Appendix 3

Literature review of potential ecological effects of mercury, dioxins, thallium and cadmium on bird receptors and otter

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1. Introduction

Considerable public, scientific and regulatory concern exists regarding the possible adverse health effects of chronic exposure to trace levels of persistent organic pollutants and heavy metals. In particular the class of compounds made up of the polychlorinated dibenzo-p-dioxins (PCDD) and polychlorinated dibenzofurans (PCDF), often collectively known as dioxins, has received widespread attention and attracted a great deal of research. The following report attempts to examine this research in the light of the proposed Ringaskiddy Resource Recovery Centre, with a view to determining potential environmental impacts of the Waste-to-Energy (WtE) plant. This literature review will focus on the impacts of such compounds on wildlife, particularly those occupying the designated areas adjacent the proposed development site i.e. Cork Harbour SPA and Great Island Channel SAC. This will help to inform the Environmental Impact Assessment Report (EIAR) and NIS for the licence application.

2. History of dioxin and heavy metal emissions from incineration

Waste incineration has a long history in Europe, with the first dedicated waste incinerators dating back to 1876 in the UK (Bontoux 1999). In the past, many US cities had thousands of residential incinerators in the city without any air pollution controls. At one time New York City had an estimated 18,000 residential incinerators and 32 municipal incinerators (Psomopoulos *et al.* 2009), the environmental impacts of which can still be detected in deep lying cores of the Central Park soil. Activities such as these have understandably left a lasting bad impression of waste incineration. During this time, when the effects of emissions on health and the environment were not well understood, all high temperature processes, including metal smelting, cement production, coal-fired power plants and incinerators, were the sources of enormous emissions to the atmosphere. In particular, incinerators were the major sources of toxic organic compounds (dioxins and furans) and heavy metals such as mercury. However, legislation on emissions over the last two decades has led to huge reductions in waste incineration emissions.

The first EU legislation to reduce dioxin emissions from municipal waste incineration was adopted in 1989. This set out operational conditions, or Emission Limit Values (ELV) , which led to significant reductions in dioxin emissions (Waste Incineration Directive 2000/76/EC). While difficult to quantify overall dioxin trends in the EU due to differences between the EU Member States, it is estimated that industrial sources of dioxin and furan emissions in the EU have reduced by almost 80% over the last 20 years (EC, 2006). Individual member states have also published reports supporting this trend. In Germany, for example, incinerator dioxin emissions have fallen from 400g to less than 0.5g per annum and now represent less than 1% of total dioxin emissions (German Federal Ministry, 2005). In a long-term study of a Portuguese Municipal Solid Waste (MSW) incinerator operational post 2000, no significant differences were recorded in the levels of dioxins and furans found in breast milk and human blood, before and after the opening of the facility (Coutinho 2012). Similar trends have been observed in the US. In 1995, the US EPA adopted new emissions standards for WtE facilities pursuant to the Clean Air Act. These regulations dictated that WtE facilities with large units (i.e., >227 tonnes per day) should comply with new Clean Air Act standards by December 2000.

While WTE companies in the US represent 84% of the total Municipal Solid waste combusted, they account for 0.54% of the controlled industrial dioxin emissions, and 0.09% of all dioxin emissions from controlled and open burning sources (Dwyer & Themelis, 2015). Several studies suggest that WtE facilities now represent less than 1% of the US emissions of dioxins and mercury (US EPA, 2003; Millrath *et al.* 2004; Psomopoulos et al. 2009).

Within the EU 25 member states the main sources of dioxin emissions are from non-industrial sources, with 30% from residential combustion and 15% from backyard burning (EC, 2006). Similarly, in the US backyard burning emits close to 600g of dioxins annually, accounting for the majority of dioxin emissions (Deriziotis, 2004).

Falling trends in incineration dioxin emissions have been observed in other developed countries, particularly Japan, whose main method of waste management is incineration. According to United Nations Environment Programme (UNEP) data, Japan generated almost 40% of the total worldwide dioxin emissions in 1995 (UNEP, 1999). Other reports from the same period concluded that while previously the largest source of dioxin-like compounds was from agricultural sources, municipal waste incineration had then become the largest source of dioxin emissions in Japan (Masunaga and Nakanishi, 1999; Masunaga *et al.*, 2001). Following these reports, an intensive programme of measures was implemented to reduce dioxin-like emissions. This has led to a 95% reduction in the amount of dioxins emitted between 1997 and 2003 (Yoshida *et al*. 2009).

In line with the rest of the EU, the dominant sources of dioxin emission in Ireland are from non-industrial activities (Concannon, 2014). Since 1995, the Irish EPA have regularly monitored dioxin levels in cow's milk. The primary mechanism for dioxins entering the food chain is through atmospheric deposition. Therefore, cow's milk is seen as a suitable means of assessing the presence of dioxins in the environment, since cows tend to graze over relatively large areas and these compounds will, if present, concentrate in the fat content of the milk. Levels of dioxins in cow's milk have been consistently low since the surveys began. The levels of dioxins found in the most recent surveys are well below the EU limit in milk and milk products of 2.5pg WHO-TEQ/g for dioxins only, and 5.5pg WHOTEQ/ g for dioxins and PCBs combined (Concannon, 2014). The EPA results are in line with results from Cork County Council's animal health surveillance programme, which has been operating in the Cork Harbour Region since 1991 (Buckley *et al.*, 2013). The Cork Harbour report found dioxin, furan and PCB levels were significantly less than the applicable limits. This study, which began in 2005, found dioxins and furan levels remained generally stable at values considered as low background levels in European terms.

3. Dioxins and Furans

3.1 Impact of dioxins on wildlife

After the Second World War, scientists began to recognise that certain chemical pollutants were capable of persisting in the environment for long time, migrating in air, water, soil and sediments and accumulating to levels that could harm wildlife and human health. These chemical pollutants were labelled persistent organic pollutants (POPs) (Manahan 2000). Because they can be transported by wind and water, most POPs generated in one country can and do affect people and wildlife far from where they are

used and released. They persist for long periods of time in the environment and can accumulate and pass from one species to the next through the food chain. Many POPs were widely used during the boom in industrial production after World War II, when thousands of synthetic chemicals were introduced into commercial use. Many of these chemicals proved beneficial in pest and disease control, crop production, and industry. These same chemicals, however, have had unforeseen effects on human health and the environment. POP's include Polychlorinated Biphenyls (PCBs), DDT and dioxins. While some POPs are intentionally produced for pest control (e.g. DDT) or for industrial processes (PCBs), others are unintentional by-products of industrial processes or combustion (e.g. dioxins and furans).

"Dioxins" is a collective term for the category of 75 polychlorinated dibenzo-para-dioxin compounds (PCDDs) and 135 polychlorinated dibenzofuran compounds (PCDFs). Seventeen PCDD and PCDF compounds are considered to be of toxicological significance. The most toxic of these is 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD). The toxic responses include skin effects, immunotoxicity and carcinogenicity, as well as reproductive and developmental toxicity. These compounds, or congeners, arise mainly as unintentional bi-products of incomplete or poorly controlled combustion and from certain chemical processes.

In the Great Lakes area of the US, which has been extensively polluted with dioxin and dioxin-like compounds, multiple species of birds, fish, reptiles, and mammals have exhibited developmental toxicity, reproductive impairment, compromised immunologic function, and other adverse effects correlated with these exposures. Specific observations correlated with dioxin or dioxin-like compound levels in multiple vertebrate species included hyperplasia of the thyroid and adrenal glands, porphyria, suppressed T-cell-mediated immunity, mammary and ovarian pathologies, reduced viability of offspring, congenital malformations, growth retardation, and an edematous syndrome among the offspring of fish-eating birds comparable to chick edema disease (Fox 2001).

In animal studies, TCDD and dioxin-like chemicals demonstrate many effects including: altered transcription of genes; induction of various enzymes; wasting syndrome; hepatotoxicity; altered immune function; testicular atrophy; altered thyroid function; chloracne; porphyria; neurotoxicity; teratogenicity; and carcinogenicity (EPA, 2004). Laboratory studies on birds have shown a variety of effects from dioxins including lethality, chick oedema, decreased growth rates (Hoffman *et al.* 1996) decreases in locomotory responses, deficits in body motions and balance, aggressive behaviour (Dahlgren and Linder 1974, Kreitzer and Heinz 1974) and changes in brain neurotransmitters (Sharma *et al.* 1976). Fish eating birds which inhabit areas contaminated with TCDD are chronically exposed during embryonic development via the yolk and this has anti-oestrogenic effects (Janz and Bellward 1996). *In ovo* exposure to these compounds during the perinatal period may also be responsible for certain behavioural characteristics and reproductive dysfunction.

Hart *et al.* (1991) examined the relationship between concentrations of PCDDs and PCDFs in Great Blue Heron (*Ardea herodias*) eggs and the effects on chicks. Eggs were collected from sites of low, intermediate and high contamination. Levels of TCDD in eggs were 10 ng/kg (wet weight), 135 ng/kg and 211 ng/kg, respectively. There was little difference in mortality of chicks from eggs collected from the various sites, suggesting no effect on survival at levels of PCDD/PCDFs seen in the eggs. Effects of contamination included decreased growth with increased TCDD level, depression of skeletal growth

with increased TCDD levels and subcutaneous oedema which increased with increasing PCDD and PCDF contamination. Also observed were shortened beaks and a scarcity of down follicles in the chicks from the more contaminated sites.

Uptake of PCDD and PCDF in laying ducks was determined at different degrees of feed contamination (Shun-I Shih *et al.* 2009). To observe the extent of the transfer of 17 PCDD/Fs from feed to the duck eggs and duck meat, 18 ducks were divided into three groups (six in each group) and fed feed with two different levels of PCDD/Fs. As a control, one group of ducks was fed with the non-contaminated feed for comparison, while the other two groups were exposed to the feed mixed with fly ash. The experiment lasted for 60 days, with an exposure duration of 41 days and the subsequent non-contaminated feed being given for an additional 19 days. PCDD/F levels in the eggs of the all three groups were observed to increase significantly on the 15th day. For the low contaminated group, PCDD/F levels reached 2.61pg WHO-TEQ/g lipid at day 41, whereas those of the high contaminated group accounted exceeded 3pg/g lipid on the 15th day. Furthermore, PCDD/Fs levels in the duck meat were analysed before and after exposure duration, and at the end of the experiment. The results showed that the level of PCDD/F in the duck eggs and the duck meat may reach unacceptable levels due to the effect of accumulation, although the PCDD/Fs in the duck feed were at acceptable levels.

3.2 Dioxins in birds and incineration

A number of studies have attempted to examine the direct links between dioxin contaminated sites and morbidity effects in birds occupying adjacent habitats. Rumbold *et al.* (1997) examined levels of TCDD, tetrachlorodibenzofuran (TCDF) and selected metal concentrations in eggs and nestlings of Anhingas (*Anhinga anhinga*) and White Ibises (*Eudocimus albus*) collected from a colony next to a Florida municipal solid-waste (MSW) combustor and ash landfill. Most of the measured residues, including TCDD, TCDF, arsenic, beryllium, cadmium and nickel, remained at pre-operational levels during the first five years of facility operation. Selenium (in Anhingas) and mercury (in both Anhingas and Ibises) occurred at their lowest concentrations in samples collected during the fifth year of facility operation (Year-5). Concentrations of lead in Ibis nestlings were highest in Year-1 and Year-5 compared to Year-0. The MSW combustor could neither be ruled out nor confirmed as the source of this lead. In a follow-on study at the same facility Rumbold and Mihalik (2002) found that concentrations of most residues, including TCDD, TCDF, arsenic, beryllium, cadmium and lead, were at levels comparable to those found during previous survey prior to facility start-up. Nickel residues were detected only sporadically in Anhingas and in eggs from Ibises, and decreased significantly in concentration in Ibis nestlings in 1999 compared to 1989. While concentrations of mercury in Anhinga nestlings and in eggs of both species were comparable to 1989 levels, its concentration was significantly greater in Ibis nestlings in 1999. However, levels of mercury in the Ibises remained relatively low when compared to birds from other areas of Florida and did not appear to represent a health threat. While lead did not increase in 1999, its concentration in Ibis nestlings remained a concern. The most notable temporal trend observed in birds at this site was a general monotonic decrease in levels of selenium residues during the 10-year monitoring period.

In 1991, eggs of the Common Tern (*Sterna hirundo*) were collected at eight different locations and incubated artificially (Bosveld *et al.* 1995). The toxicity of a mixture of dioxins and dioxin-like compounds can be expressed in a single number - the toxic equivalency (TEQ) and this was used as a metric to examine the residual yolk sacs of

the hatchlings. Highly polluted colonies, located in the main sedimentation area of the Rhine and Meuse rivers, contained on average 16ng TEQ per gram lipid, which was fivefold higher than the concentrations in the reference colony. The hepatic EROD activity (a highly sensitive indicator of contaminant uptake) was induced fourfold compared to the reference colony ($p < 0.005$). At the individual level, a significant relationship was found between log TEQ and EROD levels. Average TEQ concentration in chicks that hatched after 23 d of incubation were twice the concentration of those that hatched after 21 d ($p < 0.05$). No concentration-related effects on morphology and physiology were found. The non-ortho- and mono-ortho-PCBs were predominant regarding the total TEQ, while the PCDDs and PCDFs contributed less than 10%. The PCB patterns were highly similar among the individual birds and among different locations. In contrast, PCDD and PCDF patterns were not similar at all locations, and two distinct patterns could be recognised and related to sediments that were deposited during different time periods.

Naito and Murato (2009) carried out a population-level assessment of the ecological risks of dioxin-like PCB exposure to piscivorous birds in Tokyo Bay, Japan. Egg mortality risk and changes in the population growth rate in relation to the contamination levels of dioxin-like PCBs in eggs of Grey Heron (*Ardea cinerea*), Great Cormorant (*Phalacrocorax carbo*), Osprey (*Pandion halieaetus*), and Kingfisher (*Alcedo atthis*) were determined by integrating the results of exposure analysis, effect analysis, and a life-history model for each species. The results concluded that the levels of dioxin-like PCBs observed in the Tokyo Bay area alone would not have significant population-level effects on the fish-eating bird populations. This study follows a 95% reduction in Japanese incinerator dioxin emissions between 1997 and 2003 (Yoshida *et al.* 2009).

It should be noted that the majority of the studies referred to above were carried out prior to the implementation of the Waste Incineration Directive in the EU and the Clean Air Act in the US, which as discussed in **Section 2** of this review, has led to substantial reductions in incinerator emissions.

4. Heavy metals

4.1 Impacts of mercury in ecosystems

Mercury is a well-studied environmental contaminant, with wide-ranging impacts on the health of various taxa (Scheuhammer *et al.* 2007). Because mercury can be methylated and therefore become bioavailable in aquatic systems, it has historically been considered a problem for species directly associated with aquatic ecosystems, such as piscivorous birds (Wolfe *et al.* 1998).

There is a huge scientific literature on the distribution of mercury in ecosystems. Mercury has been measured in aquatic and terrestrial invertebrates, in a variety of plants, and in many higher organisms including humans. High concentrations of mercury have been associated with developmental and behavioral abnormalities, impaired reproduction and survival, and in some cases with direct mortality.

During the decades when seed grains were treated with organo-mercurial fungicides, huge numbers of wild birds were poisoned fatally with mercury. In the 1970s, declining

use of organo-mercurial fungicides greatly reduced the severity of mercury exposure. However, mercury residues either through natural or anthropogenic sources remain. Several reports of mercury concentrations in avian species have been published in the peer-reviewed literature (Bowerman *et al*. 1994; Spalding *et al*. 1994; Langlois and Langis 1995). Based on historical and recent information, mercury is a common contaminant of avian tissues from diverse geographic locations. The feeding habits of particular avian species have been major predictors of risk of mercury toxicity. When seed grains were treated with organo-mercurial fungicides, herbivorous, omnivorous, and carnivorous species were all at risk of mercury toxicity. Because of the bio-magnification of methylmercury in the aquatic foodweb, birds which feed on fish, crayfish or shellfish now have higher exposures to methylmercury than do non-fish eating birds. Birds, such as Grey Heron, that consume large fish as their prey, are predicted to be at greater risk of methylmercury poisoning than birds that consume smaller fish (Spalding *et al*. 1994; Sundlof *et al*. 1994). When the quantities of fish consumed on a body weight basis is also considered for smaller birds such as the Kingfisher, there is an elevated risk of methylmercury poisoning. Senthilkumar *et al.* (2002) found that as well as feeding habits, specific elimination and metabolism influenced differing contamination patterns between species.

Several estimates exist on mercury concentrations in soft tissues (liver, kidney, brain) that are associated with mercury poisoning in bird species. Experimental studies of survival and reproductive success of Black Ducks (*Anas rubripes*) indicated that adult ducks would tolerate liver mercury concentrations of 23 ppm and appear in good health (Findley and Stendell 1978). However, it was found that although the Black Ducks fed methylmercury in their diet appeared in good health, they had impaired reproductive success as indicated by reduced hatchability of eggs and high duckling mortality. Findley *et al.* (1979) concluded that concentrations of mercury in excess of 20 µg/g fresh weight in soft tissues should be considered extremely hazardous to avian species. Scheuhammer (1991) indicated that the major effects of methylmercury in avian species were neurological, developmental and reproductive. The neurological changes included weakness, walking or flying difficulties and incoordination that were associated with brain mercury concentrations of 15 µg/g (fresh weight), or liver or kidney mercury concentrations of 30 µg/g (fresh weight). Schuehammer (1991) observed that significant reproductive impairment due to methylmercury occurred at about one-fifth the tissue concentrations required to produce overt neurotoxicity. Liver mercury concentrations of 2 to 12 µg/g (fresh weight) in adult breeding Pheasants (*Phasianus colchicus*) and Mallard (*Anas platyrhynchos*) were linked to decreased hatchability of eggs (Schuehammer 1991). Barr (1986) noted that clinical signs of mercury poisoning, such as impaired vision and ataxia in loons. Barr (1986) also noted that impairment of vision or ataxia in a visual hunter such as loon would be likely to reduce its chances of procuring adequate food and defending a territory.

Mercury concentrations in the livers of wading birds in Southern Florida (Sundlof *et al.* 1994; Spalding *et al.* 1994) and the merganser in northern Quebec (Lanlois and Langis 1995) are in the range associated with adverse reproductive and neurological effects in other species of birds. Sundlof *et al.* (1994) reported that four Great Blue Heron collected from the central Everglades contained liver mercury at concentrations typically associated with overt neurological signs (>30 µg mercury/g fresh weight). Furthermore, these investigators found between 30% and 80% of the potential breeding-age birds collected in an area encompassing the central Everglades contained liver mercury at concentrations associated with reproductive impairment in ducks and Pheasants. In a

parallel study, Spalding *et al*. (1994) determined the magnitude of mercury contamination associated with death of Great White Herons (*Ardea herodias occidentalis*). Birds that died of acute causes (e.g., trauma from collision with power lines or vehicles) had much lower liver mercury concentrations (geometric mean 1.8 µg/g fresh weight, range 0.6 to 4.0 µg/g fresh weight) than did birds that died of chronic diseases (geometric mean 9.8 µg/g fresh weight, range 2.9 to 59.4 µg/g fresh weight).

Dietary methylmercury reduces the appetite and growth rates of baby birds. In the baby birds fed methylmercury (Spalding *et al.* 2000a) at 0.5 mg/kg body weight (comparable to doses encountered in the wild) anemia, feather abnormalities, neurological changes, and immunology damage occurred. At higher doses birds showed gait disturbances. Birds in the wild died at lower doses than laboratory birds, presumably due to multiple stressors (Spalding *et al.* 2000b).

Although the hazards of environmental mercury were already well recognized by 1970, there was considerable speculation regarding the relative contribution of anthropogenic versus natural sources. Hammond (1971) questioned whether the magnitude of human releases was sufficient to alter the concentration of mercury in the marine ecosystem. Thompson *et al.* (1998) addressed this question by showing that the mercury content of seabird feathers had increased between 65% and 394% (or 1-4% per year) in five species of North Atlantic seabirds for which pre-1931 and post-1979 feather samples were available. Based on mercury levels in the feathers of museum specimens, mercury levels in carnivorous birds in Europe were low prior to the mid-20th century and then increased, reflecting the increased anthropogenic (both industrial and agricultural) contribution to the environment. Odsjö (1975) documented the dramatic jump in mercury concentration of Scandinavian Goshawk (*Accipiter gentilis*) feathers from < 5 µg/g prior to 1940 to about 20 µg/g after 1940. Peregrine Falcons (*Falco peregrinus*) averaged less than 3 µg/g prior to 1940, jumping to almost 38 µg/g (1964-1966) and declining to 7-17 µg/g in the 1970s (Lindberg and Odsko 1983) as agricultural uses and industrial pollution were curtailed. Smaller differences occur in non-predatory species, such as the Bar-tailed Godwit (*Limosa lapponica*) in the Netherlands where values increased from 0.4 µg/g (1904-1963) up to 2.0-4.9 µg/g (1979-1982).

Monteiro *et al.* (1999) examined mercury concentrations in egg and plumage samples of adults and chicks of seabirds from colonies in the Portuguese Atlantic. Species and tissues were selected to ensure varied levels of ecological (epipelagic vs mesopelagic), spatial (coastal vs pelagic) and temporal (short-to medium-term) integration of mercury contamination. Results show a four-fold increase in mercury bioaccumulation from the epipelagic to the mesopelagic areas. Such background uniformity of mercury contamination in the North Atlantic conveys further evidence of global pollution by mercury due to atmospheric deposition at long distance from emission sources.

4.2 Waste incineration and mercury in birds

Probably no area with mercury pollution has been as extensively studied as the Everglades in south Florida. The Everglades system was historically highly contaminated with mercury, which had been linked to local waste incineration (Dvonch *et al.* 1999, Frederick, 2000). Elevated methylmercury concentrations were recorded in many species of wetland wildlife and have been extensively studied in Great Egrets (*Egretta alba*), one of the top trophic level piscivorous species.

However, the implementation of the Clean Air Act in 2000 led the significant decreases in local mercury inputs and Lange *et al.* (2000), found that the mercury levels in Everglades Egrets declined rapidly. Frederick *et al.* (2002) examined mercury contamination in piscivorous birds over six years in the Everglades. Species examined included Great Egret and White Ibises. Over the course of the study, Everglade's colonies maintained their mercury concentration rankings relative to one another, but all showed strongly declining mercury concentrations (mean of 73% averaged across colonies, between 1994 and 2000). The study concluded that the Everglades has undergone a biologically significant decline in mercury availability in the wetland food web, possibly because of decreased local inputs.

Herring *et al.* (2008) attempted to link mercury contamination in the Florida Everglades with declines in the numbers of wading birds. Feather mercury levels in Great Egret and White Ibis nestlings were assessed. Nestlings were in better physiological condition when feather mercury levels were higher. These results support the hypothesis that nestlings are protected from the harmful effects of mercury through deposition of mercury in growing feathers. The study also found evidence to suggest shifts in diets of the two species, as a function of prey availability, thus altering their exposure profiles. However, no evidence was found to suggest that birds responded differently to mercury exposure.

Blanco *et al*. (2004) assessed whether levels of heavy metals in blood have detrimental effects on the health of pre-fledgling Black Kites (*Milvus migrans*) exposed to emissions from a solid waste incinerator near Madrid, central Spain. These results suggest that the participation of metallothioneins in detoxification and metal regulation may also indirectly enhance the adrenal stress response to contaminants, probably through a competition of Cd and Zn for Zn binding sites on metallothioneins, reducing the ability of Cd to stress the immune system. In a more recent study, Carneiro *et al.* (2018) found that free-living Black Kites had almost four-fold higher blood mercury levels compared to captive birds.

Birds are clearly vulnerable to mercury contamination. The majority of the research done to date, however, has been focused on select taxa (waterbirds), trophic levels (piscivores), habitat types (aquatic systems), geographic regions (North America and Europe), and life history stages (reproduction), leaving the assessment of mercury's threats to birds incomplete. While much is known about mercury's effects on birds, many relationships between mercury and bird biology remain poorly understood. The taxonomic groups, geographic ranges, life history stages, habitat associations, and foraging guilds of birds that are significantly threatened by mercury pollution need to be better identified.

4.3 Other Heavy Metals

Cadmium (Cd) occurs in association with zinc and is found in soils, mud, humus and organic matter. It is a significant anthropogenic contaminant, but soils derived from carboniferous black shales may contain up to 200 ppm. Major environmental sources of cadmium include electroplating, zinc and lead mining and smelting, paint and pigments, batteries, plastics, coal-fired power plants, and municipal wastewater and sewage sludge. Cadmium is a known teratogen and affects calcium metabolism causing excess calcium excretion, which negatively impacts both skeletal and cardiovascular systems (Eisler 1985). In addition, growth retardation, anemia, and testicular damage occur in cadmium-exposed wildlife (Eisler 1985). Cadmium is readily bio-accumulated and

available data suggests that it is biomagnified through food chains (Larison *et al.* 2000). Cadmium is readily taken up from the soil (especially by leafy plants and under acid pH) but, although the initial rate of uptake can be linear relative to soil concentration, it tends to tail off with time (Vernet, 1992). Cadmium is relatively mobile and shows potential for accumulation through the lower terrestrial trophic levels, although leaf crops are more problematic than root or seed crops from a human perspective (Lepp 1981). It accumulates in the soft tissues of grazing animals where it is highly toxic, with pasture herbs being a more dangerous pathway of transmission than grasses. White-tailed ptarmigan (*Lagopus leucura*) in Colorado are at threat from cadmium poisoning due to bio-magnification in willow (*Salix* spp.), a primary food plant for these birds (Larison *et al.* 2000). Cadmium residues in vertebrate kidney or liver that are >10 ppm fresh weight or 2 ppm whole body fresh weight should be viewed as evidence of probable cadmium toxicity; residues of 200 ppm kidney (fresh weight), or >5 ppm whole animal fresh weight are indicative of cadmium poisoning. Wildlife, especially migratory birds, which feed on crops growing in fields fertilized with municipal sewage sludge, may be at considerable risk from cadmium toxicity (Eisler 1985).

Thallium (Th) is often chemically associated with arsenic, and may also be concentrated in soils derived from weathered limestone (Lepp 1981). The main anthropogenic source is flue gases, primarily from coal burning, non-ferrous smelting and cement production (Alloway 1995, Nriagu 1998). In this context, the contribution from MSW incineration seems negligible, but this may be understated since MSW and industrial wastes have been used to fuel cement kilns in France since 1986 (Porteus 1996). The forms of thallium found in flue gas are fairly soluble and affect both plants and animals (enzyme inhibitor), the toxicity to animals possibly being enhanced by microbial biomethylation (Lepp 1981). Although soil/plant exchange increases at acid pH, thallium mobility in the aquatic environment is largely independent of major water characteristics such as hardness. Aquatic food-source invertebrates such as Daphnia are affected at levels below the vertebrate toxicity threshold and therefore have indirect effects upon fish stocks (Nriagu 1998).

Cadmium may arise in air emissions of MSW incinerators due to its thermal mobility. Although some countries restrict applications of Cd, frequent sources in MSW incinerators are electronic devices (including accumulators), paints, Ni-Cd batteries, and cadmium-stabilised plastics. Under the conditions usually observed in the furnace, Cd is mainly converted into $CdCl_2$ (Quina *et al*. 2011). The air pollution control (APC) devices normally lead to a concentration of Cd in APC residues, and less than 1% is released to the atmosphere. The reported range is usually 0.0002-0.03 mg/Nm3 (11% of O2) (BREF, 2006). The thallium amount in municipal solid waste is virtually non-existent and very often below any detection limit (Quina *et al.* 2011).

5. Mammals

Piscivorous mammalian wildlife, such as Otter and Mink, are exposed to mercury in the same way as piscivorous birds i.e. via the consumption of contaminated fish. Concentrations of mercury in wildlife have been observed at concentrations causing adverse effects in laboratory studies using the same species (U.S. EPA 1997). Toxic effects from the consumption of contaminated fish have been observed in mammalian wildlife in areas with point sources of mercury emission (U.S. EPA 1997). The U.S. EPA (1997) investigated population impacts on piscivorous wildlife and estimated an adverse

effect level for methylmercury in trophic level 3 fish (between 0.077 and 0.3 µg/g). This indicates that it is possible that individuals of some highly exposed wildlife subpopulations are experiencing adverse toxic effects due to mercury in the food chain. In a study of Mink and Otter, the U.S. EPA (1997) concluded that field data were insufficient to determine whether these species suffered adverse effects.

Zwiernik *et al.* (2008) examined dioxin and furan levels in Mink at Tittabawassee River, Michigan, U.S. where sediments have been historically contaminated with dioxins, furans and PCBs. Significant concentrations of PCDF's and PCDD's have been recorded within the sediments, and the area is a major industrial and population centre along the river. However, the resident mink survey, including number of mink present, morphological measures, sex ratios, population age structure, and gross and histological tissue examination, indicated no observable adverse effects. Based on the lack of negative outcomes for any measurement endpoints examined in the study and direct measures of effects on individual mink and their population it was concluded that these high concentrations of PCDDs/PCDFs were not causing adverse effects on resident mink of the Tittabawassee River.

Otters are consistently fish eaters whose diet consist of at least 95% fish (Toweill and Tabor, 1982). Mercury concentrations in Otter tissues have been positively associated with mercury levels in prey (for example; fish, shellfish, crayfish) (Wren and Stokes, 1986; Foley *et al.* 1988; Langlois and Langis, 1995). Otter accumulated about ten times more mercury on a concentration basis than did predatory fishes from the same drainage areas (Kucera, 1983). These correlations were statistically significant (Foley *et al.* 1988) on the basis of mercury in the watershed because of the importance of fish, shellfish and crayfish in the diet of Otter. Case reports of clinical mercury poisoning exist for wild Otter (Wren, 1985). The magnitude of the concentration in one organ for Otter (for example, liver) is highly correlated with other organs (for example, kidney or brain) (Wren and Stokes 1986). The range of concentrations reported in different geographic locations is substantial. Consequently, broad generalisations are difficult regarding how close liver mercury concentrations of wildlife are to liver mercury concentrations of experimentally poisoned Mink. However, the upper range of liver mercury concentrations of Mink from northern Quebec (Langlois and Langis, 1995), Otters from Georgia (Hallbrook *et al.*, 1994) and Otters from Ontario (Wren *et al.* 1986) approximate those of clinically poisoned animals.

Lethal effects of mercury have also been recorded in the wild. Sleeman *et al.* (2010) reported a moribund 5-year-old female northern river otter (*Lontra canadensis*) was found on the bank of a river known to be extensively contaminated with mercury. It exhibited severe ataxia and scleral injection, made no attempt to flee, and died shortly thereafter of drowning. Tissue mercury levels were among the highest ever reported for a free-living terrestrial mammal: kidney, 353 µg/g; liver, 221 µg/g; muscle, 121 µg/g; brain (three replicates from cerebellum), 142, 151, 151 µg/g (all dry weights); and fur, 183 ug/g (fresh weight). Histopathologic findings including severe, diffuse, chronic glomerulosclerosis and moderate interstitial fibrosis were the presumptive cause of clinical signs and death. This is one of a few reports to document the death of a free-living mammal from presumed mercury poisoning.

Sub-lethal effects can be projected to be more wide-spread with additional reports showing average liver mercury concentrations approximately one-third of those in moribund mink with experimental methylmercury poisoning. Liver mercury

concentrations of river otters from the lower coastal plain in Georgia averaged 7.5 µg/g (Hallbrook *et al.* 1994); this is approximately 33% of the concentrations associated with severe intoxication and/or death in a closely related species, the Mink (Wobeser *et al.* 1976a,b). In many geographic regions [e.g. Georgia (Halbrook *et al.* 1994), New York State (Foley *et al.* 1988)], mercury concentrations in Mink and Otter tissues are 10-30% of the concentrations associated with severe, clinically evident methylmercury poisoning in Mink. For example, data showing Mink liver mercury concentrations averaging 2 µg/g or higher were reported in several regions of New York State (Foley *et al.* 1988), Ontario (Wren *et al.* 1986), and Manitoba (Kucera, 1983). Concentrations in excess of 20 µg/g occurred in Mink dying of methylmercury poisoning (Wobeser, 1973; Wobeser *et al.* 1976a, b). There may be other factors in addition to methylmercury concentration in the food supply of the mink and otter that are responsible for the association.

Another consideration is the combination effects of various contaminants on mammals. Polychlorinated biphenyls (PCB), DDT, dieldrin and mercury (Hg) are most often associated with the decline in Otter *Lutra lutra* populations in Europe (Norrgren and Levengood, 2012). However, it is possible that the PPCB/PCDD/PCDF congener profile is influencing toxicity in a way that is not accounted for. One complication of feeding mink diets containing fish collected from contaminated waters is that it is probable that other unaccounted contaminants are influencing the overall toxicity of the mixture. This may explain why Heaton *et al.* (1995) reported effects on kit survival and growth at dietary tPCB and TEQ concentrations that were 2.5-fold and 3.4-fold less, respectively, than the No Observed Adverse Effect Level (NOAEL) reported by Bursian *et al.* (2006a). Similarly, Bursian et al. (2006b) reported that diets containing 3.7 µg tPCB/g feed or 50.4 pg TEQ/g feed provided by carp collected from the Housatonic River, Massachusetts, caused reduced kit survivability, resulting in a LOAEL that is 5.6 and 3 times greater than the LOAEL reported by Heaton *et al.* (1995) when expressed on a tPCB and TEQ basis, respectively. There have been a few mink studies conducted with single PCB, PCDD and PCDF congeners that avoid the complexities associated with exposure to a mixture of congeners as well as other contaminants. Hochstein and associates conducted studies that involved exposure of mink to TCDD. The first study (Hochstein et al., 1988) established a 28-day LD50 of 4.2 µg TCDD/kg body weight. A 28-day LC50 of 4.8 ng TCDD/g feed and a 25-day LC50 of 0.85 ng TCDD/g feed was reported in the second study (Hochstein *et al.*, 1998). Beckett *et al.* (2008) reported that dietary concentrations of 24 and 2.4 ng 3,3',4,4',5-pentachlorobiphenyl (PCB 126)/g feed resulted in complete reproductive failure (2,400 and 240 pg TEQ/g feed, respectively), while a dietary concentration of 0.24 ng PCB 126/g feed (24 pg TEQ/g feed) was the NOAEL. Interestingly, Zwiernik *et al.* (2009) reported that an equivalent TEQ concentration (242 pg/g feed) provided by 2,3,7,8-tetrachlorodibenzofuran (TCDF) had no effect on reproduction and survivability of offspring.

6. Conclusions

Historically dioxin and mercury emissions from incinerators have been the cause of much public concern. However, the implementation of the EU Waste Incineration Directive 2000/76/EC and the Clean Air Act in the US has led to large reductions in incinerator emissions across the developed world. In Germany, for example, incinerator dioxin emissions have fallen from 400 grams to less than 0.5 grams a year, since the year 2000. Incinerators now represent less than 1% of total dioxin emissions in Germany. In Ireland dioxin levels are currently significantly less than applicable limits and are considered as low background levels in European terms.

Laboratory studies on birds and mammals have found an array of negative effects from dioxins and heavy metals. While several studies have attempted to replicate these effects in the wild, in areas which are heavy polluted, the results have been inconclusive. In one study, uptake of dioxins in the bird population resident near a waste incinerator were not increased in the ten years following the facility's opening. One complication of feeding animals diets containing fish collected from contaminated waters, appears to be that other unaccounted for contaminants can influence the overall toxicity of the mixture. While gaps in the research exist, between lab and field-based studies, it does appear that the impact of polluted sediments on animals is complex. The complexities of examining individual and combination effects of dioxins, PCBs and heavy metals in the field seem, at present, to be beyond the reach of researchers. However, it does appear that the contribution of waste incineration to these pollutants has decreased significantly. In the years since strict legislation governing waste incineration has been implemented, mercury concentrations in bird feathers has dropped significantly alongside declines in mercury inputs from industrial sources.

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