AIR & ENVIRONMENTAL SCIENCES LIMITED

Estimation of exposures associated with 2378-TCDD in soils, and possible current serum residues from those exposures.

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1. INTRODUCTION

Multi-pathway exposure modelling has indicated that emissions from the incinerators that operated at the IWD factory in Paritutu cannot explain the concentrations of 2378-TCDD measured in soils in the vicinity of the plant. This implies that substantially larger releases of 2378-TCDD occurred via other means, such as from process releases or poor housekeeping of materials that may have been highly contaminated with 2378-TCDD. This is most likely to have occurred relatively early in the history of the plant, probably during the 1960s.

Surveys of soil concentrations, including the recent survey undertaken by Pattle Delmore for MfE and ESR (*"Dioxin Concentrations in Residential Soil, Paritutu, New Plymouth")*, indicate that the maximum soil concentrations of 2378 TCDD at residential locations around the plant are about 20 ng/kg. Combination of this soil concentration with multi-pathway modelling allows investigation of the likely range of air concentrations that would have to have persisted over some specified period (for example 10 years) to result in the accumulation of the measured concentrations of 2378-TCDD in soils. These estimated air concentrations of 2378-TCDD in air can then be used to estimate the exposures to which residents in the area at the time might have been subject through a range of exposure pathways.

A toxicokinetic model for 2378-TCDD has been developed and used to estimate the historical pattern of 2378-TCDD intakes that must be invoked to explain the serum concentrations of 2378-TCDD in population groups of varying ages found in the recent serum survey undertaken for MfE. This model also has the ability to estimate the increase above the expected "background" 2378-TCDD concentrations in serum that are likely to persist in the year 2000 from exposures occurring many years ago.

This paper outlines the combination of the estimation of the range of likely exposures for people living at the sites of the maximum measured soil concentrations of 2378-TCDD with the toxicokinetic model to estimate what increases in 2378-TCDD concentrations in their serum may still be measurable in the year 2000.

2. 2378-TCDD DEPOSITION PROCESSES.

Of the dioxin and furan congeners included in toxicity assessments, 2378-TCDD is one of the most volatile, and at typical ambient temperatures about 50% is commonly estimated to be in the vapour phase, with the remainder being bound on fine particulate material. Deposition processes can include:

- Dry deposition of particulate material
- Wet deposition of particulate material by rain
- Vapour deposition, typically by absorption onto organic substrates, such as vegetation cuticles and possibly organic matter in soils.
- Wet deposition of vapour, by absorption of vapour phase 2378-TCDD into rain
- Deposition onto vegetation, such as grass, with subsequent incorporation of the grass residues in soil organic material.

The rates of these processes are not well defined, and it is uncertain whether some of them (for example wet deposition of vapour) make any significant contribution to overall deposition rates. This situation exists partly because, while there have been studies of some of these processes for the full range of polychlorinated dioxin and furan congeners, the concentrations of 2378-TCDD are often at or below detection limits. Accordingly it is difficult to undertake meaningful studies in atmospheres that are not highly contaminated, such as normal ambient air. The deposition processes and rates are better established for the more highly chlorinated dioxins and furans, which are mostly bound on particulates.

The US EPA Human Health Risk Assessment Protocol (1998) includes wet and dry particulate deposition and vapour deposition in its assessment of the accumulation of dioxins and furans in soils from combustor emissions. The algorithms for these estimation are commonly developed from data unrelated to dioxins, such as heavy metal deposition or particulate deposition in general, although some dioxin congener-specific factors, such as the fraction present in the vapour phase, are included in the estimation. These assessment procedures are one of the approaches used to estimate the air concentrations likely to have been associated with the accumulation of 2378-TCDD in soils in the vicinity of the IWD plant.

Direct studies of deposition of dioxins have been undertaken, and these indicate that the typical deposition velocity for dioxins from ambient air is about 0.1-0.2 cm/sec. Effectively, this means that each second, the total dioxin content of a layer of air 0.1-0.2 cm thick is deposited onto the ground or other surfaces. The effective deposition velocity from the Human Health Risk Assessment Protocol is about 0.45 cm/sec, significantly greater than the actual measurements. One of the estimations used for the IWD situation adjusts the Protocol deposition rates to better match the measured deposition rates, taking the deposition velocity as 0.2 cm/sec. However, while the deposition velocity is quite well established for the less volatile dioxins and furans, the deposition velocity for 2378-TCDD is less certain, because of the low concentrations generally present relative to detection limits. It is possible that the 0.1-0.2 cm/sec deposition velocity is associated mainly with dioxins and furans that are predominantly associated with particulate material, and that those dioxins present in substantial proportions in the vapour phase, such as 2378-TCDD may deposit more slowly.

The best validated rates of deposition appear to be those for the uptake of dioxins and furans by grass. There is good experimental evidence that dioxin and furan uptake by grass behaves as if it were entirely vapour uptake, even for the more highly for chlorinated dioxins and furans which are overwhelmingly present in the particulate phase. It appears that this mechanism applies particularly appropriately to the more volatile dioxins and furans such as 2378-TCDD. A deposition model for this process has been published by McLachlan at al, and this is also used to estimate the concentrations of 2378-TCDD that would be required to account for the levels of 2378-TCDD in soil via this mechanism, with incorporation of the grass in the soil as organic matter residues. The effective deposition velocity for this process is about 0.03 cm/sec, so that appreciably higher concentrations of 2378-TCDD in air are required to account for the accumulation of 2378-TCDD in soils than for the other two estimation methods.

The annual grass production was taken to be 10,000 kg/ha dry matter, all of which was considered to be incorporated into the soil.

3. ESTIMATED AIR CONCENTRATIONS AND EXPOSURES.

The recent Pattle Delmore report, *"Dioxin Concentrations in Residential Soil, Paritutu, New Plymouth"* provides concentrations of 2378-TCDD in the top 75 mm of soil and also in soil from 75-150 mm below the soil surface for 6 of the locations sampled. The total 0-150 mm depth of soil sampled at these locations contained between 1.3 and 1.9 times as much 2378-TCDD as the top 0-75 mm, with a mean of 1.7 times. One of these soils sampled at two depths contained 14 ng/kg 2378-TCDD in the top 0-75 mm, while the full 0-150 mm depth contained 1.8 times the amount of 2378-TCDD in the top 0-75 mm only. This indicates that it is reasonable to take the mean increase between the top 0-75 mm only and the 0-150 mm depth as 1.7 times, including those locations where relatively high concentrations were found.

Concentration of 20 ng/kg 2378-TCDD in the top 0-75 mm of soil is a reasonable approximation to the highest concentrations found at residential locations in the Pattle Delmore survey, and this is taken as the basis for estimation of exposures here. Increasing the quantity of 2378-TCDD to account for the full 0-150 mm depth of soil sampled (ie by 1.7 times) is equivalent to considering the soil depth of 0-125 mm to have been at 20 ng/kg 2378-TCDD. This provides the soil mixing depth used in the multi-pathway modelling determination to estimate the concentrations in air required to account for the quantities of 2378-TCDD found in the soil.

It should be noted that there may be significant quantities of 2378-TCDD below the deepest depth sampled below the soil surface, as indicated by the modest decreases in 2378-TCDD concentrations between the 0-75 mm samples and the 75-150 mm samples. Accordingly, the estimates of 2378-TCDD concentrations in air, and consequently exposures via other pathways may be underestimated.

The standard US EPA Human Health Risk Assessment Protocol (HHRAP), as updated in 2000, specifies a 20 mm soil mixing depth for situations where the soil is not actively cultivated. If this were an accurate description of the situation at Paritutu, it would be expected that almost no 2378-TCDD would have been found in the 75-150 mm depth soil samples. It is possible that most of the movement that has evidently occurred from the soil surface, for example as a result of either physical disturbance through the activity of earthworms and other soil biota, or via chemical migration, has occurred during the 30 years since 1970, and that the soil profile of dioxins might have been much more similar to the 20 mm soil mixing depth of the HHRAP over the period when high rates of deposition were actually occurring. This would result in much higher concentrations of 2378-TCDD in the shallow surface soil over the deposition period, which would then result in much greater exposures via soil ingestion and via consumption of home-grown chicken and eggs. In the exposure estimates that follow, the soil mixing depths of both 20 mm and 125 mm are modelled, to cover the range of possibilities.

3.1 EXPOSURES FOR A 125MM SOIL MIXING DEPTH

Table 1 sets out the concentrations of 2378 TCDD estimated to be required in air to account for the accumulation of 2378-TCDD to a level of 20 ng/kg in the top 125 mm of soil over a 10-year period. These estimates are given for the HHRAP, for the HHRAP estimates adjusted

to a deposition velocity of 0.2 cm/sec (Deposition velocity), and for the model describing the uptake of dioxins by grass, with subsequent incorporation of the grass into soil organic matter (McLachlan).

The upper section of Table 1 sets out the exposures that people living where the 2378-TCDD concentrations were such that 2378-TCDD accumulated in the soil to a concentration of 20 ng/kg over 10 years, assuming that they received 100% exposure to each of the pathways considered. This would mean, for example, that they would be breathing air at that location continuously over the 10 years, ingesting soil at typical rates used in the risk assessment, obtaining all of their fruit and vegetables from the location, and raising their own free-range poultry at the location to supply all of their egg and chicken meat consumption.

Table 1. Estimates of air concentrations and associated exposures to 2378-TCDD for 125 mm soil mixing depth

Soil dioxin accumulation scenario		US EPA HHRAP	Deposition velocity	McLachlan
Soil concentration after 10 years (ng/kg)		20	20	20
Concentration in air (fg/m3)		3600	8300	58000
Equivalent deposition velocity (cm/sec)		0.46	0.20	0.03
Pathway	Exposure to pathway	2,3,7,8-TCDD exposure pg/kg-BW/day		
Air	100%	1.0	2.4	17
Soil	100%	0.008	0.008	0.008
Exposed fruit & vegetables	100%	1.0	2.4	157
Protected fruit and vegetables	100%	0.022	0.022	0.022
Root vegetables	100%	0.011	0.011	0.011
Eggs and chicken	100%	0.5	0.5	0.5
Total	100%	2.6	5.3	174
Exposure Scenario 1				
Air	100%	1.0	2.4	17
Soil	100%	0.008	0.008	0.008
Exposed fruit & vegetables	10%	0.10	0.24	15.7
Protected fruit and vegetables	10%	0.002	0.002	0.002
Root vegetables	10%	0.001	0.001	0.001
Eggs and chicken	0%	0.000	0.000	0.000
Total	100%	1.1	2.6	32.2
Exposure Scenario 2				
Air	100%	1.0	2.4	17
Soil	100%	0.008	0.008	0.008
Exposed fruit & vegetables	10%	0.10	0.24	15.7
Protected fruit and vegetables	10%	0.002	0.002	0.002
Root vegetables	10%	0.001	0.001	0.001
Eggs and chicken	10%	0.05	0.05	0.05
Total	100%	1.2	2.7	32.3

The lower portions of Table 1 give the exposure estimates for people only partially exposed to some of the exposure pathways, as for the Scenario 1 and Scenario 2 used in the report "Multi-pathway Assessment of Exposures from Dioxin Releases in the Paritutu Area".

The HHRAP estimates require the lowest 2378-TCDD concentrations in air to account for the 20 ng/kg 2378-TCDD concentration in soil considered here. The exposures via the pathways for which exposures are determined predominantly by the air concentrations (air and exposed fruit and vegetables) are therefore lowest for these estimates. At the other end of the scale, the McLachlan model calculations require the highest 2378-TCDD concentration in air, 16 times higher than for the HHRAP estimates. Associated with this, the exposures via inhalation and consumption of exposed fruit and vegetables are about 16 times higher than for the HHRAP estimates. The estimates for a deposition velocity of 0.2 cm/sec are intermediate between these two extremes.

For those pathways for which exposure is determined predominantly by the accumulating concentrations in soil, all of the different estimation methods give the same exposures, because of the same rate of accumulation of 2378-TCDD in soil in each case. These pathways are soil ingestion, protected fruit and vegetables, root vegetables and eggs and chicken.

3.2 EXPOSURES FOR A 20MM SOIL MIXING DEPTH

Table 2 sets out the exposures resulting from the same air concentrations over the deposition period as in Table 1, but with the 2378-TCDD accumulation restricted to the top 20 mm of soil. The concentration of 2378-TCDD in the top 20 mm of soil (126 ng/kg) is correspondingly higher than the 20 ng/kg averaged over the top 125 mm of soil, as for Table 1. However, the same quantity of 2378-TCDD is present in the top 125 mm of soil in both cases.

The higher concentrations of 2378-TCDD in the surface layer of soil increases the exposures via soil ingestion and consumption of eggs and chicken meat from locally-raised free-range poultry approximately six-fold.

Table 2. Estimates of air concentrations and associated exposures to 2378-TCDD for 20 mm soil mixing depth

Soil concentrations after 10 years 0-125 mm depth			20 ng/kg	
Soil concentrations after 10 years 0-20 mm depth			126 ng/kg	
Soil dioxin accumulation scenario		US EPA HHRAP	Deposition velocity	McLachlan
Concentration in air (fg/m3)		3600	8300	58000
Equivalent deposition velocity (cm/sec)		0.46	0.20	0.03
Pathway	Exposure to pathway	2,3,7,8-TCDD exposure pg/kg-BW/day		
Air	100%	1.0	2.4	17
Soil	100%	0.048	0.048	0.048
Exposed fruit & vegetables	100%	1.0	2.4	157
Protected fruit and vegetables	100%	0.022	0.022	0.022
Root vegetables	100%	0.011	0.011	0.011
Eggs and chicken	100%	3.2	3.2	3.2
Total	100%	5.3	8.0	176
Exposure Scenario 1				
Air	100%	1.0	2.4	17
Soil	100%	0.048	0.048	0.048
Exposed fruit & vegetables	10%	0.10	0.24	15.7
Protected fruit and vegetables	10%	0.002	0.002	0.002
Root vegetables	10%	0.001	0.001	0.001
Eggs and chicken	0%	0.000	0.000	0.000
Total	100%	1.2	2.7	32.3
Exposure Scenario 2				
Air	100%	1.0	2.4	17
Soil	100%	0.048	0.048	0.048
Exposed fruit & vegetables	10%	0.10	0.24	15.7
Protected fruit and vegetables	10%	0.002	0.002	0.002
Root vegetables	10%	0.001	0.001	0.001
Eggs and chicken	10%	0.32	0.32	0.32
Total	100%	1.5	3.0	32.6

3.3 CONTRIBUTIONS VIA EXPOSURE PATHWAYS

For Scenario 1 and Scenario 2, inhalation is the largest exposure route for all three estimation methods, and very much largest exposure route for the HHRAP and the deposition velocity estimates. Inhalation is usually a very small contributor to overall dioxin exposure, with food being the predominant contributor. The large contributions from inhalation in this instance arise from the combination of the high 2378-TCDD levels in air and the absence of meat, dairy products and fish exposure pathways, which are usually major contributors to typical dietary intakes.

For the estimates of blood lipid 2378-TCDD concentrations arising from the estimated exposures, it is assumed that 100% of the inhaled 2378-TCDD is absorbed into the body. This may overestimate exposures.

If people obtained 100% of their exposed fruit and vegetables (those of which the outer surfaces exposed to air are consumed, including for example silverbeet and kale), the fruit and vegetable pathway would contribute about the same level of exposure as the inhalation pathway, based on the HHRAP and deposition velocity estimates. The McLachlan model estimate shows a very much higher contribution from exposed fruit and vegetables, 150 and 66 times higher than the HHRAP and deposition velocity estimates respectively. For this estimate, the exposed fruit and vegetables are considered to take up the same concentrations as taken up by grass exposed to the same level of 2378-TCDD in air. This is almost certain to be an overestimate for the total exposed fruit and vegetable consumption, which includes vegetables such as silverbeet, for which the 2378-TCDD level in grass may be reasonably close, and fruit, such as plums and apples, for which the surface/volume ratio is very much smaller than for grass and for which the grass concentrations would clearly be a gross overestimate. Nevertheless, the very high grass-based estimate for this exposure pathway does indicate that, depending on the vegetable grown in the contaminated air, only modest proportions of the total fruit and vegetable intake may make significant contributions to the overall 2378-TCDD intake.

For Scenario 1 and Scenario 2, fruit and vegetables make only minor contributions to exposure compared with inhalation for the HHRAP and deposition velocity estimates. However, the exposed fruit and vegetables pathway contributes about the same as the inhalation pathway for the McLaren estimates. If this maybe quite reasonable if the 10% of total consumption of fruit and vegetables grown at the exposure site are those likely to show higher concentrations because of high surface/volume ratios, such as silverbeet. This indicates that it may be important to question possible candidates for serum sampling and analysis about both the quantities of home-grown vegetables consumed, and the type of vegetables. Fruit is likely to be a much less important contributor, because of the relatively low surface/volume ratios.

Depending on the soil mixing depth, eggs and chicken are likely to contribute between half and three times the levels of exposure from inhalation for the HHRAP estimates if 100% of the egg and chicken meat consumption are from home-grown poultry. Because the inhalation exposure estimates are higher for the deposition velocity and McLachlan estimates than for the HHRAP estimate while the eggs and chicken exposures are the same for all scenarios, the relative contribution is smaller for the deposition velocity and McLachlan estimates. Typical consumption of eggs and chicken meat are about the same, so if only eggs were consumed, this pathway would contribute about half of that for both chicken and eggs. However, this would depend on how many eggs were actually consumed.

Consumption of eggs and chicken is excluded from Scenario 1. The 10% of total eggs and chicken consumption from the exposure site in Scenario 2 contributes less than 5% of total exposure to any of the exposure estimates for a 125 mm soil mixing depth and between about 1% (Mclachlan estimates) and 20% (HHRAP estimates) for a mixing depth of 20 mm.

4. ESTIMATES OF RESIDUAL 2378-TCDD IN 2000.

The range of exposure estimates set out in Table 1 and Table 2 have been used as additional exposure inputs in the toxicokinetic model developed for 2378-TCDD. For this, the daily exposures from Table 1 and Table 2 were considered to have continued over a 10 year period from 1960-1969. This has been chosen as a likely period when high levels of 2375-TCDD emission from the IWD plant may have occurred. It may be desirable to try to obtain additional information that might better identify the likely period of exposures to high releases from the plant, but this is probably unnecessary at this stage. It seems that obtaining definite information of this type may be difficult, and it may be necessary in any event to work in terms of a range of possible exposure periods and times.

The estimated residual 2378-TCDD concentrations in serum resulting from both typical general population background exposures and from the estimated additional exposures resulting from the releases from the IWD plant are set out in Table 3. The exposure levels selected (1.1, 2.7, 5.3, 8 and 32.6 pg/kg-bw/day) cover the ranges of exposures estimated in Table 1, except that the extreme exposure estimates for 100% exposure to all pathways for the McLachlan estimates (176 pg/kg-bw/day) is omitted because this would certainly be an overestimate.

The additional exposure estimates range from about the 1998 WHO provisional tolerable daily intake of 1 pg TEQ/kg-bw/day to about 10 times the 1998 WHO maximum provisional tolerable daily intake of 4 pg TEQ/kg-bw/day. The estimates of the general population background exposures to 2378 TCDD in the 1960s from the toxicokinetic model are about 0.4 pg/kg-bw/day. The background exposure to all polychlorinated dioxins and furans and to dioxin-like PCBs would increase this background exposure, but has not been estimated. Extension of the model to include other dioxin and furan congeners and PCBs would be necessary for this.

The smallest of the estimated additional exposures (1.1 pg/kg-bw/day) is estimated to result in a 58% increase for males and a 81% increase for females in the concentration of 2378-TCDD in serum in 2000 compared with the 2378-TCDD in serum for the optimised background exposures from the model. The 2378-TCDD level in serum from the optimised background exposures of the model are also closely similar to the measured serum levels from the serum survey.

For the larger estimated additional exposures, the increase in the concentration of 2378-TCDD in serum in 2000 increases in proportion to the exposure level, with the 32.6 pg/kg-bw/day exposure level resulting in 2378-TCDD concentrations about 17-24 times higher than those measured in the serum survey.

Table 3. Estimated residual serum levels of 2378-TCDD from estimated additional exposures

Age Group	MfE (2000) Blood Sample		Optmised Background Function		Background + Additional Intake		% increase	
	Average blood lipid concentrations of 2,3,7,8-TCDD ng/kg lipid							
	Male	Female	Male	Female	Male	Female	Male	Female
	Additional intake 1960-69 (pg/kg-BW/day)					1.1		
15-24yr	1.0	1.1	0.9	1.0	0.9	1.0	0%	0%
25-34yr	1.3	1.6	1.4	1.6	1.4	1.6	1%	1%
35-49yr	1.8	2.2	1.9	2.5	2.3	3.3	21%	34%
50-64yr	2.5	3.6	2.4	3.4	3.8	6.2	58%	81%
	Additional intake 1960-69 (pg/kg-BW/day)					2.7		
15-24yr	1.0	1.1	0.9	1.0	0.9	1.0	0%	0%
25-34yr	1.3	1.6	1.4	1.6	1.4	1.6	2%	3%
35-49yr	1.8	2.2	1.9	2.5	2.9	4.5	52%	83%
50-64yr	2.5	3.6	2.4	3.4	5.8	10.3	142%	200%
	Additional intake 1960-69 (pg/kg-BW/day)					5.3		
15-24yr	1.0	1.1	0.9	1.0	0.9	1.0	0%	0%
25-34yr	1.3	1.6	1.4	1.6	1.5	1.7	4%	6%
35-49yr	1.8	2.2	1.9	2.5	3.8	6.5	101%	162%
50-64yr	2.5	3.6	2.4	3.4	9.1	16.9	278%	392%
	Additional intake 1960-69 (pg/kg-BW/day)					8		
15-24yr	1.0	1.1	0.9	1.0	0.9	1.0	0%	0%
25-34yr	1.3	1.6	1.4	1.6	1.5	1.7	7%	9%
35-49yr	1.8	2.2	1.9	2.5	4.8	8.5	153%	245%
50-64yr	2.5	3.6	2.4	3.4	12.5	23.8	420%	592%
	Additional intake 1960-69 (pg/kg-BW/day)					32.6		
15-24yr	1.0	1.1	0.9	1.0	0.9	1.0	0%	0%
25-34yr	1.3	1.6	1.4	1.6	1.8	2.2	27%	36%
35-49yr	1.8	2.2	1.9	2.5	13.8	27.2	624%	998%
50-64yr	2.5	3.6	2.4	3.4	43.6	86.3	1711%	2413%

5. IS IT LIKELY THAT "EXCESS" 2378-TCDD CAN BE DETECTED IN EXPOSED INDIVIDUALS NOW?

For the lowest estimates of additional 2378-TCDD exposure associated with a 20 ng/kg 2378-TCDD level in 125 mm of soil, the estimated increases in 2378-TCDD in serum are in the range 58-81% of the pool concentrations from the serum survey. The variability information presented in the serum survey report, and also the summary data from Hannah et al (1994) for the levels of dioxins and furans in individuals indicates that individual serum levels of 2378-TCDD are likely to vary by a factor of about 2-4. If the only way of estimating the background level of 2378-TCDD were from the data of the serum survey, this level of the variability between individuals would mean that increases of the expected magnitude from the lower estimates of additional exposure could not be detected with any acceptable level of certainty. However, if the additional exposures were above about 15 pg/kg-bw/day or half the maximum estimated additional exposure, the increases compared with background levels could almost certainly be detected with a high level of certainty.

However, it is likely that the ratios between 2378 TCDD and other dioxin and furan congeners in serum are less variable between individuals than the concentrations of 2378-TCDD. This is because the range of sources contributing to typical background levels of exposure generally contain a wide range of congeners, and the multiple pathways and products contributing to an individual's exposure probably averages out much of the congener ratio variability between different sources of dioxins in the diet and other background exposures. In contrast, the additional 2378-TCDD exposures from releases from the IWD plant are likely to be overwhelmingly 2378-TCDD only. This means that for any individual serum sample, the background contribution to the current 2378-TCDD level can be estimated from the levels of the other dioxin and furan congeners and information about the congener ratios typical of the general population. This approach should correct for factors such as high or low levels of food intake, high or low proportions of high dioxin foods in the diet and high or low levels of body fat for the individual under consideration, which are likely to be the most important factors determining the levels of 2378-TCDD from background sources in the individual's serum.

The variability of congener ratios between individuals not known to be exposed to sources that contain predominantly 2378-TCDD have not yet been assessed, and therefore it is not possible to give a definitive estimate of how small an increase above background levels of 2378-TCDD could be reliably identified. However, it does seem likely that the uncertainties might be reduced to the level where it may be possible to identify an "excess" 2378-TCDD level of about the same magnitude as the background concentrations, or in other words a doubling of the current 2378-TCDD level.

At this level of increase, the additional intake over a 10-year period (about 1.5 pg/kg-bw/day) would be of the same order as the 1998 WHO target for the Provisional Tolerable Daily Intake of 1 pg TEQ/kg-bw/day. Background exposures to both 2378-TCDD, the other dioxin and furan congeners and dioxin-like PCBs will increase the overall levels of exposure at the time of exposure to releases from the plant. However, if there were gross exceedances of the 4 pg TEQ/kg-bw/day maximum Provisional Tolerable Daily Intake, these would have been largely attributable to background intakes, rather than to the 2378-TCDD exposures from releases from the plant. This suggests that the likely ability to detect increases in the levels of

2378-TCDD in serum above those expected from background exposures is probably sufficient to provide a reasonably good indication of a low likelihood of effects attributable to the 2378-TCDD releases from the plant. For higher exposures, with greater likelihood of effects, the certainty of reliable detection increases progressively.

More recently, the FAO/WHO Joint Expert Committee on Food Additives (JECFA) (June 2001), has established a Provisional Tolerable Monthly Intake (PTMI) of 70 pg/kg-bw, equivalent to 2.3 pg/kg-bw/day. From consideration of data from the GEMS/regional diets, the JECFA concludes that:

> *"...the results suggest that a considerable fraction of the population will have long-term mean intake above the PTMI."*

This presumably relates mostly to the European and North American situation rather than New Zealand. The JECFA also states that:

> *"The PTMI is not a limit of toxicity and does not represent a boundary between safe intake and intake associated with a significant increase in body burden or risk. Long-term intakes slightly above the PTMI would not necessarily result in adverse health effects but would erode the safety factor built into the calculations of the PTMI. It is not possible given our current knowledge to define the magnitude and duration of excess intake that would be associated with adverse health effects."*

Accordingly, it is likely that the ability to detect "excess" levels of 2378-TCDD above those resulting from background exposure to dioxins will be sufficient either to give a fairly reliable estimate of the level of that "excess", or to demonstrate at a reasonable level of probability that any "excess" exposure to 2378-TCDD is unlikely to have been at levels likely to have resulted in adverse health effects either over the period of the original exposure or subsequently.

As discussed in the description of the toxicokinetic model, there is some concern about whether the true half-life for elimination of dioxin from the body may be shorter than in the model as used here, particularly for the female estimates. A shorter half-life would result in smaller residual "excess" levels of 2378-TCDD in serum in 2000 from exposures occurring some decades ago and therefore affect the level of past exposure that can be detected.