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REVIEW OF HEALTH RISKS ASSOCIATED WITH EMISSION OF DIOXINS IN HONG KONG

By Professor J W Bridges

Senate House

University of Surrey

Guildford, GU2 5XH, UK

Tel 0044-1483-873802

Fax 0044-1483-873803

<j.bridges@surrey.ac.uk>

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I Waste Management and Health Hazards: General Principles

In evaluating the risk to health from emissions that may arise from any waste management process, it is important to bear in mind the following:

- Virtually all waste materials contain substances that are a hazard to health. Many wastes contain trace amounts of dioxins.
- One of the requirements of any modern waste management option is that such substances are either destroyed or contained. In principle, the former option is preferable.
- During any process of waste management a fraction of the original contaminants may be released to the environment and/or new substances, generated by the waste management process may be emitted.
- Public exposure to emitted substances may arise from the waste before processing or during processing through:
  - Inhalation of the substances or products produced from them in the atmosphere
  - Contamination of food as a result of deposition and/or uptake by plants and animals;
  - Contamination of soil and other surfaces leading to dermal contact or inhalation of contaminated dust;
  - Contamination of drinking water.
- Emission profiles of substances that are hazardous to health differ according to the waste management option selected. Since all forms of waste treatment result in some emitted substances of potential hazard to human health, it is very unwise to select waste management options simply on the basis that they appear to have negligible emissions of a single substance or class of substances.
- A specific form of waste treatment is unlikely to result in any emitted substances unique to the source.
- Public concern and actual health risks often bear very little logical relationship to one another.

NB: In the UK the health effects from waste combustion and landfilling are the subjects of recent publications by the Institute of Environment and Health. Waste incineration is the subject of a House of Lords Select Committee publication in 1999.

As observed by Claude Bernard several centuries ago, all substances are toxic provided the dose is high enough and exposure occurs for a sufficient period of time. Thus, in judging whether or not there is a risk from a chemical, which evokes public concern, such as dioxins, we need to have information on:

i) the level of exposure, the duration of exposure and the physical form of the chemical(s) of concern;
ii) the inherent toxicity of the chemical of concern (often termed "the hazard") to humans from the route(s) of exposure considered relevant, eg: air, food, dermal contact;
iii) vulnerability of the individual who is exposed. For populations this is clearly not possible, so in practice vulnerable population groups need to be identified, for example, those with a particular disease, very young children, pregnant women, etc.

It follows that, without a reasonable estimate of exposure to a chemical of concern, no sensible estimate can be made of the risk. The general public often incorrectly confuses the hazard from a chemical as being synonymous with the risk. Consequently, great public concern may be aroused by the mere detection of a chemical or even the suspicion that it is present.

Regulatory authorities, wherever possible, set standards or guidelines for "chemicals of concern". For the great majority of chemicals, it has been demonstrated experimentally (or it is highly likely from the toxicological properties) that there is a threshold dose (exposure level) below which no adverse effects will occur. The standard (guideline) is set therefore at an appropriate value below this level. The actual level selected will depend on various considerations including:

- Confidence that the threshold level identified is valid and appropriate for public protection. Where there is doubt safety (uncertainty) factors are often employed to set a "safe" level.
- Any special considerations which are required to ensure protection of vulnerable groups. Again absence of suitable information may require the use of an additional safety (uncertainty) factor.
- Practical issues such as the economic implications of setting a particular values for a standard.
- Public and political factors

It follows from the above that as more information is obtained on a chemical it may be appropriate to revise the standard or guideline either by relaxing it or by making it stricter.
Dioxins are considered to be a class of chemicals for which there is a threshold dose (exposure level) and therefore, in principle, a "safe" level (standard or guideline) can be set.

II Dioxins

Background and History of Dioxins

As a result of an incident that occurred in a trichlorophenol manufacturing plant near the town of Sevaso in Italy in 1976, a large population was exposed suddenly to a major increase in the airborne levels of dioxins (Bertazzi et a/, 1998). The local food supplies and soil were heavily contaminated, as a consequence of the incident.
About the same time, concerns were expressed regarding the adverse human health effects, which resulted from the spraying of the defoliant 'Agent Orange' in the Vietnam War. This 'Agent Orange' comprised trichlorophenol heavily contaminated with dioxins (Kramarova E *et al, 1998)*. These events triggered a major effort to investigate the toxicological and other properties of dioxins. The problem was recognised from the outset to be complex because the term "dioxin" is used to describe a family of compounds comprising two related basic structures, for example:

- Polychlorinated dibenzo-para-dioxins (PCDDs) of which there are 75 different varieties (termed "congeners");
- Polychlorinated dibenzofurans (PCDFs) of which there are 135 congeners.

Particular attention in research has been given to one specific congener whose proper chemical name is 2,3,7,8 tetrachlorodibenzodioxin (often known as TCDD) because this is viewed as the most toxic.

As a consequence of this enormous research effort over the past twenty years we know a great deal about the toxic effects of dioxins in both man and animals (Mukeerjee D, 1998). Very few chemicals have ever been subjected to such extensive investigations. However, no toxicological database can be considered entirely complete. Despite the large quantity of research data on dioxins, it is appropriate to maintain a precautionary ("conservative") approach in defining an "acceptable" level for human exposure because they are persistent in the environment and the human body.

Expression of the Toxicity of Mixtures of Dioxin Congeners

There are some congeners of both PCDD and PCDF and different sources vary considerably in the levels of individual congeners. In view of this fact a standardised form of expressing the overall toxicity is required. The internationally accepted form is to use toxicological equivalence quotients or TEQs. This is based on an allocation of toxicity rating to each congener (so called a TEF). The most potent 2,3,7,8 tetrachlotodibenzodioxin (TCDD) is assigned a value of 1 and the remaining dioxins are assigned a value lower than 1. A TEQ in a particular source can be calculated by taking into account the percentage of each congener.

In 1998 WHO conducted a new assessment of the health risks from dioxins with a view to the re-evaluation of the tolerable daily intake (TDI). This report has not yet been published, but I have access to a final draft copy. The report covers the following themes:

- exposure
- toxicokinetics
- mechanistic aspects
- application of the TEQ concept
- health risks for infants
- cancer and non-cancer endpoints.

The report states that in all investigated countries over 90% of human background exposure to dioxins (PCDD's and PCDF's) occur through the diet with food of animal origin being the predominant source of exposure. The report identifies the sources of dioxins in the food from:

a) Deposition from various sources onto farmland and water bodies and subsequent bioaccumulation in terrestrial and aquatic food chains;
b) Contaminated feeds for cattle, poultry and farmed fish
c) Improper application of sewage sludge to land and waste effluents to sea

Analysis of intake of dioxins from many industrialised countries shows an average daily intake in order of 50-200 pg I-TEQ/person/day or 1-3 pg I-TEQ/kg body weight/day for an adult with the weight of 60 kg.

However, there are a number of substances, which have a similar mechanism of toxicity, for example many polychlorinated biphenyls (PCB's). If exposure to PCB's in the diet is added, then the total daily TEQ intake can be two or three times higher than the figures given above. Dietary habits of different population groups influence the total daily intake of dioxins. If the diet is high in animal fats, for example from milk, dairy produce and oily fish, the daily intake of dioxins will be higher than the average. Conversely, the levels of dioxins intake for vegetarians are likely to be lower than average.
In recent years better control of the composition of industrial emissions has resulted in a slow fall of dioxins in the diet in many countries.

Mechanism of Toxicity

Recently, the mechanism of toxicity of dioxins and analysis of their adverse effects in animal and man has been the subject of a number of national and international reviews (for example, USEPA, 1996; IARC, 1997; WHO 1998) as well as many conferences and workshops. The subsequent sections of this report represent a brief summary of the key points from the recent reviews. In view of its particular relevance to food I have used primarily the WHO report (1998, in press).

The scientific basis for the use of the TEF/TEQ is that the congeners have a common mode of action on biological systems. For toxicity to arise at low exposure levels the congeners must first bind to a receptor termed the Ah receptor. This binding then triggers a range of biological and toxicological events. The ability to bind to the receptor and persist on it governs the potency of the congener. TCDD is the most potent congener in this respect. Dioxins are very fat-soluble and therefore need to be metabolised in the body in order to be excreted. Congeners with the higher chlorine content tend to be the least well metabolised and tend to accumulate in the fatty (adipose) tissues (Mukerjee D, 1998). Lactating women secrete non-metabolised dioxins in the breast milk because milk has a relatively high fat content.

Toxicity

Dioxins may have the ability to produce a wide spectrum of responses in animals (Grassman *et al,* 1998). Presumably, if the dose is high enough dioxins may produce a wide range of effects in man. There is a debate on whether it is valid for this class of substances to rely principally on the use of animals' findings for risk estimate purposes. However, to ensure public protection it is reasonable to assume that the animal findings are relevant to humans (USEPA, 1997). A number of the effects produced by dioxins in animals are clearly adverse to health, while other effects might be considered as physiological responses, for example drug metabolism enzyme induction. The principal effects of toxicological concerns are summarised below. I have concentrated on human data for the purpose of this report:

a) Carcinogenicity

A number of epidemiological studies have been conducted on population groups exposed to high levels of dioxins due to an accident or an occupational exposure in specific industries In many of these studies there was simultaneous exposure to chemicals other than dioxins. This fact complicates interpretation of the findings.

From these studies the consensus view is that dioxins increased the risk for all cancers combined. However, the magnitude of this increase was low and there was no statistically significant increase in any particular type of cancer.

The relative risk for all cancers combined have been calculated at 1.4 (compared with a norm of 1). This difference is 'not likely to be explained by confounding factors (for example, life style, other chemicals) but this possibility cannot be excluded' (WHO Report, 1998, in press). Thus, the doubt remains as to whether dioxins are carcinogenic in humans.

However, animal studies support the view that TCDD (the most potent dioxin) is a carcinogen, although it is clearly not a typical (genotoxin) carcinogen. Thus, there may be, although this is not proven, a threshold level below which it will not produce tumours. This consideration is relevant to evaluating the significance of the reports of an association between all cancers combined and high level of dioxin exposure. In each of these epidemiological studies human exposure was a minimum 10-fold and a maximum 100-fold higher than that to which the general public is exposed typically.

In the animal studies the TCDD exposure was between a 100 and 1000-fold higher than the present background exposure in the human population. Based on this knowledge, in 1997 the International Agency for Cancer Research conducted an evaluation of the relationship between the magnitude of the exposure in the experimental systems and the magnitude of the response (i.e. dose response relationship). The evaluation does not permit conclusions to be drawn on the human health risks from background exposure to 2,3,7,8-TCDD. For all other dioxin congeners there is an even greater degree of uncertainty about their carcinogenicity at exposure levels experienced by the general population (IARC, 1997).

b) Effects on the nervous system of young children

Maternal ingestion of high levels of dioxins has been correlated with a variety of persistent severe adverse developmental and neurological effects in infants (Schecter *et al,* 1994).
This finding is supported by observations of adverse developmental and reproductive effects in both rats and monkeys. In animals studies alterations in learning behaviour were also observed. This effect appears to be strongest when the foetus is exposed in utero (i.e. before birth). A weaker effect has also been noted in weaning animals (EPA, 1996). It can be deduced from animal studies that a threshold for this effect is highly likely, but the actual level still remains uncertain.

c) Other effects

In humans other effects observed after a substantial dioxin exposure include increased incidence of cardiovascular disease and diabetes. In Seveso a persistent skin condition, termed chloroacne, also occurred (Batas *et al,* 1998). However, there was no evidence among the Seveso population of impaired immune competence. In other human studies there is some rather limited evidence of effects on immune system (Jung *et al,* 1998). In animals, however, there is clear evidence of immune suppression following in utero exposure. The immune system has a critical role in maintaining health, however, there is a general agreement among experts that for each of these effects a threshold is probable. But there is insufficient information in humans to determine the threshold level directly

d) Identifying a safe level (standard)

The WHO report identified a range for the lowest observable effect levels between 14 and 37 pg of TCDD/kg body weight/day. In view of the fact that a threshold value has not been confirmed for these effects, WHO applied an uncertainty (safety) factor of 10 and, therefore, arrived at a provisional standard Tolerable Daily Intake (TDI) value of 4 pg/Kg body weight/day with an ultimate goal to reduce human intake levels below 1 pg TEQ/kg body weight/day.
WHO report emphasised that 'the TDI represents a tolerable daily intake for life-time exposure and that occasional short-term excursions above the TDI would have no health consequences, provided that the average intake over long periods is not exceeded' (WHO, 1998, in press). The report recognised that 'certain subtle effects may be occurring in some sections of the general population of industrialised countries at current intake levels (2-6 TEQ pg/kg body weight/day. These subtle effects were not considered overtly adverse and that there were questions as to the contribution of non-dioxin compounds to these observed effects' (WHO, 1998, in press).
Although environmental levels of dioxins and, consequently, levels of dioxins in food and breast milk are falling, more efforts are required to limit the release of dioxin-like substances into the environment. This should be done in order to reduce the presence of dioxins in food chains. Zero dioxin emissions may be impossible to achieve, but tight limits need to be set on many processes that emit dioxins.
The European Union has introduced an emission standard of 0.1 ng/m3 for dioxins in respect of incineration. However, the EU has not set a standard for ambient air and there is no intention at present to do so. Presumably this state of affairs reflects the view of the EU experts that inhalation is a very insignificant route of exposure to dioxins.
In 1999 in the USA the Agency for Toxic Substances and Disease Registry (ATSDR) set a minimum risk level for acute duration oral exposure to TCDD using a different approach from that

of WHO (1998) of 200 pg/kg/day. ATSDR based their standard on findings in animal studies, rather than on human data (Pohl *et al,* 1999). The key concern was immuno-supression (increased susceptibility to infection) in female mice. A non-observed adverse effect level (NOAEL) of 0.005 pg/kg was identified. An uncertainty factor of 3 was then applied for extrapolation from animals to humans, a factor of 10 for possible human variability and a factor of 0.7 was then used to adjust for bioavailability. In the experiment in mice TCDD was given in oil, rather than their food. As a consequence, it was probably better absorbed than in the process of dietary ingestion.
For intermediate exposure to dioxins the minimum risk level was set at 20 pg/kg/day based on a NOAEL of 0.0007 ug/kg/day for atrophy of the thymus in guinea pigs (again an immunological effect) and liver hepatotoxicity. Uncertainty factors of 3 (for animal to human extrapolation) and 10 (for human variability) were then applied.
For chronic exposure an MRL of 1 pg/kg/day was identified. This was based on a lowest observable adverse effect level (LOAEL) of 0.12 ug/kg/day in monkeys. An uncertainty factor of 3 was applied to LOAEL value because a true threshold was not identified in the monkey study. Further uncertainty factors of 3 and 10 were then introduced to allow for any doubts about extrapolation from monkeys to man and for possible human variability.
In contrast to the approach of other countries, Japan has recently set an ambient air standard of 0.6 pg/m3. Japan also set standards for dioxins in water (1pg/litre) and dioxins in soil (1ug/Kg soil).

e) Other dioxin-like substances

A number of other chemicals in the environment can combine with the Ah-receptor and therefore have the potential to trigger the same effects as those produced by dioxins. However, none of these chemicals bind as avidly and/or persistently as TCDD does.
Among these chemicals are the polychlorinated biphenyls (PCB's). TEFs have been identified for a number of the 209 PCB's congeners. Only some of these congeners have dioxin-like properties. PCBs are found in many wastes and are widely distributed in the environment. They are effectively destroyed by high temperature incineration. There is a very extensive scientific literature on their toxicity (e.g., Bridges and Perry, 1993; WHO 1993). An assessment of the total dioxin exposure of a population group should incorporate an assessment of exposure to dioxin-like substances. One calculation estimates that the inclusion of PCBs will double the total TEQ exposure (WHO, 1998, in press).

III. KEY ISSUES FOR THE HONG KONG GOVERNMENT IN RELATION TO HEALTH RISKS FROM DIOXINS

Evaluation of the current and future risks to the Hong Kong population from exposure to dioxins and the relevance of HK current and future waste disposal policy requires the following specific information:

a) Identification of the principal routes and levels of dioxin exposure

As indicated above, information from other countries demonstrates that **diet** is by far the most important route of dioxin exposure for all but a few industrial workers and individuals who have been exposed through a major incident (eg: Sevaso). This is certain to be the case in Hong Kong as well.
Thus, any evaluation of exposure needs to concentrate particularly on the nature of the HK diet and the extent of foodstuff contamination with dioxins.

This should include consideration of levels of dioxins in HK mothers' breast milk, particularly from women having their first child (milk from this group is likely to have the highest levels of dioxins). Although ambient air represents a very minor route of exposure to dioxins in comparison with food intake, information is also needed on ambient air and urban dust measurements of dioxins expressed as TEQ. Levels of dioxins in drinking water are likely to be extremely low because dioxin has very poor water solubility.

b) Consideration of dioxin contribution from disposal of local waste and other emission sources in Hong Kong

For waste disposal to be set in the context of other sources of dioxin airborne emissions an inventory is required of all the principal sources of dioxins in Hong Kong. I understand that Professor Rappe is addressing this issue. In regards to waste the following data is required:

i) Current levels of dioxins in HK waste of various kinds;
ii) Releases to air from the current systems for waste disposal (to include landfill as well as incinerators);
iii) Modelling of dioxin emissions for the planned means of waste disposal with special attention to incinerators;
iv) Evaluation of dioxin emissions which would occur if other means of disposal than incineration were adopted;
v) Evaluation of other emissions from the various means of waste disposal which are used in HK in order that the dioxin risk is evaluated in the context of the risk form all chemicals which may be emitted.

c) Approach used to deal with information gaps

Since dietary intake is the principal route of exposure, where direct information is limited, data from countries with a broadly comparable diet to that of the general population in HK should be drawn on. Such data must be treated with caution unless:

a) the analytical methodology was properly validated;
b) it is considered in the context that generally dioxin levels in food are reducing progressively;
c) The relevance of the findings to the general population is considered.

d) Benchmarks (standards) recognised by the HK authorities as providing adequate public protection

As explained above, the adequacy of public health protection is normally judged in relation to a standard for dietary intake or total intake. I assume, in the absence of information to the contrary, that HK will follow the WHO values.

IV. COMMENTS ON THE APPROACH USED BY ERM TO CHARACTERISE THE RISKS

The general approach used by ERM is valid. It follows closely approaches widely employed in the UK and elsewhere to identify the risk from dioxins associated with emissions from new plants. In relation to the two documents:

I An assessment of dioxin emissions in HK
II An assessment of dioxin emissions in HK - Draft Health Risk

I understand that Professor Rappe is addressing the general issue of emission sources and how these sources affect ambient air levels.

I have the following comments in relation to the human health aspects in relationship to principal routes of exposure. Three approaches may be used: assessment of actual intake based on information on dietary and other dioxin intake values in Hong Kong with time data derived from other countries with a likely comparable exposure situation; modelling based on ambient air exposure levels in Hong Kong. From the point of view of assessing future impacts, this latter approach is generally favoured.

a) Intake of dioxins via the diet

The main concern is that the consultants have identified:

- There is no information given on the principal components in the diet of the HK population. The diagram provided on p.3 of Report II is, I presume, from Western Country sources. If a realistic assessment is to be made of actual dietary intake the HK authorities need to confirm that these assumptions are broadly correct or provide their own assessment of the typical diet of the HK population. I am particularly concerned that information about consumption of foods of a fatty nature, fish, milk and dairy products is not available. From the figure on p.3, one would have to deduce that milk and dairy products were completely dominant sources of any dioxins, with fish being an insignificant source. However, from my very limited experience of HK I am not sure that this is a valid assumption.
- The nature and amount of the diet of the HK population which is from local sources. From my limited knowledge of HK I assume that in relation to milk and milk products, the percentage from local sources is very low/negligible for the great majority of the HK population. I presume that most of the fruit and vegetables, as well as meat, is also imported. I have no information on sources of fish and shellfish, although I am aware from previous visits to HK that there are several local fish farms. Without this very important information the consultants have had to assume that an individual might consume entirely locally derived produce. This is unrealistic.
- Levels of dioxin in various dietary components. Although the great majority of the HK food is imported, in order to assess the total exposure of the HK population, it is important to have a picture of the dioxin content of the diet. In the absence of such information, the assumption has to be made that the HK diet is typical of that of Western countries - this may or may not be correct.
- No data is provided on the levels of dioxins in the breast milk of HK mothers. This information would be very valuable as an indicator of the actual exposure to dioxins of the Hong Kong population.

This deficiency in the availability of data for Hong Kong rules out the first two approaches cited above.

b) Intake via inhalation

In assessing this, it needs to be re-emphasised that this is a somewhat minor route of exposure to dioxins for most, if not all, individuals in HK. At a maximum, it represents no more than 2 per cent of the total intake of dioxins and probably rather less than this. In contrast to the serious lack of information for food, the data reviewed by the consultants for dioxins in ambient air and for emissions from the CWCT facility is rather good. It is set out in p.21-24 of Report I. This information has been interpreted in my view correctly as indicating:

- The lack of a discernible difference between the local air quality at the two quite separate measurement points (receptors): Central / Western Hong Kong and Tsuen Wan. This may

indicate the general back ground levels of dioxins in Hong Kong. However, ambient air measurements at other sites in Hong Kong would be necessary to confirm this.

- The apparent lack of a significant contribution of the CWTC plant to the background dioxin levels in ambient air. This seems to be confirmed by the generally low levels of dioxins in the emission from the CWTC stack. It is apparent from this data that of the total airborne exposure to dioxins of the residents in Tsuen Wan, only a very small contribution is from the CWTC Plant. Bearing in mind that this route of exposure in any case amounts to less than 2% of an individual's total dioxin content (see above), the contributions of the CWTC plant may be deemed negligible.

c) Urban dust

Urban dust may be contaminated with very low levels of dioxins. Such dusts are normally considered to be a very minor source of human exposure to dioxins, either by inhalation or ingestion. This source is not considered in the ERM report. Young children may be regarded as the most at risk group in respect of dust exposure because of their short height and the greater likelihood of ingesting dust (pica). No measurements of dioxins in dust or soil have been provided. However, it is very unlikely that this will be a significant means of exposure of young children in comparison to dietary intake of dioxins.

d) Uptake of dioxins by various exposures routes (bioavailability)

Dioxins can bind tightly to certain surfaces, e.g.: soil, fly ash, which may interfere substantially with their absorption from the intestine. Bioavailability of dioxins from different foodstuffs is likely to be variable, but there is little reliable data on this subject in the published scientific literature.

e) Modelling of exposure

The consultants have employed an established calculation to estimate the risk from inhalation exposure. Some of the input data for these calculations are not specific to Hong Kong for the reasons identified above. These calculations, however, can be considered to provide a conservative estimate (i.e. a very cautious one) of the likely exposure to dioxins by inhalation of the general population in Hong Kong. For example, it is assumed that 'all of the diet of an individual is produced in the area around an incinerator at which the emissions have their maximum input'.

I agree with the consultant's conclusions that based on the modelling approach they have used 'incremental ground level concentrations of PCDD/F's due to emissions from the WEIF would have to be two to three orders of magnitude greater than those predicted for there to be the potential for a breach of the EPD criteria'.

V. Recommendations

1. Information should be obtained on the dietary composition of the general population in Hong Kong. In particular, some assessment should be made of the diet of pregnant women and very young children.
2. An evaluation needs to be made of the contribution of locally produced raw food ingredients in the Hong Kong diet. It would be helpful to have an indication of the highest percentage of locally produced food consumed in Hong Kong, as well as average figures.
3. Analytical data of dioxin content is required for the following:

a) Imported foods with a substantial fat content where these foods are an important component of the diet in Hong Kong.
b) Dioxin content of selected items of locally grown food
c) Dioxin content in breast milk of a group of pregnant/lactating women.

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