case that in most if not all cases the initiation of the cancer is not by a genotoxic mechanism.

#### **2.4.1.1 ARSENIC**

##### *Sources of exposure*

2.57 Arsenic is widely distributed in the environment. Arsenic may be released into the air by various industrial processes, particularly those involving coal burning. Around smelting works arsenic concentrations of 0.01 to 0.075  $\mu\text{g}/\text{m}^3$  has been found. Arsenic concentrations in food are typically around 1ppm although higher values may occur, particularly in shell fish.

##### *Summary of effects*

2.58 Arsenic reacts with sulphhydryl groups on proteins and thereby inhibits cellular respiration. Acute effects in animals include cardiac dysfunction, electrolyte disturbance, coma and even death. There may also be skin lesions, sensory loss and respiratory distress. Symptoms of chronic exposure include skin and mucous membrane damage, effects on the respiratory and nervous system. Much is known about the acute toxicity of arsenic in man, because arsenic was favoured by murderers in the past. Epidemiology studies in the workplace have shown that arsenic is a lung carcinogen following chronic, high level inhalation exposure, although this effect is not seen in experimental animals. Nonetheless, arsenic is genotoxic in animal studies. Skin cancer has also been observed in humans. Arsenic has been shown to be a teratogen in experimental animals but not in man.

##### *Basis for the standards*

2.59 A recently introduced standard for long-term exposure to arsenic is 0.006  $\mu\text{g}/\text{m}^3$ . The short-term (1 hour standard) is 15  $\text{ug}/\text{m}^3$ .

The unit risk factor used to calculate lifetime risk is 0.0043  $\text{ug}/\text{m}^3$ . It should be noted that this and other unit carcinogenic risk factors are worst case estimates which assume a linear extrapolation from the effects at high exposures.

*Tier 1 assessment -*

Proposed CERC alone

2.60 The 1 hour value is 0.0028 $\text{ug}/\text{m}^3$  which when compared with the acute standard gives a very large safety margin of 5357. It is evident that a tier 2 assessment is not needed for the acute exposure to arsenic. The modelled annual mean value is 0.000048 $\mu\text{g}/\text{m}^3$ . This gives a safety factor of 125. Thus a tier 2 assessment is not required.

Plus background levels

2.61 The 1 hour background value is 0.00048  $\text{ug}/\text{m}^3$ . This when added to the modelled ground level value from the proposed plant gives a value of 0.0033 $\text{ug}/\text{m}^3$ . This is well below the air quality standard of 15 $\text{ug}/\text{m}^3$  (0.022% of the standard). The background air quality annual value is 0.00024 $\text{ug}/\text{m}^3$  which is low in comparison with other UK urban sites. When added to the worst case contribution from the proposed plant a value of 0.00029  $\text{ug}/\text{m}^3$  is calculated. This value is considerably below the relevant annual standard of 4 $\text{ug}/\text{m}^3$  (4.8% of the standard).

Life time risk estimate

2.62 The calculated lifetime risk is virtually 1 in a million (ie  $1.1 \times 10^{-6}$ ). Bearing in mind this is a worst case estimate e.g. it assumes 70 years of exposure to the maximum ground level concentration, it more than meets the criteria for an acceptable risk 1 in 14,286 ( $70 \times 10^{-6}$ ).

*Summary of the risks from inhalation of arsenic*

2.63 (i) size of the safety margin. The safety margin for acute exposure is substantial;

(ii) Impact on compliance. The proposed CERC will not have any significant impact on the compliance with the air quality standards.

iii) Analysis using life time risk data. The estimated worst case risk is very small in comparison with many other forms of risk.

It can be concluded that in health terms the contribution of the short-term and long-term ground level concentrations of arsenic from the operation of the proposed plant is not significant.

#### **2.4.1.2 CADMIUM**

*Sources of exposure*

2.64 There are many sources of exposure to cadmium that can result in environmental contamination. Cadmium is widely used in the manufacture of alkaline batteries, paints and plastics for example. It may also be present in fertilisers, sewage sludge and compost. Air concentrations up to 5000  $\mu\text{g}/\text{m}^3$  have been found at some industrial metal processing sites. These values can be compared with typical rural and urban levels of 0.001-0.005  $\mu\text{g}/\text{m}^3$  respectively. In soil levels in unpolluted areas are typically below 1ppb. Overall, the most important source of cadmium exposure of the public is through the diet. Various foods (meat, fruit and fish) may contain 1-50ppb. However, shell fish may accumulate higher levels. Cigarette smoke is a further source of individual cadmium exposure.

*Summary of effects*

2.65 High levels of acute inhalation of cadmium may lead to coughing and

tightness of the chest, with subsequent oedema. Chronic inhalation of cadmium can result in anaemia and renal disease, emphysema, liver damage. Cadmium may also damage bones, suppress testicular function and cause hypertension. Cadmium is carcinogenic in some animal models. However, the evidence that cadmium is carcinogenic in humans is weak. Cadmium can be considered as at the most, a weak genotoxic agent. It is probably appropriate therefore to consider it to be a non-genotoxic carcinogen

#### *Basis for the standards*

2.66 The following guideline values are in use in environmental standard (EAL):-

- Short term EAL  $1.5 \mu\text{g}/\text{m}^3$
- Long term (annual) EAL  $0.005 \mu\text{g}/\text{m}^3$

The unit risk from lifetime exposure is  $0.0018\mu\text{g}/\text{m}^3$

#### *Tier 1 assessment-*

##### Proposed CERC alone

2.67 The modelled 1 hour value is  $0.0012\mu\text{g}/\text{m}^3$ . This gives a calculated safety factor of 1250. Thus a tier 2 assessment is not required.

The modelled maximum annual level value is  $0.000022\mu\text{g}/\text{m}^3$ . The calculated safety factor is therefore 227. Thus, a tier 2 assessment is not required.

##### Plus background levels

2.68 The 1 hour background value is  $0.00018\mu\text{g}/\text{m}^3$  which if added to the potential worst case contribution from the proposed plant would give a value of  $0.0014\mu\text{g}/\text{m}^3$  (Equivalent to 0.09% of the acute standard).

Annual background value is  $0.00009 \mu\text{g}/\text{m}^3$ . This if added to the potential contribution from the proposed plant would give a total value of  $0.00011\mu\text{g}/\text{m}^3$  (Equivalent to 2.2 % of the acute standard). It is clear that even using the worst case air quality modelling assumption the contribution from the plant will have no

impact on the compliance with the standard.

#### Life time risk estimate

2.69 The calculated lifetime risk is 1 in 82 million ( $1.22 \times 10^{-8}$ ). This is a worst case estimate and therefore clearly meets the criteria for an acceptable risk.

#### *Summary of the risks from the inhalation of cadmium*

2.70 (i) size of the safety margin. The safety margin for acute exposure is substantial

(ii) Impact on compliance. The proposed CERC will not have any significant impact on the compliance with the air quality standards. The NSAQ classification would be neutral.

(iii) Life time risk estimate. The estimated risk based on worst case consideration indicates that there is no significant risk.

It can be concluded that in health terms the contribution of the short-term and long-term ground level concentrations of cadmium from the operation of the proposed CERC is not significant.

### **2.4.1.3 CHROMIUM**

#### *Sources of exposure*

2.71 The main sources of chromium in ambient air include the steel industry, cement industry and combustion of fossil fuels. In rural areas chromium levels are usually less than  $10 \text{ ng/m}^3$  while in industrial cities levels may approach  $100 \text{ ug/m}^3$ . Chromium can exist in two forms Cr III and Cr VI. Typically, the ratio in air is 2:1. This is because Cr VI released to air is reduced by other pollutants present. Chromium levels in soil can range from 0.1 to 400ppm with an average value of around 50ppm. In food most important sources of chromium are meat, vegetables and unrefined sugar. Most foods have less than 100ppb. Beer, spirits, herbs and spices may have different chromium levels.

### *Summary of effects*

2.72 Chromium III is an essential element for blood cell production. The toxicity of chromium is mainly associated with Cr VI. The main toxic effect is on the kidneys. However effects on the liver and brain have also been reported. Chromium is a lung carcinogen, in experimental animals following chronic airborne exposure. In humans chronic inhalation of high levels of chromium can result in ulceration of the nasal septum as well as ulceration of other skin surfaces and damage to the respiratory and immune systems. Chromium is a lung carcinogen by inhalation. Chromium is genotoxic in a range of tests and should be considered therefore to be a genotoxic carcinogen.

### *Basis for the standards*

2.73 The following guideline values (worst case assumed of most of chromium exists as chromium VI) are in use:

- Short term EAL 3.0  $\mu\text{g}/\text{m}^3$
- Long term EAL 0.1  $\mu\text{g}/\text{m}^3$

The unit lifetime risk factor for cancer is 0.012ug/m3

### *Tier 1 assessment*

2.74 Proposed CERC alone The modelled 1 hour value for chromium is 0.0028 ug/m<sup>3</sup> giving a safety factor of 1071. This is a very large safety factor thus a tier 2 assessment is not needed. The modelled annual value for chromium is 0.000048  $\mu\text{g}/\text{m}^3$  which leads to a safety factor of 2083. A tier 2 assessment is therefore not required.

### Proposed CERC plus background air quality

2.75 The 1 hour background air quality is 0.0068ug/m<sup>3</sup> when added to the contribution from the proposed plant the overall air quality level could reach

0.0069ug/m<sup>3</sup>. This is 0.23% of the standard and will therefore have a neutral impact on air quality. The annual background air quality is 0.0034 ug/m<sup>3</sup> ,which is low in comparison with measurements at other UK urban sites. When added to the contribution from the proposed plant the overall air quality level could reach 0.0034ug/m<sup>3</sup>. This is 3.4% of the standard and therefore of no significance (neutral) in terms of overall impact on the air quality

#### Life time risk estimate

2.76 The calculated lifetime risk is 1 in 250 million (4x10<sup>-9</sup>). This clearly meets the criteria for an acceptable risk.

#### *Summary of the risks from chromium VI inhalation.*

2.77 (i) Size of the safety margin. The safety margin for both acute and annual exposure is very substantial.

(ii) Impact on compliance. The proposed CERC will not have any impact on the compliance with the air quality standards. The NSAQ classification would be neutral.

(iii) Lifetime risk The worst case risk estimate indicates that the impact on health will be insignificant.

In health terms the contribution of the short-term and long-term ground level concentrations of chromium from the operation of the proposed plant is not significant.

#### **2.4.1.4 LEAD**

##### *Sources of exposure*

2.78 In the last century the main general source of airborne lead was leaded petrol. Following the ban on the use of lead additives in petrol this is no longer the case. There are many other sources of lead exposure (from smelting works,

to lead piping for drinking water and lead supported wicks in some candles).

Particle size, as for many other metals, has a crucial influence on the bioavailability of airborne lead. Lead in air range from  $0.05 \mu\text{g}/\text{m}^3$  in remote areas of the world to  $10 \mu\text{g}/\text{m}^3$  near some old-fashion smelting works. Typical levels in the UK are  $0.05 \mu\text{g}/\text{m}^3$  in rural areas and  $0.01 \mu\text{g}/\text{m}^3$  in urban areas. Lead from waste disposal in the UK constitutes about 1% of the total lead emitted.

#### *Summary of effects*

2.79 The main source of human exposure to lead is the diet, typically 97% of the total exposure. Lead binds to SH groups in proteins. Toxic effects include: impaired renal function, anaemia and loss of central nervous system functioning. Young children are a particularly vulnerable to lead exposure. There is some debate as to whether or not lead is carcinogenic.

#### *Basis for the standards*

2.80 The Environmental Agency's long-term standard (EAL) is  $0.25 \mu\text{g}/\text{m}^3$   
The USA unit risk factor for cancer is  $1.2 \times 10^{-5}$

#### *The Tier 1 assessment-*

##### Proposed CERC alone

2.81 The modelled annual value for lead is  $0.000048 \mu\text{g}/\text{m}^3$ . When compared with the standard there is a safety factor of 5208. Thus no tier 2 assessment is required.

##### Proposed CERC plus background air quality

2.82 The annual background air quality value is  $0.00283 \mu\text{g}/\text{m}^3$  which when added to the proposed plant contribution gives a total of  $0.0029 \mu\text{g}/\text{m}^3$ . This represents 1.16.% of the standard thus it will have no effect on compliance with the standard.

#### Life time risk estimate

2.83 The calculated lifetime risk is 1 in 250 billion ( $4 \times 10^{-12}$ ). This undoubtedly meets the criteria for an acceptable risk.

#### *Summary of the risks from lead inhalation*

2.84 (i) Size of the safety margin. The safety margin for annual exposure is very large.

(ii) Impact on compliance. The proposed plant will not have any significant impact on the compliance with the air quality standards. The NSAQ classification would be neutral.

(iii) Lifetime risk estimate indicates an insignificant risk. It can be concluded that there will be no implications for the health of the local community from lead emissions from the proposed CERC.

#### **2.4.1.5 MERCURY**

##### *Sources of exposure*

2.85 Mercury is emitted to the atmosphere particularly by re-evaporation of previously deposited mercury. The main anthropogenic source of mercury is combustion. In Europe in rural areas, remote from industrial activity, levels of 2-4 ng/m<sup>3</sup> are not uncommon. In urban areas typical levels are between 6-16ng/m<sup>3</sup>. Concentrations of mercury in most food stuffs are below 20ng/gram fresh weight of food. In fish (such as tuna), levels of mercury of between 50 and 1400ng/gram fresh wt may be encountered. The principal sources of mercury in municipal waste are electrical switches and lighting components, paint residues and thermometers.

##### *Summary of effects*

2.86 Acute effects of high level airborne exposure to mercury are severe airway irritation and pulmonary oedema. Following chronic exposure the kidney and the central nervous system are the main targets. Mercury is not a carcinogen .

#### *2.87 Basis for the standards*

The following guideline values are in use:

1 hour - 7.5ug/m<sup>3</sup>; Long term EAL - 0.25 µg/m<sup>3</sup>

#### *2.88 The Tier 1 assessment-*

##### Proposed CERC alone

The modelled 1 hour value for mercury is 0.0025 µg/m<sup>3</sup>. The safety factor for the maximum 1 hour ground level exposure is therefore 3000. A tier 2 assessment is not required. The modelled annual value for mercury is 0.000044 µg/m<sup>3</sup> which results in a safety factor of 5682. Thus, a tier 2 assessment is not needed.

##### 2.89 Proposed CERC plus background air quality

The 1 hour background air quality is 0.00026 ug/m<sup>3</sup>. If the contribution of the proposed CERC is added the total air quality level may reach 0.00017ug/m<sup>3</sup>. This is 0.068% of the relevant standard.

#### *2.90 Summary of the risks from inhalation of mercury*

- (i) size of the safety margin. The safety margin for acute exposure is substantial.
- (ii) Impact on compliance. The proposed plant will not have any significant impact on the compliance with the air quality standards. The NSAQ classification would be neutral. It can be concluded that in health terms the contribution of the short-term and long-term ground level concentrations of chromium from the operation of the proposed plant is not significant.

### **2.1.1.6 NICKEL**

#### *Sources of exposure*

2.91 Nickel is used in the steel, ceramics, pigment and battery industries. Nickel is also released to air through the combustion of fossil fuels. Atmospheric levels of nickel in large cities and industrial areas may vary between 120 and 170  $\mu\text{g}/\text{m}^3$ . In contrast, in rural and suburban areas values of 6 and 19  $\mu\text{g}/\text{m}^3$  have been found. Nickel is also present in tobacco smoke. Nickel content in soils varies widely (3-1000ppm). In food, typically levels of nickel range from 0.7 to 34ppm but in nuts and legumes the levels are much higher (up to 230ppm).

#### *Summary of effects*

2.92 There is a debate whether nickel is an essential element in man. Nickel can cause dermatitis as a consequence of contact with nickel jewellery, dental prosthesis and other objects. Asthma and nasal perforation have been observed in the nickel plating industry where high levels of airborne nickel are common. Nickel is a lung carcinogen in animal models. It is also a lung carcinogen in man. However nickel and its compounds are not potent genotoxins. Nickel is widely considered as a non-genotoxic carcinogen.

#### *Basis for the standards*

2.93 The 1 hour standard is 30 $\mu\text{g}/\text{m}^3$ . The annual standard is 0.02  $\mu\text{g}/\text{m}^3$

#### *Tier 1 assessment -Proposed CERC alone*

2.94 The modelled 1 hour value for nickel is 0.0028  $\mu\text{g}/\text{m}^3$ . The resultant safety factor is thus 1071. A tier 2 risk assessment is not required.

The modelled annual mean value for nickel is 0.000048  $\mu\text{g}/\text{m}^3$ . This gives a calculated safety factor of 417. It is not necessary to carry out a tier 2 risk assessment.

*Proposed CERC plus background air quality*

2.95 The 1 hour background air quality is 0.003ug/m<sup>3</sup>. When this is added to the contribution from the proposed CERC 0.003.063ug/m<sup>3</sup>. This represents 0.3% of the standard. Thus the impact of any nickel emissions to the acute air quality is neutral. The annual background air quality is 0.00153ug/m<sup>3</sup>. When the contribution of the plant is added the total could reach 0.00153ug/m<sup>3</sup>. Thus the overall air quality will is at most reach 6.5% of the standard. Again the impact may be categorised as neutral.

*Summary of the risks from the inhalation of nickel.*

2.96 (i) size of the safety margin. The safety margin for acute exposure is very large and for annual exposure substantial. (ii) Impact on compliance. The proposed plant will not have any significant impact on the compliance with the air quality standards. The NSAQ classification would be neutral. In health terms the contribution of the short-term and long-term ground level concentrations of nickel from the operation of the proposed CERC is not significant.

#### **2.1.4.7 THALLIUM**

*Sources of exposure*

2.97 Thallium is present in nearly all environmental media. In the earth crust a mean thallium concentration of 0.1-1.7 mg/kg is found mainly in sulphides ores along with copper, zinc, coal and lead (Peters and Viraragavan, 2005). The largest anthropogenic sources of thallium are combustion processes involving coal, sulphide ores, ferrous and non-ferrous smelting. Thallium is used in the semiconductor and laser industry. Coal-burning power plants, cement production, use of phosphate fertiliser, brick works and car emissions also produce thallium emissions. Thallium is volatile at high temperatures and easily released into the atmosphere.

#### *Summary of effects*

2.98 Thallium ingestion is the most significant route of exposure of non-occupational nature. Acute poisoning of thallium is a result of accidental dispersal (often in occupational environment) or a deliberate attempt to poison. The target organ in thallium poisoning is the central and peripheral nervous systems. Acute effects are gastroenteritis, polyneuropathy and alopecia. Chronic occupational exposure to thallium can result in effects on the nervous system (Peter and Virarghavan, 2005). This may be manifested in peripheral neuropathy.

#### *Basis for the standards*

2.99 The EA has set values of 30 µg/m<sup>3</sup> as the 1 hour standard and 1 µg/m<sup>3</sup> as the long-term standard.

#### *Tier 1 assessment -Proposed plant alone*

2.100 The 1 hour modelled value is 0.0012 µg/m<sup>3</sup>. This gives a safety factor of 25000. A tier 2 assessment is thus not needed. The modelled value for thallium 0.000022 µg/m<sup>3</sup> for the annual ground level concentration. This gives a safety factor of 45455. A tier 2 assessment is therefore not required.

#### *Summary of the risks from the inhalation of thallium*

2.101 Size of the safety margin. The safety margin for acute exposure is extremely large. In health terms the contribution of the short-term and long-term ground level concentrations of thallium from the operation of the proposed CERC is not significant.

### **2.4.2 DIOXINS**

#### *Sources of exposure*

2.102 Dioxins are emitted from many sources including vehicles, bonfires,

domestic and industrial combustion process. Sidhu *et al* (2005) identified uncontrolled burning of waste as a major source of dioxin emissions. The main route of dioxin exposure is through food.

2.103 Each year many new publications on aspects of the toxicology and epidemiology of dioxins appear. Dioxin levels in food and the general environment throughout the western world have been falling, but public concern and new research results in frequent evaluations of acceptable levels of these chemicals (by national and international bodies). Many of the new studies showing effects of dioxins come from the Far East, where general pollution levels have been rising.

2.104 The overall conclusion of many studies is that at body burden levels, within an order of magnitude of those in the general western population, subtle adverse effects may occur. However it should be noted that body burden of dioxins have been falling over the past two decades in Europe and this trend appears to be continuing. This is confirmed by a DEFRA review (2004), which stated that even in a rural environment any increased deposition of dioxins from an incinerator would be too small to be of concern with regard to health.

#### *Summary of effects*

2.105 The consensus view is that dioxins increase the risk for all cancers combined. However, the magnitude of this increase appears to be low and no statistically significant increase in any particular type of cancer has been identified. A substantial dioxin exposure leads to elevated incidence of cardiovascular disease and diabetes although other studies challenge this conclusion. In workers a persistent skin condition (termed chloro-acne) may occur handling contaminated materials. In animals dioxin is a teratogen. There is clear evidence of immune suppression following exposure as a foetus. However,

there is no good evidence among exposed communities such as the Sevaso population of impaired immune competence. In other human studies there is some rather limited evidence of effects on the immune system. There is insufficient information from human studies to determine the threshold level directly. In a number of studies dioxins were found to be endocrine disrupters, a property it has in common with a number of persistent polyhalogenated aromatic chemicals.

#### *Basis for the Standard*

2.106 Dioxins are generated from many processes as trace contaminants. The levels of individual congeners (related structures) of dioxins vary considerably by source. In view of this fact a standardised form of expressing the overall toxicity is required. The internationally accepted form is to use Toxicological Equivalents (TEQs), which is based on an allocation of a toxicity rating to each congener (so called toxic equivalency factor TEF)). The most potent - 2, 3, 7, 8-tetrachlorodibenzodioxane (TCDD) - is assigned a value of 1 and the remaining 17 dioxin congeners with chlorine in the 2, 3, 7 and/or 8 positions are assigned a value lower than 1. A TEQ for a particular source can be calculated using measurements of the percentage of each congener in the total amount of dioxin, and its concentration.

2.107 Ingestion is the primary exposure route in relation to dioxins. In 1998 the World Health Organisation (WHO) proposed a revised Tolerable Daily Intake (TDI) of 1-4 pg/kg body weight for dioxins, on the basis of a comprehensive review of the health effects of these chemicals (Van Leeuwen and Younes, 1998). A monthly intake standard of 70pg/kg body weight per month was proposed by JECFA. The WHO emphasised, in its analysis of the risks from dioxin exposure at different levels that (as far as the protection of the public is concerned), and it is the exposure over a prolonged period of time (rather than short term fluctuations

in levels) that is important. Most countries have not set ambient air quality guidelines or standards for dioxins in air and have concentrated on applying the WHO standards for dioxins in food instead. However, Japan uses an annual ambient air standard of 0.6 pg/m<sup>3</sup> (Environmental Panel, 1999) while in the State of Connecticut, USA, an ambient air limit of 1 pg I-TEQ/m<sup>3</sup> has been adopted for some time. (Rao and Brown, 1989). Both values have a degree of in built conservatism. A lifetime cancer risk estimate is between 1x10<sup>-2</sup> and 1x10<sup>-3</sup> for a 1pg lifetime exposure.

*Tier 1 assessment –*

Proposed CERC alone

2.108 For airborne exposure, the modelled maximum annual average ground level concentration of dioxins is 0.000087 pg/m<sup>3</sup>. If the stricter of the two available air quality standards (i.e. the Japanese standard of 0.6 pg/m<sup>3</sup>) is used, the safety margin is 6897. This is a very large safety margin and therefore a tier 2 assessment is not needed.

Plus background levels

As in most other urban locations in the UK there are no direct local measurements of dioxin in air since there are no standards to apply.

Life time risk

The estimated worst case cancer lifetime cancer is 1 in 15.5 billion (8.7x10<sup>-11</sup>). In practical terms this indicates no increased risk.

*Summary of the risks from inhalation of dioxins*

2.109 (i) size of the safety margin. The safety margin for annual exposure is very substantial

ii) consideration of background exposure

The main source of any dioxin exposure for members of the local community, as is the case for the public throughout the western world will be via their food.

iii) Analysis using life time risk data. The estimated worst case risk is very small and in public health terms insignificant.

It can be concluded that any emission of dioxins from the proposed CEC will not cause any health risk to members of the local community.

### **2.4.3 VOLATILE ORGANIC COMPOUNDS**

2.110 All methods of waste disposal contribute about 1% to the total VOC's emitted in the UK. Most of these come from landfills. Jay and Stieglitz (1995) conducted a detailed study of the types and quantities of VOCs in emissions from two German MSW incinerators, which had comparable emission limits to those in the UK. The authors identified and quantified more than 250 compounds above a detection limit of  $0.05 \text{ ug/m}^3$ , and these accounted for 42% of the mass of the total organic carbon (TOC) of  $525 \text{ ug/m}^3$ . Individual concentrations ranged between  $0.05$  to  $400 \text{ ug/m}^3$ . The remaining 58% of the TOC content of the gases consisted of aliphatic hydrocarbons. DEFRA review (2004) states that less than 0.02% of UK emissions of benzene are due to municipal solid waste operations.

2.111 VOCs describe a large group of organic compounds that are present in ambient and indoor air. They can appear as gases or in particulate form or a combination of the two. There are numerous sources of VOCs, for example, evaporation from paints, solvents and petrol. VOC's are also formed during combustion and microbial degradation of waste in composting and landfill. The most commonly occurring VOC's in the UK are butane and toluene. However, these VOC's have a low toxicity. VOC's can have three types of impact on man and the environment namely:

as precursors to the photochemical production of ozone in the troposphere

- direct toxicity.

- Odour

#### **2.4.3.1 Benzene**

2.112 In terms of the overall toxicity benzene is probably the most potent of the simple VOC's. Therefore it will be considered specifically. The more volatile VOC's, by their nature are unlikely to be found in food in any significant amounts. Thus this assessment concentrates only on the potential exposure of the local population by inhalation.

##### *Sources of exposure*

2.113 The annual mean concentrations of benzene in the UK range from 0.4-2.5ppb. Transport is the main source of benzene, accounting for 47% of UK emissions (DEFRA, 2004).

##### *Summary of effects*

2.114 Benzene can cause both acute and chronic effects:

- acute effects include vertigo, drowsiness, headache and nausea;
- chronic effects include anaemia and acute non-lymphoid leukaemia;
- benzene has also been found to cause chromosomal abnormalities in experimental systems and in man.

##### *Basis for the standard for benzene*

2.115 The UK has adopted a standard of 5  $\mu\text{g}/\text{m}^3$  as a running annual mean based on the risk of developing non-lymphoid leukaemia. However, a target has been set for Northern Ireland of 3.25 $\mu\text{g}/\text{m}^3$  to be achieved by 2010. Because benzene may be classified as a genotoxic carcinogen the UK government has determined that the levels should be reduced as low as is practicable and has therefore set a long term target value of 1ppb. The life time cancer risk estimate of 1 in 2.2 million for a lifetime exposure to 1 $\mu\text{g}/\text{m}^3$  of benzene.

### *Tier 1 assessment*

#### 2.116 Proposed CERC alone

The modelled worst case ambient ground level concentrations of benzene is 0.087  $\mu\text{g}/\text{m}^3$ . This assumes that the VOC's are emitted as benzene the most potent of the VOC's in terms of long term effects. Despite this worst case assessment, comparing this modelled value with the UK Objective Value gives a safety of factor of 575.

#### Life time risk

Using the worst case estimate of the ground level concentration gives an estimated increased cancer risk from the proposed CERC of 1 in 250 million ( $3.95 \times 10^{-9}$ ) for a lifetime exposure. This is an insignificant risk.

### *Summary of the risks from inhalation of benzene*

2.117 The risks may be summarised as follows:

- (i) size of the safety margin. The safety margin for annual exposure is very large.
- (ii) The lifetime risk estimate is much lower than the bench mark for acceptable risk of 1 in 14,286 ( $70 \times 10^{-6}$ ).

It can be concluded that emissions from VOC's such as benzene will not have any adverse effect on the health of the local population.

#### **2.4.3.2 POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)**

##### *Sources of exposure*

2.118 There have been several studies of either emissions of PAHs or their deposition due to incinerators. Colmsjo *et al*, (1986) found no difference in the concentrations of PAHs in the air at 900 and 2,500 metres from an older incinerator. Their conclusion was that the incinerator was not a significant contributor to ambient concentrations of PAHs. It should be noted that this

incinerator had much higher emission levels than is the case for modern waste to energy plants. Sadha and Wheatley (2007) measured the levels of various PAH's emitted from the stack of a clinical waste incinerator in the UK. The levels found were very low. No correlation was found between PAH's emissions and CO or other acid gases emissions.

#### *Summary of effects*

2.119 The effect of cancer is lung cancer (WHO 1987). A number of polycyclic aromatic hydrocarbons (PAH's) are mutagenic in a range of mutagenicity tests. Benzo(a) pyrene is among the most studied and the most potent of the PAH's. Many of the PAH's produce tumours in animals following skin painting or intratracheal instillation. In workers there is sufficient evidence of skin cancer in workers among tar workers. Coke oven and coal gas workers who in the past were exposed to very high airborne levels of PAH's have been shown to have a higher incidence of lung cancer

#### *Basis for the standards*

2.120 The EU has set a value of 1ng/m<sup>3</sup> to be achieved by 2012. The cancer risk value is 9x10<sup>-2</sup> for a 1ug/m<sup>3</sup> exposure for a lifetime.

#### *2.121 Tier 1 assessment*

##### Proposed CEC alone

The modelled value for polycyclic aromatic hydrocarbons is 0.00087.ng/m<sup>3</sup> for the annual ground level concentration. This gives a safety factor for the modelled annual ground level concentration of 1149. This is a worst case estimate which assumes that all the PAH's exist in the form of the very potent benzo(a)pyrene. It is evident therefore that a tier 2 assessment is not required. There are no local background values as currently there is no air quality standard in operation.

### Lifetime cancer risk

2.122 Based on the WHO lifetime risk factor the estimated worst case risk for lung cancer is 1 in 100 billion ( $1.07 \times 10^{-11}$ ).

### *2.123 Summary of the risks from inhalation of benzopyrene*

The risks can be summarised as follows:

- i) Size of the safety margin. The safety margin is very large and indicates no health concerns
  - ii) The lifetime risk estimate indicates a very negligible risk
- It can be concluded that emissions of PAH's such as benzo(a) pyrene will not constitute a health risk to members of the local population.

## **2.5 CONSIDERATION OF THE RISK FROM INGESTION OF THE RELEVANT CHEMICALS OF INTEREST**

2.124 Food contamination is a potential additional concern for chemicals of interest that are persistent in the environment namely metals and dioxin like chemicals. This issue has been raised in the statement of case of the Cornwall Sustainable Waste Network. It is therefore appropriate to consider further the modelled estimates of the possible contamination of locally foods for these two categories of chemical. Animals could ingest dioxins and/or metals through either consumption of plants or other contaminated food or through ingestion of contaminated soil. Most metals are not well taken up or concentrated by plants. In the air quality section of the ES the levels of dioxins and metals in food have been calculated using conservative assumptions on the extent of contamination. Modelling for a number of potential hypothetical individuals ( often termed for modelling purposes receptors) has been carried out. It is assumed that the individuals receiving the highest intake of metals and dioxins are likely to be local farmers and their families because they are likely to consume more locally/on-farm produced food than other local residents. Local residents growing their own

food, in an area where ground levels of these chemicals of interest are estimated to be the highest, should also be considered.

The following are estimated to receive the higher intakes:

- Local farmer
- Child of a farmer
- Resident adult (South and East)
- Resident child (South and East)
- Resident adult (Trevisco)
- Resident child (Trevisco)

2.125 In the following analysis the worst case value from these receptors has been set for the tier 1 risk assessment. These values can be compared with either the standards set by WHO (JECFA) or the USA ingestion reference doses (this is a standard for acceptable levels of contaminants in food):

*Table. 2.4 Food safety standards for metals and dioxins*

Chemical of interest	JECFA standard. ug/kg body weight/day	USA Ingestion reference dose ug/kg/day
Arsenic	2	0.03
Lead	3.6	0.043
Mercury	1.5.	0.03
Cadmium	1.0	0.04
Dioxins	0.001	.001

2.126 Since the US reference dose values are in general much stricter these will be used in the subsequent tier 1 estimations of the risk although it should be noted that in the UK and elsewhere in the EU it is the JECFA values that apply in practice. The USA values can be used to estimate the carcinogenic risk and non

carcinogenic risks for the most sensitive receptor site using conservative emission, deposition and ingestion estimates. The estimated safety factors (non-carcinogenic risk) are calculated by comparing the standard against the actual exposure. The same approach that was used in previous sections of this Proof.

2.127 In each case it must be borne in mind that the estimates involve very conservative assumptions both regarding the levels of food contamination and the amount of locally grown food consumed. For even the hypothetical individual estimated to consume the highest intake of metals and dioxins in their food, the safety factor for each chemical of interest is very substantial. Bearing in mind the number of worst case assumptions used in the estimation, it is clear that there will be no changes in the overall health of the local population due to the consumption of locally grown food.

*Table. 2.5 Calculated safety factors for the total intake from food consumption of relevant chemicals of interest based on conservative estimates*

Chemical of interest	Ingestion reference dose mg/kg/d	Worst case receptor mg/kg/d	Safety Margin
Arsenic	3.0 x10 <sup>-4</sup>	4.7x10 <sup>-7</sup>	638
Cadmium	4.0x10 <sup>-4</sup>	1.6x10 <sup>-7</sup>	2500
Chromium 6+	3.0x10 <sup>-3</sup>	2.5x10 <sup>-6</sup>	1200
Mercuric chloride	3x10 <sup>-4</sup>	5.0x10 <sup>-7</sup>	600
Lead	4.3x10 <sup>-4</sup>	6.4x10 <sup>-7</sup>	672
Dioxin	1x10 <sup>-9</sup>	4.7x10 <sup>-14</sup>	2128

The worst case total cancer risk, taking all the chemicals of interest with

carcinogenic properties together, is 1 in 15.6 million ( $6.4 \times 10^{-8}$ ). This is clearly a worst case not only in modelling terms but also because it is unlikely that arsenic, cadmium, lead or dioxin are genotoxic carcinogens and consequently a linear extrapolation of data (which is used to estimate the cancer risk from low level exposures) is not realistic. Using the commonly used bench mark of acceptable risk of 1 in 14,286 ( $70 \times 10^{-7}$ ) it is evident that the risk worst case cancer risk is more than sufficient. It can be concluded that even for the receptor that is estimated to be exposed to the highest intake of metals and dioxins in their food, the safety factor for each chemical of interest is very substantial. Thus it is evident that the proposed CERC will not affect the quality of locally produced food. Bearing in mind the number of worst case assumptions used in the estimation, it is clear that there will be no changes in the cancer risk of the local population due to the consumption of locally grown food.

## **2.6 CONSIDERATION OF THE RISKS FROM THE COMBINATION OF INHALATION AND INGESTION.**

In addition to consideration of the risks from separate routes of exposure it is valid to also consider the impacts of the exposure by all routes.

*Table 2.6. Safety factors for inhalation and ingestion*

<b>Chemical of interest</b>	<b>Safety factor Ingestion</b>	<b>Safety factor Inhalation</b>
Arsenic	638	125
Cadmium	2500	227
Chromium 6+	1200	2083
Mercuric chloride	600	5682
Nickel	672	5205

Dioxin	2128	6897
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2.129 It can be seen from the above table that the safety factors for both inhalation and ingestion are very substantially for each of the chemicals of interest. Bearing in mind that these calculations incorporates a number of worst case assumptions, it is evident that there will be no increase in the health risk to the local population from the emissions from the proposed CERC due to inhalation or extensive consumption of locally grown food.

## 2.7 CONCLUSIONS

2.130 The risks to health from the inhalation of each of the chemicals of interest has been estimated in the following ways:

- Based on comparison of the relevant standard with conservative/ worst case estimates of the maximum ground level concentrations of each chemical of interest. Estimation of the risk (which is inversely related to the size of the safety factors) in each case shows that the health of the local population will not be adversely affected by the operation of the proposed plant (see summary table below).

*Table 2.7. Summary of calculated safety margins for each chemical of interest*

Chemical	Inhalation- Short term safety margin (bench mark of acceptability 10 or more)	Inhalation- Long term safety margin (bench mark of acceptability 100 or more)	Ingestion of food- Long term safety margin (bench mark of acceptability 100 or more)

Hydrogen fluoride	3200	NS	NS
Hydrogen chloride	1500	2299	NS
Nitrogen dioxide	30.3	308	NS
Sulphur dioxide	233	2941	NS
Carbon monoxide	6250	NS	NS
Particulates (PM10)	1429	4598	NS
Arsenic	5357	125	638
Cadmium	1250	227	2500
Chromium VI	1071	2083	1200
Lead	NS	5208	672
Mercury	3000	5682	600
Nickel	1071	417	-
Thallium	25000	45455	-
Dioxin	NS	6897	2128
Benzene	NS	575	NS
Benzo(a)pyrene	NS	1149	NS

NS = no standard has been set either because the effects are almost exclusively short term (eg hydrogen fluoride) or long term eg lead.

- Consideration of the influence of the operation of the proposed plant on the local air quality. Again the conservative/ worst case estimates show that there will be no adverse impact on compliance with any of the air quality standards.

- Estimation of changes in life time cancer risks. The calculations show that the impact of the proposed plant will be insignificant.
- Assessment of the conservative/worst case contribution of emissions from the operation of the proposed plant on local food contamination. The findings demonstrate that the proposed CERC will not adversely affect the quality of locally produced food.

2.131 It is also clear that the combination of inhalation of the chemicals of interest with ingestion of possibly contaminated food does not significantly increase the risk identified above.

## **CHAPTER 3 RISK ASSESSMENT OF COMBINATION OF CHEMICALS**

3.1 A comprehensive assessment of the source to receptor pathways for combination of chemicals is very important. In this chapter the risk factors are presented in the form of hazard quotient. In assessing hazard quotients value close to or approaching 1 indicate the need for further assessment or the introduction of additional air emission control methods. The lower the value the lower is the risk.

### **3.1 ASSESSMENT OF THE COMBINATION OF CHEMICALS WHICH WILL BE EMITTED FROM THE PROPOSED CERC**

3.2 The study of chemical mixtures has started in the last century and involves interdisciplinary collaboration, relying heavily on computational technology (Suk *et al*, 2002). However, the literature on the effects of mixtures/cocktails of chemicals is still limited. One of the reasons for this is that so many factors to consider at different levels of exposure. The effect of the mixture's interaction can be independence, additivity, antagonism or synergism. Agent-to agent interaction prior to crossing an organism's boundaries or once the mixtures have crossed the boundaries constitute general types of interactions among mixture's components. There is no clear evidence of synergism between pollutants at the concentrations studied.

3.3 Since there is no good scientific data on inhibition or synergism in the toxic action of mixtures, the approach has been adopted for this Proof of Evidence that the toxicity of some chemicals may be additive. Additive effects could occur when the individual components of a mixture act on the same organs or cell types or receptors. (NB This approach is used to determine the TEQ for dioxins).

The most likely situations for additive effects to occur are:

- Acute mucous membrane irritation (throat, lungs, nose etc ) due to the combined effects of irritant chemicals (ie SO<sub>2</sub>, NO<sub>2</sub>, PM<sub>10</sub>, HCl and HF)

- Carcinogenic action on the lung ( nickel, arsenic, benzo(a)pyrene, chromium,).

#### *Acute irritants*

3.4 In the case of the acute irritants the approach used for assessment is to calculate a hazard index based on that used by the UK HSE (Health and Safety Executive) to assess the impact of combined exposure to chemicals in the work place with the help of the equation:  $C1/L1 + C2/L2 + C3/L3 + \dots Cn/Ln = X$  (where: C1, C2, ... Cn = are the airborne concentrations of each chemical; L1, L2, ... Ln = the Occupational Exposure Levels (OELs); and X = the Hazard Index). (The intervening, resulting ratios (C1/L1, C2/L2, etc) are called Hazard Quotients).

3.5 If the Hazard Index is less than 1, the mixture is considered not to represent a health hazard; if the Hazard Index is greater than 1, steps should be taken to reduce the concentrations of one or more of the chemicals involved. If no synergistic or additive effect is known or considered likely, the HSE consider that it is sufficient to ensure that there is compliance with each OEL individually. Using this approach the index is 0.039 that is it is substantially below a value of 1. It can be concluded that an additive effects resulting is irritation of the mucous membranes is very unlikely.

#### *Lung carcinogens.*

3.6 The approach for carcinogenic chemicals is to add the calculated life time risks for each chemical together to calculate an overall lifetime risk for the most exposed receptor. This gives a life time risk value o  $1 \times 10^{-7}$ . Well below the value of  $1 \times 10^{-6}$  which is a common bench mark of acceptable risk.

### **3.2 CONCLUSIONS**

3.7 The two possible scenarios for a combined effect of the chemicals of interest

are: an acute effect due to enhanced irritancy of mucous membranes ( eye, nose throat, lung) and or a long term effect in particular lung cancer. Estimation based on the ground levels that could arise from the proposed CERC indicate that a cocktail effect is most unlikely.

## **CHAPTER 4 OTHER ASSESSMENTS OF THE POSSIBLE HEALTH IMPACTS FROM MODERN INCINERATORS**

4.1 There several sources of data on the impacts of modern incinerators on the environment and on human health namely:

- i) findings from the published literature
- ii) conclusions of government agencies and independent scientific committees
- iii) conclusions of planning inspectors of inquiries into proposals for specific incinerators

### **4.1 FINDINGS FROM THE PUBLISHED LITERATURE**

#### **4.1.1 Dioxins**

4.2 An evaluation of the likely impacts arising from emissions from the operation of an incinerators/waste to energy plant can be made by considering the available publications on:

- Measurements of air, soil and plant levels of certain chemicals of interest (exposure investigations)
- Measurements of certain chemicals of interest in human blood and breast milk (Biological monitoring)

4.3 The focus of research in the last two decades has been on measurements of dioxins and metals as these are most likely to persist in the environments.

Dioxins levels in ambient air are very low, in part a reflection of their very low volatility. Maximum quarterly concentrations of dioxins and furans in major cities in the UK range from 33.4-169.2 fgTEQ/m<sup>3</sup>. In parallel the emissions from modern waste to energy plants in the EU countries are a minimum of 2 -3 orders of magnitude lower than was the case in the 1950s and 1960's.

4.4 An investigation of dioxin levels in soil and vegetation around a MSW waste-to-energy plant in Spain by Domingo *et al* (2001) that emitted somewhat higher levels of dioxins than is currently permitted, resulted in the conclusion "in comparison with other emission sources of PCDD/Fs in the same area" (traffic, other industrial activities, bonfires) "the current PCDD/F emissions from the MSW incinerator would be of small significance for the population living in the neighbourhood of the MSW incinerator".

4.5 Caserini *et al* (2004) examines air and soil levels of dioxins around three MSW incinerators in Italy. At all three sites dioxin concentrations in soil were at the lower limit of the average values for rural areas. Mari *et al* (2007) carried out a temporal assessment of environmental contamination around a modern hazardous waste incinerator. The author's conclusion was that the incinerator did not significantly increase dioxin levels in soils around the plant.

4.6 Marti-Cid *et al* (2008) measured PCDD/PCDFs in foodstuffs in Tarragona (Spain) near a hazardous waste incinerator (in operation since 1998). The authors concluded that the levels of dioxin were higher prior to the installation of the incinerator and concluded that 'the notable decrease in the atmospheric levels of PCDD/PCDFs over the world would explain notable differences between the results in the dietary intake in the base line, 2002 and current surveys'.

4.7 Both for humans and animals the intake of dioxins (and dioxin-like materials) is influenced by the nature of their diet, regardless of age. Diet high in fat (particularly oily fish) will lead to relatively high intake of dioxins. In an investigation of the blood levels of dioxins in a local population Spanish authors compared measurements made in individuals before and for two years after a new MSW waste to energy plant became operational (Gonzales *et al* 2000, a). Two population groups were selected: one living within 1.5km of the plant and

the other 3.5-4km away. There was a control group, which lived in an area without an incinerator. All three populations showed increased blood levels of dioxins over the two-year period regardless of the distance from the incinerator.

4.8 Measurements of the dioxin levels of populations living within 1 km of a MSW waste -to-energy plant was conducted in Japan by Yoshida *et al* (2000). The findings were compared with the assessed dioxin levels of the general population in the country. The results showed a trend to lower levels of dioxins in the blood lipids and milk lipids in the residents around the MSW waste- to -energy plant than the average for the general population. Levels of dioxins were very variable within each population. This meant that different routes of dioxin exposure were likely. The authors concluded that living close to a modern waste- to-energy plant does not result in increased body levels of dioxins.

4.9 The same trend was observed in the vicinity of a modern hazardous waste incinerator (Evans *et al* ,2000). In the test group the blood levels of dioxin actually decreased (the samples taken from the pre-incineration period were compared with four months of incineration of the contaminated material). The control group was 15 or more kilometres away from an incinerator. A similar reduction but a smaller one was also found in the control population. Two studies conducted in Portugal found no increase in either blood levels or in breast milk in the local population in the vicinity of modern incinerators compared to a control population (Reis *et al* 2007a, 2007b).

4.10 Measurements of dioxin in air around waste incinerators that are performing to current EU emission standards indicate ambient air levels that are indistinguishable from those in other urban locations. There is little or no indication of increased blood or other breast milk levels of dioxins. Numerous studies also show that background dioxin levels have been falling in food over

the past decade. Kulkarni *et al* (2008) stated 'over the past several years there has been a shift in the major sources of dioxins in large part due to regulations and focused voluntary efforts.' The published literature in relation to dioxins and modern incinerators may be summarized as follows:

- No detectable increases in blood levels, breast milk levels or umbilical cord blood samples;
- No detectable increases in contamination of plants or animals;
- No measurable increases in soil or air levels.

#### **4. 1.2 PM 10 and ultra-fine particles**

4.11 Sources of PM10 range from the burning of candles to motor vehicles and power station emissions. Not surprisingly the composition is heterogeneous. Thus particles may incorporate metals and other inorganic substances such as salt, carbon, hydrocarbons, dioxins and/or polycyclic hydrocarbons. In terms of ambient air, the amount of ultra-fine particles is rather similar in urban and rural areas, with as much as  $10^6$  to  $10^8$  nanoparticles per litre of air depending on conditions.

##### *Environmental monitoring*

4.12 At present it is not technically possible to distinguish quantitatively between fine particles emitted from different sources except by measuring them at the point of emission. It is clear that the highest particle number concentrations and smallest particle size are associated with road traffic (See appendix 9).

4.13 Buonnano *et al* (2009) compared the emissions of particulate matter from the top of the stack of a modern incinerator with those from motor vehicle emissions. Their calculation is that the stack emissions for one hour are equivalent to 20 vehicles (based on a typical traffic mix) travelling at normal speed on a motorway for 2 miles.

4.14 A comparison of relative ground level exposure to particulate matter can be made based on measurements of particulates around an incinerator with those found indoors. Typically the calculated values for ground level concentrations from a modern waste to energy plant are of the order of 0.02-0.2ug/m<sup>3</sup>. These values can be compared with those arising from activities in the home or office, which range from >20 to >100 ug/m<sup>3</sup>.

#### **4.1.3 Metals**

4.15 Contamination of plants by various metals can occur simply by deposition or due to a combination of deposition and uptake. The predominant route of metal exposure in humans is the diet. Airborne exposure to metals is, generally, low. Ferre-Huguet et al (2007) studied the levels of a range of metals in both soil and vegetation around a new hazardous waste incinerator in Spain. They concluded that the operation of the incinerator did not result in a significant source of metal pollution in the area.

4.16 Rimmer *et al* (2006) studied the levels of metals and arsenic around the Byker MSW incinerator in Newcastle upon Tyne. Soil levels of metals, including arsenic were measured at distances up to 2.5km from the incinerator stack. It was concluded that the levels of metals were typical of those of an urban environment but above those of a designated rural community.

4.17 Several investigations have examined the possible contribution of incinerator emissions on the level of metals in blood. Gonzales et al (2000) examined the impact of a new MSW waste to energy plant on blood metal concentrations of the local population in Spain. They measured four metals - lead, cadmium, chromium and mercury, which in their opinion, could result in identifiable changes in blood levels compared with a non-exposed population. A

small reduction in lead levels of the exposed population was observed. This finding probably reflected the increased use of unleaded petrol in cars in Spain. However, there were no differences in the blood levels of chromium, cadmium or mercury between exposed and non-exposed populations. The conclusion was that the waste to energy plant did not have a significant impact on metal exposure/uptake of the local population.

4.18 A study (by Agramunt *et al*, 2003) on workers at a modern waste to energy plant, confirmed that there was no increase for any of the metals that were analysed for with the exception of vanadium. Indeed the blood levels of many metals were either below the base line or not detectable. In respect of vanadium the source was unclear but it was noted that the same increase was found in the control worker population (laboratory and administrative staff). Reis *et al* (2007c, d, and e) carried out a very detailed investigation of human exposure to metals around two modern solid waste to energy plants in Portugal. They examined levels of lead, cadmium and mercury in the blood of the general population around the plants and also investigated umbilical cord and maternal blood for lead. The data on the population living in the area around the plant was found not to differ significantly from that of the control population.

The published literature in relation to metals and modern incinerators may be summarized as follows:

- No detectable increases in blood levels or umbilical cord blood samples;
- No detectable increases in contamination of plants or animals;
- No measurable increases in soil levels.

## **4.2 EPIDEMIOLOGICAL INVESTIGATION OF HEALTH EFFECTS**

4.19 Epidemiological investigations have looked of various diseases in populations living around incinerators compared with the incidence of the same diseases in 'control populations' the incidence :

- effects on the lung
- cancers
- reproductive effects

An important constraint in reviewing the data is that the key studies are all retrospective and therefore are focussed on incinerators that are poorly performing by today's standards. The findings from the literature are briefly summarised here (For further details see appendix 4).

Roberts and Chen (2006) assessed the potential health impacts of a waste to energy plant, designed to burn 52,500 tons of refuse derived fuel (RDF) annually (assuming current EU regulations on emissions are in force). Making the worst case scenario that the plant could emit the maximum permitted levels of every chemical of interest throughout the normal operating period, they calculated the impact on the local population of 25,000. They estimated that if the plant operated for 25 years it might contribute to a cancer increase of 0.018 per million of population. In addition, 0.46 deaths per million of the population might be brought forward due to sulphur dioxide and 0.02 deaths per million brought forward due to particles. The overall risk of dying as a consequence of the plants operation calculated to be  $2.49 \times 10^{-7}$ . The overall conclusion is that the impact of the proposed plant on the health of the local community would be negligible.

#### **4.2.1 Respiratory/irritant effects and incinerators**

4.20 Hu *et al*, (2001) investigated chronic health effects in communities living near to three separate MSW incinerators in the USA (which performed to levels that are substandard to the current EU ones between 1992 and 1994).

Participants in the study were assessed each year by a spirometric test. The results were not statistically significant between lung function and proximity of residence to any of the three incinerators.

4.21 Gray *et al* (1994) compared the prevalence of asthma , in children living around a sludge burning incinerator and in a control area. No significant differences were found. Miyake *et al* (2005) examined the possible contribution to respiratory symptoms (and some other effects) in young Japanese school children whose schools were near incinerators. The authors conclude that the presence of a school close to the incinerators causes a small increase in the prevalence of one or more of the symptoms. However the design of this study makes the interpretation of the findings very difficult.

#### **4.2.2 Effects on reproduction and the developing foetus**

4.22 The main contributor to foetal abnormalities appears to be genetic . The overall level of congenital abnormalities in the UK is generally rather constant from year to year. Dolk and Vrijheid (2003) reviewed the epidemiological studies for correlations between congenital abnormalities and exposure to chemicals associated with environmental pollution and considered a number of possible causes and contributory factors. The authors concluded that there are relatively few environmental pollution sources for which strong conclusions can be drawn regarding their potential to cause congenital abnormalities. A Japanese study of adverse reproductive outcomes (in relation to proximity to 63 municipal solid waste incinerators) did not find any statistically significant outcomes within the distance of 2 to 10 km from the incinerators (Tango *et al*, 2004). However, it showed a peak-decline in risk with distance for infant deaths with all congenital malformations combined. Cresswell *et al*, (2003) conducted a study in a population around the Byker (Newcastle- upon-Tyne) waste combustion plant. No significant overall association between the number of congenital abnormalities and geographical proximity to the plant was found in the study.

#### **4.2.3Cancers**

*(i) non-Hodgkins lymphomas*

4.23 Zambon *et al* (2007) investigated the incidence of sarcomas in a case control study involving individuals living in an area which had some 22 incinerators of various kinds as well as a number of other industrial plants. The data was collected in the 1990's and relates to old incinerators. They found an increased incidence of sarcomas which they attributed to dioxins. No direct evidence was put forward to link the sarcomas either to the incinerators nor to dioxins.

4.24 A Finnish research project which studied the association between soft-tissue sarcoma and dioxin identified that the highest risk of sarcoma was found at low levels of dioxin concentration (Tuomisto *et al*, 2004).

No increased risk associated with increased dioxin concentration was found.

*(ii) Adult cancers particularly liver cancer*

4.25 One of the best conducted studies on possible adult cancer risks was that of Elliott *et al* ,1996. They used postcode data to investigate the cancer incidence among 14 million people living near any of the 72 MSW incinerators in the UK. A statistically significant trend for a decline in risk was observed with increasing distance from the incinerators for all cancers combined (and for stomach, liver and lung cancers specifically). When allowance was made for socio-economic deprivation scores in each location no adverse effects could be identified.

*(iii) Child cancers including leukaemias*

4.26 Knox (2000) studied possible health risks to children from both landfill and incineration emissions the study focused exclusively on child deaths from cancer (both solid tumours and leukaemia's). Knox's view was that with proximity (7.5- km) to very old MSW waste incinerators and old hospital incinerators there was a small increased relative risk for children to develop cancer. If these very old incinerators are omitted, there is no identifiable increased cancer risk. Knox

acknowledged that this 'seemed to exonerate the more modern plants' (built in the 1960's and 1970's). In another review the authors stated that there is no clear relationship between childhood cancer and incinerator emissions, even if some results were statistically significant (Franchini *et al*, 2004).

4.27 The published literature in relation to health effects is retrospective and do not include modern incinerators. The findings for older incinerators with substantially higher emissions may be summarized as follows:

- No consistent increase in the incidence of respiratory effects
- No consistent increase in the incidence of reproductive effects or effects on the developing foetus
- No detectable increase in childhood cancers
- A possible small increase in sarcomas

No identifiable increase in other cancers.

### **4.3 VIEWS OF EXPERT BODIES**

4.28 As wastes degrade within a short- period of time, a range of compounds will be emitted to air. This may be a particular problem when the waste is from domestic sources because the condition and composition of the waste on arrival at a treatment plant can be very variable.

4.29 In common with other EU governments, The UK commissioned reports and various research projects in order to understand the risks associated with municipal solid waste incinerators (MSW) and modern waste to energy plants. In 1997 in one of such reports the Institute of Environmental Health (IEH) stated 'no consistent pattern of ill-health has emerged from studies of incinerator workers or populations living near incinerators. Any future epidemiological studies investigating the health effects of living near incinerators should be designed so that any small increases in risk can be detected and should

adequately account for the various confounding and modifying factors.'

4.30 The UK Committee on Carcinogenicity (2002) considered the cancer risk from living near a municipal solid waste incinerator. Its conclusion was 'any risk of cancer due to residency near to MSWIs is exceedingly low and probably not measurable by the most modern epidemiology techniques'. The Committee concluded that there was no need for 'further epidemiological investigations of cancer incidence near MSWIs'

A report by the Institute for European Environmental Policy (2001) came to a similar conclusion.

4.31 The DEFRA Report (2004) reviewed the UK data on the health and environmental effects of waste management. The report pointed out that the data should be viewed in the light of the benefits of collection and disposal of the wastes. If waste were not collected treated and disposed of, it would become a source of disease, odours, litters and pests. Nonetheless, the DEFRA Report recommended that a number of research studies were conducted in order to improve understanding of health implications of waste disposal.

4.32 This report also attempted to quantify the total impact of waste disposal (by all methods) on the entire UK population. It concludes 'on a national scale, taking into account the amount of waste managed by each process at present, emissions to air from waste management are estimated to result in approximately five hospital admissions for respiratory disease per year and one death brought forward due to air emissions per year in the UK as a whole. Emissions to air are forecast to result in a much lower increase in the incidence of cancers: only about one additional case every 500 years'.

4.33 The overall conclusion of DEFRA report is that 'while the information is

incomplete and not ideal, the weight of evidence from the studies so far is that present-day practice for managing municipal solid waste has at most a minor effect on human health and the environment'. The review 'did not find a link between the current generation of municipal solid waste incinerators and health effects'. It stated that 'the current generation of waste incinerators result in much lower levels of exposure to pollutants and no evidence for a link between the current generation of incinerators and the incidence of cancer, respiratory diseases and birth defects was found'. Finally, the report compares the risk from waste management against other risks such as traffic and firework display accidents. The report found that emissions from municipal solid waste are much less significant than these other hazards.

4.34 DEFRA published 'The Waste Strategy for England' in 2007. This report stated that 'concern over health effects is most frequently cited in connection with incinerators. Research carried out to date shows no credible evidence of adverse health outcomes from those living near incinerators. The relevant health effects- primary cancers- have long incubation times, but the available research demonstrates an absence of symptoms relating to exposures twenty or more years ago, when emissions were much greater than they are now'.

4.35 For comparison, in the USA in 2007 12.6% of municipal solid waste was combusted with energy recovery. The total production of waste (before recycling) was estimated to reach 254 million tonnes (EPA, 2008).

The US National Academy of Sciences Committee (2003) investigated the relationship between incineration and health and concluded: 'the studies of which the committee is aware, that did report finding health effects had shortcomings, failed to provide convincing evidence. That is not surprising given the small population typically available for study and the fact that such effects, if any, might occur only infrequently or take many years to appear. Although emissions

from newer, well run facilities are expected to contribute little to environmental concentrations and to health risks, the same might not be true of some older or poorly run facilities'

4.36 The US EPA (2004) issued the Hazardous Waste Minimization and Combustion Strategy, which recommended a site specific risk assessment for each combustion facility seeking a permit to ensure that such a facility operates in a manner protective of human health and the environment. US EPA has set national air quality standards for six principal pollutants: nitrogen dioxide, ozone, sulphur dioxide particulate matter, carbon monoxide and lead. A generation ago the US Congress passed the Resource Conservation and Recovery Act (RCRA) and its goal was to 'recover energy and other resources from discarded materials' Since then a much more ambitious plan has been developed. New Resource Conservation Challenge Five Year Strategic Plan moves to a more integrated planning of chemical reduction and use.

4.37 The UK Health Protection Agency in its review (2009) stated that 'while it is not possible to rule out adverse health effects from modern, well regulated municipal waste incinerators with complete certainty, any potential damage to health of those living close-by is likely to be very small, if detectable''.

4.38 In particular, the Health Protection Agency concentrated on particulate matter in relation to incineration processes. By using meta-analytical techniques (COMEAP,1998, 2009 and WHO, 2006) the Agency concluded that the estimated effects on health of the small increases in concentration of particles produced by incinerators are likely to be small and so is the size of the potentially exposed local population.

4.39 The coefficients used by the Agency are derived from epidemiological

studies, which link effects on health with changes in mass concentrations of particulate matter, both as PM<sub>10</sub> and PM<sub>2.5</sub>

Thus, the Agency does not distinguish between the toxicity of samples of particles collected for PM<sub>10</sub> and PM<sub>2.5</sub>

4.40 The Agency believes that the contribution made by waste incineration to national emissions of particulate matter is low. In 2006 national emissions of PM<sub>10</sub> from waste incineration were 0.03% of the total compared with 27% and 25% for traffic and industry respectively. In addition, the Environment Agency calculated in an incinerator modelling study that a modelled ground level increment in PM<sub>10</sub> will be 0.0005 µg/m<sup>3</sup> as an annual average (EA, 2009). On the basis of this evidence the Agency concluded that 'it is highly unlikely that there will be a detectable effect of any particular incinerator on local infant mortality'.

4.41 DEFRA (2004) calculated that incineration of municipal solid waste accounts for less than 1% of UK emissions of dioxins, thus, making the contribution of incinerator emissions to exposure by inhalation 'a negligible component of the average human intake. Committee on the Carcinogenicity of Chemicals in Food, Consumer Products and the Environment (2009) reviewed seven studies on cancer incidence, which were published since 2000 and concluded that 'although the studies indicate some evidence of positive association between two of the less common cancers, i.e. non-Hodgkin's lymphoma and soft tissue sarcoma and residence near to incinerators in the past, the results cannot be extrapolated to current incinerators, which emit lower amounts of pollutants'

4.42 The overall conclusion of the Health Protection Agency (2009) state that modern, well managed incinerators make only a small contribution to local concentrations of air pollutants. It is possible that such small additions could have

an impact on health, but they are likely to be very small and not detectable.

#### **4.4 UK PUBLIC INQUIRIES**

4.43 Possible health issues in relation to proposed incineration/waste to energy plants have been considered in great detail at a number of Public Inquiries over the past few years. The Inspectors Reports have consistently concluded that the health risk is not significant. For example, at the Portsmouth incinerator Inquiry (2002) the Inspector 'considered the issue of health risks from the emissions from the proposed plant in great detail' and his conclusion was that 'I consider there would be very little risk to the health of the surrounding population'. A similar conclusion was reached at the Riverside Inquiry (2004). The conclusions of the Inspectors and HMG in regard to health aspects for incinerators appeals that have been turned down in recent years are given below. Two such cases are Hull and Kidderminster.

4.44 Town and Country Planning Act 1990 (section 78). Appeal by Waste Recycling Group plc. Land off Foster Street, Stoneferry Park, Kingston upon Hull application number 00026438. The inspector (Smith 2004) accepts that the atmospheric dispersion modelling system (ADMS) is conservative and usually produces results towards the 'worst case' approach and in practice, the levels of air pollutants are much lower than forecast levels. He concludes that 'the results of the prediction revealed that air quality in the locality would comply with all health based air quality standards and guidelines with or without the appeal project'. The inspector notes that although there was a perception that the impact of the incinerator will be substantial in making the air quality in Hull much worse, 'this perception did not appear to be grounded in objective evidence' He states that 'for dioxin and furans the predicted exposure levels would be less than 1% of the UK Government's recommended standard and only 6% of the levels prescribed in the EC Waste Incineration Directive'.

4.45 The Inspector also remarked on the general acceptance of all parties involved of 'a specified limits approach to pollution together with desire for effective regulation by competent authorities'. Even in a situation of plant failure short-term emissions levels would still be within relevant air quality standards and guidelines. Taking overall impact of the plant, the Inspector stated that 'it would be unlikely that there would be any increase in deaths and only a small number of hospital admissions for respiratory illness attributable to exposure from PM10 emitted by the proposed incinerator'. He came to similar conclusions in relations to sulphur dioxide, hydrogen chloride, hydrogen fluoride, carbon monoxide, volatile organic hydrocarbons, polycyclic aromatic hydrocarbons and heavy metals. The Inspector believed that the relationship between 'the presence of incinerator and poor health statistics is of mere inference or coincidence as opposed to statistical correlation'. In conclusion, the Inspector had no objections to the proposal in terms of air quality, health effects and risk. In response to the Inspector's report, the First Secretary of State (2004) agreed with the Inspector's conclusion on health effects and that the proposed plant would not conflict with the pollution control objectives. He also accepted the Inspector's conclusion that the levels of risk were 'tolerable' and would be properly controlled.

4.46 Town and County Planning Act 1990. Appeal by Mercia Waste Management Ltd. Site at British Sugar Site, Stourport Road, Kidderminster

In the Inspector's report, (Smith, 2002) it was concluded that the emissions of the pollutants of most concern (nitrogen dioxide, particulates, sulphur dioxide, ozone, metals and dioxins) would be extremely small. He stated that 'dioxin emissions have reduced 100 fold from UK incinerators in 5 years and they now produce only 0.52% of UK dioxin emissions, about 1/50<sup>th</sup> of those from the iron and steel industry and 6% of the dioxins from one bonfire night'. He accepted the DETR Panel on Air Quality Standards decision not to have a new PM2.5 standard

for fine particulates. He also stated that there is no evidence that the extremely small contribution to dioxin intake arising from the proposal would take the body burden above 2 pg/kg/day the level recommended in the UK by the Committee on Toxicity (COT). The Inspector believed that epidemiological studies do not reveal any clear indication of the health effects on nearby population from modern incinerators. The Inspector 'placed little weight on the fact that the appeal site was close to a residential area of acknowledged social deprivation'.

Expert bodies are in general agreement that modern incinerators are unlikely to cause significant adverse effects on the health of the surrounding local communities. Nonetheless further targeted studies could be helpful.

#### **4.5 RISK PERCEPTION**

Perception often has a strong temporal relationship. The perception of risk may, for some individuals, differ considerably from the actual risk (see appendix 9).

The reasons for this are complex and include:

- \* family history, personality and past experiences
- \* the influence of the media, action groups and individuals identified as peers
- \* information access and understanding regarding the proposed facility

Risk. An important aim of this proof is to provide the relevant information.

#### **4.6 CONCLUSIONS**

4.47 The findings from published literature relating to more modern incinerators/waste to energy plants are in line with the findings of the modelling data for the proposed CERC, namely that significant environmental contamination is very unlikely. The literature also indicates that for more modern incinerators those living in the local surrounding area should not experience any adverse health effects. This analysis of the available information is in agreement with the views of many expert committees that have examined the relationship between incineration and health. A similar conclusion has also been reached independently

in many recent planning Inquiries

## **CHAPTER 5 OVERALL CONCLUSIONS**

5.1 Appropriate means of waste management are a vital part of a well organized and healthy society. Incineration (waste to energy) is one of several processes that is considered acceptable in most countries and it is one key component of the current EU waste management strategy (EU 2008). The proposed CERC is likely to play a valuable role in the utilization of MSW for energy production.

5.2 However, all societal activities involve some element of risk and it is appropriate that any proposed new industrial process such as a waste management process is assessed for its possible impacts on human health and on the environment. In respect of the proposed CERC the critical factors in determining whether or not adverse effects could arise is the nature and the levels of chemicals to which members of the local population could be exposed. Two approaches have been used to assess the possible impacts on human health as a consequence of the operation of the proposed plant;

- Use of air quality modeling data for the plant based on a worst case assumption of maximum permitted emission limits.
- examination of the published literature

### **Evaluation of the modeling data provided.**

5.3 The dispersion of the airborne emission of each chemical of interest has been modeled in the air quality section of the ES. This modeling approach errs strongly on the side of caution. The conservative estimated ground level concentrations of each chemical/chemical class have been compared against the appropriate health based standards/guidelines. In each case a substantial margin of safety is evident. In other words the levels emitted from the proposed plant, even making a number of 'worst case' assumptions, are much lower than the standard.

5.4 The possible impact on members of the local population has also been

examined from a combination of airborne exposure and consumption of locally grown food that might have been contaminated as a result of the operation of the proposed CERC. This has focused on potentially persistent chemicals namely dioxins and metals. Both non-carcinogenic and carcinogenic effects have been considered. In respect of non-carcinogenic effects it can be concluded that the safety factors are substantial and that bearing in mind the conservative nature of the methodology used there will be no significant risk to health. The carcinogenic risk in each case is also substantially lower than recognized bench marks of acceptability. Finally the potential for a so called 'cocktail' effect (i.e. an effect due to combinations of chemicals) has also been assessed. The findings for combinations of chemicals do not change the conclusion that the Plant is most unlikely to cause adverse effects to the health of the local population.

**An extensive review has been conducted of the published scientific literature.**

5.5 Since there are almost no data on the potential health and environmental impacts of modern waste to energy plants because it is considered very unlikely that adverse effects will be found, the analysis of literature has been focussed on old incineration plants. It should be recognised that older incinerator plants are 'the worst case' scenario for risk assessment purposes, since modern EfW plant conforms to much stricter emission standards. The literature indicates that a number of the incinerators that were in operation in the middle of the last century caused significant environmental pollution to the local environment. There is also some very limited evidence that some adverse health effects may have occurred to members of the local community. However in judging the relevance of this information to the proposed plant it must be borne in mind that the emission levels of many of the chemicals of interest from these incinerators were probably between a hundred and a thousand times higher than the maximum EU/UK permitted emissions for the proposed plant. Published findings on modern plants

(i.e. performing within the EU emission standards) indicate that they do not cause significant environmental contamination and are without any detectable adverse effects on the surrounding communities.

5.6 The above conclusions are in line with those of a DEFRA review (2004) and Health Protection Agency (2009) which considered possible effects including cancer, respiratory diseases and birth defects,' but found no evidence for a link between the incidence of disease and the current generation of incinerators'. Similar conclusions have been reached by various other expert bodies in the UK, EU and USA. Proposals for new Incinerators/waste to energy plants are subjected to a high level of scrutiny in the UK because of political/ public concerns that are founded on poor functioning of incinerators prior to the introduction of EU regulations starting in the early 1990's. It is noted that in no case has planning permission for an incinerator/waste to energy plant been rejected because of possible health effects arising from airborne emissions.

5.7 Each of the pieces of evidence that have been considered support the conclusion that the proposed plant will not have an adverse effect on the health of the local population

## GLOSSARY

kg	Kilogramme. 1 kilogramme is 1000 grammes.
mg	milligramme. 1 milligramme is 1/1,000 of a gramme.
µg:	microgramme. 1 microgramme is 1/1,000,000 of a gramme.
ng	nanogramme. 1 nanogramme is 1/1,000,000,000 of a gramme.
ng/m <sup>3</sup>	nanogrammes of chemical per cubic metre of emission, or ambient air.
pg	picogramme. 1 picogramme is 1/1,000,000,000,000 of a gramme.
fg	femtogramme. 1 femtogramme is 1/1,000,000,000,000,000 of a gramme
Antagonistic	The ability of a chemical to negate the biological effect of another.
Bioavailable	The degree to which a chemical becomes available to the general (systemic) circulation or to a particular target organ.

CPF	Carcinogenic Potency Factor, a measure of the cancer causing potency of a chemical.
DETR	Department of the Environment, Transport and the Regions.
Dioxins	A family of 210 chemicals which are produced in trace quantities from combustion and other sources.
DoH	Department of Health
EA	The Environment Agency, the authorising authority for the proposed facility.
EAL	Environmental Assessment Level, an air quality guideline developed by the Environment Agency.
Epidemiological Studies	Investigation of the incidence, distribution and causative factors of diseases in the human population
FAO	Food and Agriculture Organisation – a WHO body.
Genotoxic	The initiation of cancer by a mechanism involving chromosomal/DNA changes.
GLC	Ambient air ground level concentration of a chemical emitted from the stack.

Hazard Index	The sum of individual hazard indices.
Hazard	The ratio of the concentration of a chemical to its safe level.
HMIP	Her Majesty's Inspectorate of Pollution, the predecessor to the Environment Agency.
HSE	Health & Safety Executive.
IPC	Integrated Pollution Control, legislation passed under the Control of Pollution Act 1900, under which the proposed facility will be authorised..
IPPC	Integrated Pollution Prevention and Control, legislation which will supersede IPC.
Lipophilic	Dissolves in oil, rather than water
Mutagenicity	The ability of a chemical to cause a permanent transmissible change in the genetic material of a cell.
Neurotoxicity	toxic effect on the nervous system
Non-criteria Pollutants	Metals, volatile organic compounds and dioxins
OEL	Occupational Exposure Limit

PAHs	Polycyclic Aromatic Hydrocarbons, a class of chemicals emitted in trace quantities from a variety of combustion sources.
Precautionary Approach	One that uses conservative assumptions with the intention of ensuring a high level of public protection
TEF,TEQ	Toxic Equivalency Factor and Toxic Equivalents, a means of expressing the toxicity of a mixture of dioxins.
Tolerable Daily Intake (TDT)	A value set by WHO/FAO for the level of contaminants in food that is deemed to be safe for consumers.
Toxic effect	An adverse effect on the person when the chemical enters the body.
Trace amounts	Very low levels of chemicals, usually less than one part in a million
Sensitisation	Have become hyper-sensitive
Synergism	When the effects of two chemicals in combination are greater than additives
Teratogenicity	Malformation of the foetus
VOCs	Volatile organic compounds, a family of trace organic

chemicals emitted from a variety of combustion and other sources

Volatile A chemical which readily evaporates from its liquid at room temperature

WHO World Health Organisation