A Study of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) Exposures in Paritutu, New Zealand

A Report to the New Zealand Ministry of Health

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ERRATA

There is an error in the text on page ii. Third paragraph: *"Specifically, participants with 15 years or more residence time between 1962 and 1987 had mean and geometric mean TCDD levels of 14.6 and 12.4 pg/g lipid respectively (n = 15), compared to age and gender-adjusted backgrounds of 2.4 (mean) and 2.2 (geometric mean) pg/g lipid. Those with less than 15 years exposure during this period had a mean TCDD concentration of 3.2 pg/g lipid (n = 37), compared to an expected mean of 1.5 pg/g lipid for a group of similar age and gender."*

The text should instead read: *"Specifically, participants with 15 years or more residence time between 1962 and 1987 had mean and geometric mean TCDD levels of 14.7 and 12.4 pg/g lipid respectively (n = 14), compared to age and gender-adjusted backgrounds of 2.4 (mean) and 2.2 (geometric mean) pg/g lipid. Those with less than 15 years exposure during this period had a mean TCDD concentration of 3.6 pg/g lipid (n = 38), compared to an expected mean of 1.5 pg/g lipid for a group of similar age and gender."*

Consequently, on page iii. Discussion. The second bullet point: *"...(14.6 pg/g lipid, on average)"* should be: *"...(14.7 pg/g lipid, on average)".*

There is an error in the text on Section 3.2, page 18: *"Of the 37 people who had lived in the area for less than 15 years, from 1962-1987, only one was demonstrably elevated (17.9 pg/g), and the next highest serum result in this group was only moderately elevated at 7.1 pg/g. The mean serum TCDD level in the 37 participants living less than 15 years in this area was 3.2 pg/g. In contrast, those 15 people having lived at least 15 years in the area from 1962-1987 had a mean serum TCDD level of 14.6 pg/g lipid."*

The text should instead read: *"Of the 38 people who had lived in the area for less than 15 years, from 1962-1987, two were demonstrably elevated (17.9 and 14 pg/g). The next highest serum result in this group was only moderately elevated at 7.1 pg/g. The mean serum TCDD level in the 38 participants living less than 15 years in this area was 3.6 pg/g. In contrast, those 14 people having lived at least 15 years in the area from 1962-1987 had a mean serum TCDD level of 14.7 pg/g lipid."*

The line 7 of the Discussion should similarly replace "*14.6 pg/g lipid*" with "*14.7 pg/g lipid*".

On page 14 *"...15 long term residents"* should be *"14 long term residents"*; *"... 31.6 pg/g..."* should be *"....31.1 pg/g..."*, *"...15 individuals..."* should be *"...14 individuals..."* and *"...16.9 pg/g lipid observed..."* should be *"...16.4 pg/g lipid observed...".*

None of the above corrections impact the tables, figures, or conclusions of the report in any way. The lead author apologises for any confusion that might have resulted from these errors.

IMPORTANT - MINISTRY OF HEALTH DISCLAIMER

The data and analyses contained in *A Study of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) Exposures in Paritutu, New Zealand A Report to the New Zealand Ministry of Health* have been supplied to the Ministry of Health by the Institute of Environmental Science and Research Ltd (ESR). The Ministry of Health cannot confirm the accuracy of the data and the analyses, and accepts no liability or responsibility for any acts or omissions, done or omitted in reliance, in whole or in part, on the data or the analyses.

ACKNOWLEDGEMENTS

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GLOSSARY OF TERMS

Term	Description
Air dispersion model	Uses meteorological information and geographical features to estimate how much of a pollutant travels in any given direction, and is deposited at ground level.
Congener	A chemical variant within a family of chemical compounds. Dioxins, furans, and PCBs all have various congeners. TCDD is one congener in the dioxin family.
Detection limit	The amount of chemical, below which, the analytical method cannot provide an accurate measure.
Dioxin	Refers generally to all of the chlorinated dioxin and furan congener families with TCDD-like toxicological properties when calculating a TEQ.
Furan	A family of compounds similar in structure to dioxins, usually associated with combustion processes.
Half-life	This is the amount of time required for half of a chemical to leave the body. For TCDD this forms a range of estimated values depending on age, sex, and body fat composition.
Multipathway exposure assessment	An approach to risk assessment that encompasses environmental exposures from air, food, water, and skin contact for a given individual.
PCB	Polychlorinated biphenyl. A type of chemical associated with heavy industrial uses, such as in transformers. Although certain PCBs have dioxin-like toxicity, they generally have very different routes of entry into the environment from dioxins and furans.
TCDD	Technically, this refers to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin – one of the chlorinated dioxin family, and the specific chemical of interest in the current serum study. This particular dioxin congener is a contaminant in the previously existing herbicide 2,4,5-T. It is the most potent of all the dioxin congeners.
TEQ	Toxic Equivalent: This is the internationally accepted way to express the combined TCDD-like toxic potency of all of the dioxin, furan, and PCB congeners in a sample. In this report we use the World Health Organization definition of TEQ, published by Van den Berg et al. (1998)
Toxicokinetic model	The fate of a toxic chemical once inside the body. Refers in this case to the elimination rate of TCDD.

SUMMARY

Background

In October 2001 the Ministry of Health (MoH) contracted the Institute of Environmental Science & Research (ESR) to investigate non-occupational exposure to dioxins among residents of Paritutu, a suburb of New Plymouth.

The investigation into suspected exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) related to a point source of production of the herbicide 2-4-5,T, namely the Ivon-Watkins Dow [IWD] plant, currently operating as Dow AgroSciences.

Subsequent to community consultation, environmental soil dioxin testing and ethics committee approval, the blood of 52 selected residents was analysed for polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs). Twenty-four of these participants were also analysed for polychlorinated biphenyls (PCBs).

Methods

Individuals were selected for testing based on spatial, toxicokinetic, and multipathway exposure modelling, particularly individuals from different residential periods in order to determine the timing and extent of exposure to airborne emissions of TCDD. The exposure model considered the location and years of residence in relation to various time periods between 1962 and 1987.

Individuals were selected from a pool of 830 respondents to advertisements. Of the 830 current or former residents, 31 were initially selected for testing using the exposure model described above. A total of 24 participants in this first round were able to give blood in February 2004. The group comprised five demographic subgroups of four to six individuals corresponding to the age/sex strata from the Organochlorines Programme (OCP) conducted in 1997. The formation of these subgroups was based on the modelled prediction of individuals most likely to show a statistically significant elevation in serum TCDD, if previous exposure had occurred. Participants with a history of possible occupational exposure were excluded from the study.

A second round of testing was conducted in October 2004, the primary purpose being to ascertain the role of more recent years of relevant residence (over the 1972-86 period) on individual TCDD levels. Twenty-eight participants were selected from the database (excluding those with occupational exposure) based on age, gender and surrogate exposure values using modelled TCDD soil concentration and years of residence.

The results of serum TCDD and other dioxin and PCB congeners from the total 52 selected individuals were subsequently compared with national serum TCDD data from the Ministry for the Environment's national OCP.

A quality control sample was tested at the US Center for Disease Control and Prevention in Atlanta, Georgia, in addition to repeat samples run at the Axys Analytical Services, Vancouver, Canada.

Results

A statistically significant elevation in serum TCDD compared to national TCDD serum concentrations was found in the combined study group of 52 participants. The range of individual TCDD concentrations was 0.85 – 33.3 pg/g lipid, representing a range from no increase above background in 6 of 52 people, to a greater than 15-fold increase in TCDD in the highest individual. The mean and geometric mean TCDD concentrations across all 52 participants were 6.5 and 4.0 pg/g lipid, compared to the overall expected mean and geometric mean of 1.7 and 1.5 pg/g lipid, respectively. Expected values were calculated based on the age and gender of the study group.

TCDD exposure to residents is likely to have been the result of gradual accumulation over a long period of time, as duration of residence was the key factor in determining the likelihood of measuring an increase in serum TCDD. Specifically, participants with 15 years or more residence between 1962 and 1987 had mean and geometric mean TCDD levels of 14.6 and 12.4 pg/g lipid respectively (n = 15), compared to expected age and gender-adjusted backgrounds of 2.4 (mean) and 2.2 (geometric mean) pg/g lipid. Those with less than 15 years exposure during this period had a mean TCDD concentration of 3.2 pg/g lipid (n = 37), compared to an expected mean of 1.5 pg/g lipid for a group of similar age and gender.

On average, TCDD made up 35% of the test participants' total dioxin toxic equivalents (TEQ) using the WHO TEQ calculation, which is approximately double the proportion observed in the 1997 national serum study and higher than that seen in other studies overseas. Although there was a significant elevation in serum TCDD among participants, the elevation in TEQ was less pronounced, and not statistically significant when compared on an age-group basis. There was no elevation in PCB compared with expected background for the first 24 individuals tested. Analysis for PCBs was dropped from the subsequent round of testing. The average elevation in TEQ was 1.2-fold for all 52 individuals, but the increase was 2-fold for participants living in the area for more than 15 years during the 1962-1987 period. TCDD was the only consistently elevated compound in sera analyses. Subtracting the contribution of TCDD to TEQ resulted in no significant differences between study participants and background results.

Consumption of home-grown produce of a specific nature appeared to contribute significantly to elevations in serum TCDD. This included leafy vegetables, apples, pears, and any food with a surface exposed to the atmosphere that is then consumed. There was no indication of a significant contribution to exposure from root vegetables, protected fruits (citrus, feijoas, etc), poultry or eggs, or kai moana (seafood).

Discussion

These findings support the premise that historical aerial emissions containing TCDD are responsible for the soil and serum dioxin concentrations in Paritutu. Observed chemical profiles of dioxin and its congeners in the Paritutu environment, its residents and the measured TCDD elevations are most likely to be the result of fugitive emissions and not a result of combustion processes, such as incineration. Evidence for exposure was observed throughout the production years (1962-1987). Whether these emissions were a result of regular, or more episodic releases cannot be determined by the current study.

The multipathway exposure modelling, in particular, duration and time of residence, predicted elevations in serum TCDD with statistical significance, whereas soil TCDD concentrations alone did not.

Based on the current data, there appear to be a number of findings of particular relevance to assessing the nature of exposure to dioxins in Paritutu:

- Elevations in serum TCDD reflect primarily duration of residence over the period 1962 – 1987 in areas of modelled soil TCDD in excess of 3.4 pg/g.
- Participants residing in the area for more than 15 years between 1962-1987 exhibited marked elevations in TCDD (14.6 pg/g lipid, on average) compared to expected background levels (2.4 pg/g lipid).
- Observed elevations are, in all probability, mainly due to inhalation exposures from aerial emissions originating from the IWD plant.
- Some contribution from consumption of fruits and vegetables exposed to the local atmosphere is apparent.
- Present soil contamination is not likely to be the source of the observed levels, nor is it likely to represent a significant source of ongoing exposure.
- The elevation in dioxin TEQ among all participants was not statistically different from 1997 background levels (1.2-times greater, on average).
- Elevations in TEQ were twice that of background in participants who lived in the area for more than 15 years between 1962-1987.
- There was evidence of exposure to TCDD both pre- and post-1974, but no clearly demarcated exposure periods within the overall 25-year 2,4,5-T production period (i.e. 1962-1987) were evident.

The following questions remain unanswered by the study:

- The temporal variation in exposures during the period 1962 to 1987.
- Serum TCDD levels in individuals who resided in areas where soil TCDD exceeded those in this study.
- Whether there was a contribution to TCDD exposure from production of chlorinated phenolic products other than 2,4,5-T.
- The potential health impact in people significantly exposed.
- The potential exposure of residents not included in this study.
- The amount of exposure to workers at the IWD plant

1. INTRODUCTION AND BACKGROUND

In October 2001 the Ministry of Health (MoH) contracted the Institute of Environmental Science & Research (ESR) to investigate non-occupational exposure to dioxins among residents of Paritutu, a suburb of New Plymouth. ESR conducted the investigation in three phases.

An initial consultation phase (Phase I, see Appendix A) took place between October 2001 and May 2002, resulting in majority agreement of the community consultation group as to the next phase (Phase II), which included:

Phase II Part I (May 2002 – May 2004)

- seeking and obtaining consent from the appropriate ethics committee;
- administration of questionnaires to current and former residents who met inclusion criteria;
- identification of a possible high exposure group through the use of a multipathway exposure model;
- discussion and informed consent to participation both for the questionnaire and blood testing;
- taking of venous blood from selected individuals;
- analysis of the blood samples for the congeners of dioxin of human toxicological significance, and comparison with serum levels of the wider NZ population; and
- dissemination of individual, group and comparative results.

Phase II Part II (May 2004 - January 2005)

- obtaining a total of 50 samples to complete the original study plan
- identification of individuals with residence times that could assist with answering questions about temporal variation in exposure, especially residence times post 1973
- participants selected on the basis of age, gender and timing of exposure

Methods for addressing these issues, in concert with findings of the study, are addressed in this report.

It should be noted that the purpose of this study was to assess only the potential exposures to dioxins in the community through measuring blood levels of dioxin. This report does not include any assessment of possible health effects related to measured dioxin.

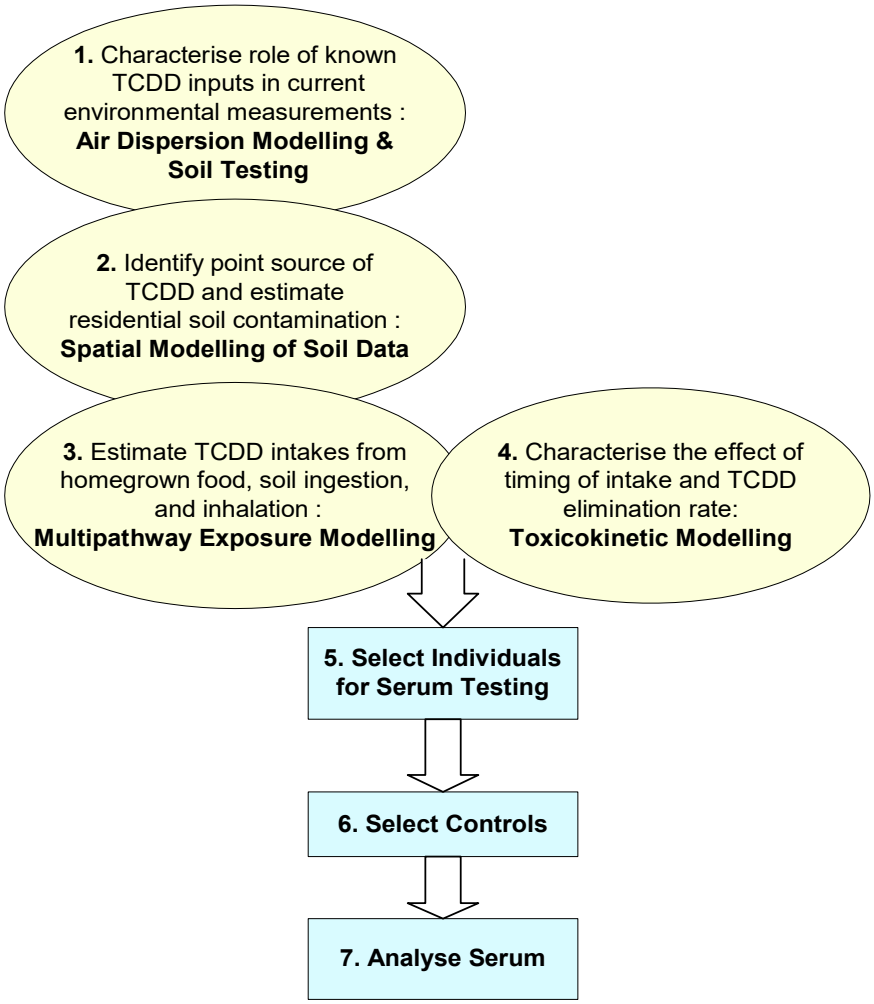
1.1. Ethical Approval

Prior to commencing the project, ethical approval was sought from the Taranaki Regional Ethics Committee. Approval for the study was granted, reference TRK/03/05/014.

2. METHODOLOGY

A number of modelling exercises were conducted to characterise the Paritutu environment, taking account of: Published reports of TCDD emissions from the IWD plant, construct exposure/uptake scenarios of inhalation and dietary intake at each address, and individual variations in TCDD elimination rate. These processes are depicted in Figure 1.

Figure 1. Sequence of modelling studies in the estimation of individual dioxin exposures in Paritutu, used as a basis for selection of study participants.



Data from two questionnaires from Paritutu residents were used to populate these models as tools to select participants. All details of model development and use are presented in a series of technical appendices. The general approach to each aspect of the study is described below for key areas:

2.1. Air Dispersion and Deposition Model (see Appendix B)

Objective: To ascertain the potential role of the IWD liquid and solid waste incinerators, in the observed soil TCDD concentrations.

Inputs/Assumptions: Incinerator parameters (stack height, location, temperatures, etc) and emissions data obtained from Pilgrim 1986, and DSIR 1986. Meteorological data from National Institute of Water and Atmospheric Research (1999) was used in the Air Pollution Model (TAPM) model.

Method: The Air Pollution Model (TAPM) developed by the Australian CSIRO was used to develop a meteorological dispersion modelling data set for the Paritutu area (http://www.dar.csiro.au/pollution/localscale/sld018.htm). The USEPA ISC3 air dispersion model was used to estimate ground concentrations and deposition rates of dioxins from the IWD point source (http://www.epa.gov/scram001/tt22.htm).

2.2. Spatial Model (see Appendix C)

Objective: To estimate TCDD concentrations in soils in the Paritutu area based on measured soil TCDD data.

Inputs/Assumptions: Soil TCDD test results from the Pattle Delamore Partners, Ltd. report to the Ministry for the Environment (PDP 2002). For the modelling, included were 34 data points from the PDP report, and 39 data points from sampling conducted in

- 1985 (Department of Health and IWD);
- 1986 (Ministry of Health); and
- 1997 (Ministry for the Environment).

A 25-year half-life correction was applied to the earlier samples to bring them to approximate 2002 levels for the combined map. In all, 73 measured soil TCDD values served as inputs to the model.

Method: ArcView Geospatial Analyst software was used to conduct Ordinary Kriging of all of the available measured TCDD soil data. The spatial model assisted in the identification of the point source, as well as defining the general area of interest for sampling.

2.3. Multipathway Exposure Model (see Appendices B and D)

Objective: To estimate TCDD exposures of residents in Paritutu from:

a) Inhalation of TCDD in air;
b) TCDD in food from home gardens and poultry; and
c) Possible ingestion of soil contaminated with TCDD.

Inputs/Assumptions: In the assessment the possible intake routes through which residents may have been exposed included:

- Inhalation of particulate and gas phase dioxins;
- Ingestion of contaminated soil;
- Ingestion of below-ground vegetables (e.g. potatoes, carrots);
- Ingestion of 'protected' above-ground vegetables and fruits (e.g. sweet corn, citrus, nuts);

- Ingestion of 'exposed' above ground vegetables and fruits (e.g. lettuce, apples); and
- Ingestion of home-grown poultry and eggs.

Produce is defined as either 'protected' or 'exposed' depending upon whether the edible proportion of the fruit or vegetable is likely to have been exposed directly to dioxin congeners either through direct deposition from the air or via vapour uptake by the plant's foliage. For instance, fruits such as oranges whose skins are not generally consumed are classified as 'protected'. The major route of contamination for 'protected' and below ground produce is via root uptake of contaminants present in the soil. As it is possible that some residents could have kept poultry for eggs or (less likely) meat, the additional intakes of dioxins associated with these pathways have also been considered in the assessment.

Total dietary intakes of eggs and poultry are based upon the estimates used in the OCP, for which the fat intakes are the same as those from the New Zealand National Nutrition Survey and similar to USEPA estimates. In the calculations it is assumed that the typical fat content of eggs is 11.2% and chicken meat 8.4%.

The MfE OCP assessment of dietary intakes for dioxins and dioxin-like PCBs was used based on diets selected to be representative of the adult New Zealand male population (Buckland et al., 2001). Dietary exposure calculations have been based on a 'typical' 80 kg adult New Zealand male, due to the relatively larger intakes of males.

The typical air inhalation rate of $20m^3$/day used is the value recommended by the USEPA (USEPA 1998) for an adult male. The intake of soil used (25 mg/day for an adult), is the same as that used by the MfE in "Health and Environmental Guidelines for Selected Timber Treatment Chemicals" (MoH, MfE, 1997).

Intakes were calculated assuming that the average resident would be potentially exposed to contaminated soil, produce and air for 350 days in a typical year. The resident is assumed to have been away from the immediate vicinity for the other 15 days and, therefore, not exposed to media contaminated by the plant. This assumption is consistent with the USEPA risk assessment methodology (USEPA 1998b).

Appendices B and D detail the methodology and calculations used to derive estimated TCDD intakes. Briefly, the intake scenarios assumed that a typical resident obtained 10% of their daily fruit and vegetables, and chicken and egg intakes from their place of residence. Therefore, 10% of typical dietary produce and poultry intakes was assumed to be contaminated by dioxin emissions. The calculations approximated exposures for a person who spends most of their day at home (ie. 100% of soil ingestion and air intake was from the home environment). A summary of intake rates used in the multipathway exposure analysis for the study of the incinerator emissions is presented in Appendix D.

Method: The USEPA Human Health Risk Assessment Protocol (1998b) (HHRAP) was followed in the multipathway exposure modeling. Exposed and protected above ground produce consumption rates are also based upon the HHRAP

recommendations. The HHRAP is based on data from the Exposure Factors Handbook (US EPA, 1997). The below-ground produce intake is taken from "Health and Environmental Guidelines for Selected Timber Treatment Chemicals" (MoH, MfE, 1997).

The estimation of airborne TCDD required to result in the measured soil TCDD concentrations was done using two models: USEPA (1998a) and McLachlan (1997) models were used to form a range of predicted inhalation rate scenarios and corresponding serum TCDD concentrations. These two models employ different assumptions regarding TCDD deposition rates into soil organic matter, and, therefore, provide differing air concentrations. These different predictions translated into a range of predicted inhalation exposures for the residents, and a corresponding range of modelled serum TCDD concentrations. The McLachlan model resulted in the best predictions of actual measured serum TCDD.

2.4 Toxicokinetic Model (see Appendices E, F and P)

Objective: To estimate age/gender-specific TCDD elimination half-life rates based on analysis of existing data, in order to assist in selecting individuals most likely to show elevated TCDD in 2004 from a past exposure, to assist in any back-calculations of original exposure and body burdens. The model predicts the TCDD body burden for New Zealanders in the years 1997 and 2000, based upon estimated historical TCDD intakes and changing body composition over an individual's lifetime.

Inputs/Assumptions: To be effective, the toxicokinetic model required an estimate of the time of exposure. We assumed initially that the key period for individual exposures to TCDD releases was most likely to be the period from 1962 to 1975. For Phase II Part II we focused on the period after 1973 as there were indications from Phase II Part I that this later period was influential in determining serum TCDD levels. The annual variation in 2,4,5-T production volumes is shown in Figure 2. TCDD emissions between 1962 and 1975 were assumed to vary in proportion to the annual 2,4,5-T production rates.

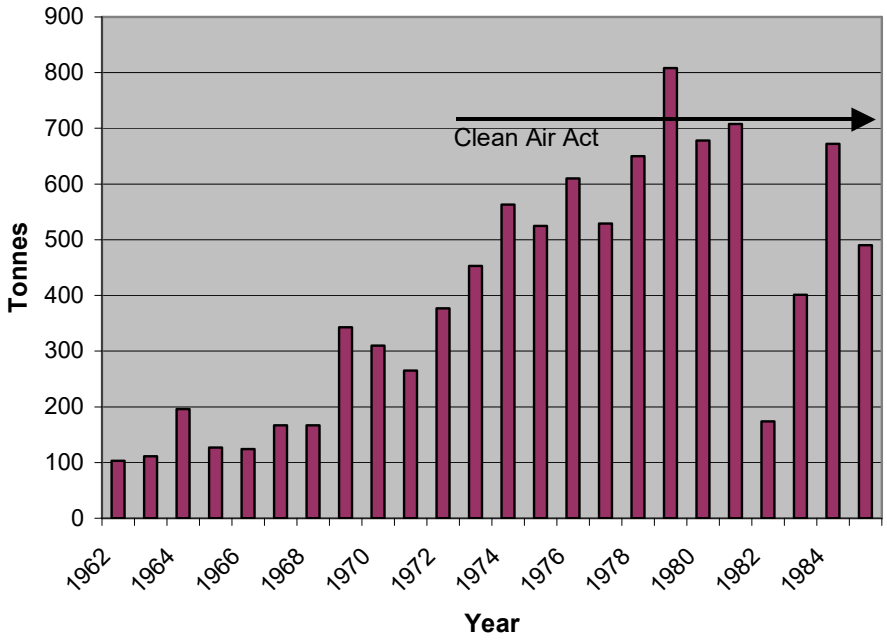
Additional elements in the calculation process are presented in detail in Appendix E and P. There are three major elements used to estimate TCDD body burden at the end of a simulated year:

1. Estimated amount of TCDD in the individual's body at the end of the previous year;

2. Elimination rate of TCDD, defined in terms of a half-life and assumed to be a function of the total percentage lipid content of the body (see peer reviewer comments in Appendix F); and

3. Intake rate of TCDD from contaminated environmental media such as food, air, and soil.

Figure 2. Annual 2, 4, 5-T herbicide production volumes at the IWD chemical plant.



Age groupings used in the model correspond to those in the OCP study to allow population of the model. In order to populate the model with the OCP serum results, we assumed that the observed TCDD blood lipid concentrations reflect the average TCDD concentration in the total body lipid (as predicted by the toxicokinetic model). In the absence of data to suggest otherwise, we have assumed that the elimination rate does not change for individuals beyond age 74.

Method: Dioxin body burdens are calculated on a year-by-year basis, accounting for variations in dietary and inhalation intakes, body weight and body fat. Profiles of male and female body compositions and dietary intakes are constructed for 'typical' New Zealanders aged between 1 and 90 years. These profiles are used to predict present TCDD blood lipid levels (picograms TEQ per gram lipid weight) based on assumed environmental media intake rates, TCDD half-lives in the body (based on total body fat), and the dilution of total TCDD body burden in total body fat.

The initial modelling was used to estimate a 'background intake' function which estimates relative changes in the concentration of TCDD in the New Zealand diet between 1937 and the year 1997 (see Appendix E). The background intake function focuses on picograms (pg) of TCDD per day, per megajoule of food ingested. The model assumes that the body absorbs all of the TCDD ingested (100%). Using any other absorption rate would proportionally increase the TCDD concentration per megajoule ingested by the inverse of that absorption rate (ie 1 / TCDD absorption rate).

The model was subsequently further developed and adapted to predict the expected incremental increase in individual blood lipid levels above background TCDD exposures for the year 2000 based upon their estimated historical exposure to TCDD contaminated environmental media both during and after the assumed emission period (see Appendix P). The estimated level of TCDD contamination in the modelled environmental media was calculated on a year-by-year basis using the integrated multipathway exposure model.

A gender-specific profile describing typical dietary intakes, body weight and percentage total body fat over an individual's lifecycle was constructed using national and international data. Each individual is assumed to follow the same life history regarding dietary intakes and body composition. An individual's body and intake characteristics are assumed to be constant over each year that is modelled. Though these assumptions are crude, they allowed us to make an initial screening/prioritisation ranking of individual participants so that objective decisions could be made regarding individual selections for serum testing.

2.5 Selection of Candidates for Serum Testing, Phase II

2.51 Part I

Part I Objective: To use predictions of individual TCDD intake, combined with estimated age/sex specific TCDD elimination rates, to derive a list of individuals having the best chance of showing any possible elevations in serum TCDD in 2004 from potential exposures beginning from 1962, in comparison with national averages and estimated variances for the individual age/sex strata.

Part I Inputs/Assumptions: Changes in body weight and body fat percentages for each of the participants were assumed to be comparable to the age dependent profiles developed for male and female New Zealanders. It was also assumed that all participants were exposed through non-occupational means, although this was not independently verified.

Two questionnaires were developed to provide input to the integrated multipathway and toxicokinetic modelling (See Appendices L, M and P). Questionnaire One is more pertinent to the selection process and provides data for the geo-spatial and multipathway exposure modelling. Questionnaire Two provides more detailed data relevant to the multipathway exposure and toxicokinetic modelling of the half-life of TCDD in the body. Questionnaire Two also provided information on some possible exclusion criteria, such as previous employment at the IWD plant, history of extensive use of herbicides, etc. These data assist with interpreting and explaining individual results, particularly the ratio of TCDD to total TEQ.

Part I Method: The sum of residential inhalation and dietary intake exposures, based on modelled air concentrations of TCDD as described in the multipathway exposure model (above). Subsequently, age/sex specific elimination rates were applied (see toxicokinetic model above), based on the assumed TCDD emission period between 1962-1975. Relative TCDD emission rates over the period corresponded to the reported annual production of 2,4,5-T herbicide at the IWD plant.

2.52 Part II

Part II Objective: A second round of testing was conducted in October 2004. The four participants who were unable to give blood in Part I were invited to give blood in Part II. New participants were selected from the database, using the same exclusion criteria as described in Part I. The objective of Part II was to select participants from the younger age groups with greatest exposure during the later years of plant operation. People with exposure in 1973 or earlier were excluded from Part II sampling.

Part II Inputs/Assumptions: A surrogate measure of exposure was estimated based on the years of residence at previously determined TCDD soil concentrations according to MfE soil sampling and subsequent kriging of the residential area grid (see figure 4, next section). The assumption was that soil concentrations were a valid estimate of historical exposure to fugitive emissions.

Part II Method: Surrogate exposure values for candidates were calculated for each participant using the following formula:

- Soil TCDD (ppt) at location * years at that location (between 1974 and 1987) = exposure surrogate value (ppt-years)

The soil TCDD ppt equals the modelled concentration based on the MfE soil study described above, at residential addresses previously geocoded according to x,y coordinates.

Note - for participants who lived at multiple locations within the study area, exposures were considered additive and summed together.

Selection criteria for Part II blood sampling;

a.) Participants from Phase II who were unable to give blood in previous sampling and wanted to participate in Part II
b.) Minimum exposure of 40 ppt-years (males) and 60 ppt-years (females). The relative difference between male and female minimum exposure values reflected the relative scarcity of males enrolled in the study population.
c.) Candidates in 1997 in the age-categories of 19-24, 25-34, 35-49 and 50-64 to supplement data gathered in Part I and to maximize the chance of detecting significant differences from age-adjusted background. (The variability in background TCDD blood lipid levels increases significantly after age 65).
d.) Those with residential exposure beginning in 1974 or later.

A total of 28 candidates with the highest exposure estimates were selected for sampling in Part II using the above sampling criteria, four of whom were re-invited from Part I.

2.6 Responses to the Questionnaires

A total of 830 questionnaires were sent out (Appendix L), as a number of people had requested questionnaires for partners and immediate family. A letter was sent with the questionnaires requesting the return of the completed questionnaire and consent form

to participate in the study by the 30th September 2003. A reminder letter was sent on the 25th September 2003.

Of the 830 questionnaires and information packets initially mailed out, 377 questionnaires were returned, giving a response rate of 45%. Fifteen declined to participate, and 438 remained outstanding, despite being sent reminder letters. Of the 377 returned questionnaires, 146 people were selected and sent the Questionnaire 2 package (see Appendix M) that included a consent form for giving blood. At this time the 231 people not selected were informed of this in writing.

2.7 The Group Selected for Testing

Blood was taken from 24 participants on the 23rd-27th February 2004, and from 28 participants on the 11th-15th October 2004. Several individuals were not given consent to participate by their GP for health reasons on either occasion; and, on the day of collection, three people had a haemoglobin level below NZ Blood service guidelines (Hb < 110 *g/l* using a Hemocue machine). These individuals (who could not give blood) included the female with the highest predicted TCDD level in the study (aged 65+), and the male with the highest predicted TCDD level (aged 50-64).

The average age of the 30 women and 22 men who gave blood was 58 years in 2004. Further statistics on the subgroups included for testing are described in Table 1:

Table 1. Overview of the Paritutu study subject selection process

	N
People registering an interest (before advertising)	151
Total people registering an interest (after advertising)	809#
Information packs and questionnaires mailed out *(Questionnaire 1)*	831
Questionnaire 1 returned	379
Modelling, initial selection, (*sent Questionnaire 2*)	146
Questionnaire 2 returned	134
Modelling, ranking and selection	58
Blood collected and tested	52

includes original 151.

It should be noted that the initial groups of 831 and 379 people included numerous individuals who were found not to have ever lived in the New Plymouth area, and many who lived in New Plymouth but never in the vicinity of Paritutu. Thus the response rate appears low, but in fact the rate was high among people who actually lived in the area (> 90%).

Each individual gave 120 - 200 mL blood, which was clotted for 1 hour, and then centrifuged at the hospital and serum immediately separated and stored at –20C. Blood was collected over consecutive days for both Parts I and II, over up to five days.

2.8 Selection of Controls and Statistical Comparisons (see Appendices G, H, and J)

Objective: To select a control group for the comparison of Paritutu resident TCDD serum dioxin concentrations.

Inputs/Assumptions: The national OCP study was conducted in 1997, representing a large number of New Zealanders grouped into pooled substrata. This information was assessed, and national, rather than regional (lower North Island), means and variances were selected for use since it was felt that these were a more robust measure for comparison (larger sample numbers minimises any effect that New Plymouth samples might have on pooled substrata). There is unanimous agreement among the scientists consulted in this project that the 1997 MfE background TCDD values overestimate what would be expected in 2004, due to declining TCDD intakes in the food supply. For this reason, we extrapolated expected background TCDD values from 1997 to 2004 for all age and gender groups, using the toxicokinetic model.

Method: Appendices G and H describe the statistical issues surrounding the estimation of variance from pooled substrata and the use of additional NZ-specific control data (Hannah et al., 1994). Means and estimated 95% confidence intervals for each stratum are also shown in Appendix G.

In the 1997 MfE survey, due to the relatively small volumes of blood collected from participants (compared to volumes needed for testing), blood samples were pooled into larger sample units. Each sample was pooled in one of 80 strata used to categorise the sample population. Each stratum was defined with respect to gender, ethnicity, age, and locality. Individuals who were likely to have been occupationally exposed to organochlorines were excluded from the blood pooling. Each individual contributed an equal volume to the total pooled blood volume.

The optimal age/gender make up of the participants in Part I was determined based on the best statistical chance of identifying elevations in TCDD compared with appropriate subgroups from the 1997 MfE survey, extrapolated to expected current day values. In Part II the criterion of residence post-1973 was added.

The primary basis for the modelled serum TCDD was the amount of time an individual spent at an address and the estimated average air concentration and modelled soil TCDD concentrations at that address. Additional factors were considered, including intakes of home produce and poultry/eggs at the address.

2.9 Serum Analyses

Sera were analysed for all seven of the 2,3,7,8-substituted chlorinated dioxins, ten furans, four coplanar PCBs and 8 mono-ortho chlorinated PCBs. For the first 24 participants, the ten coplanar and mono-ortho PCBs thought to contribute to dioxin-like activity were also included in the analyses. As PCBs were not systematically elevated in the 24 participants, they were not included in the analysis in the second group of 28. The list of dioxin and PCB congeners tested for is shown in Appendix N.

All sera samples of 120 – 200 ml were sent in sealed insulated containers via Federal Express courier to the Axys Analytical Services laboratory in Sydney, BC, Canada for testing. The Axys Analytical Services met WHO criteria for chlorinated dioxins and PCB measurements in human blood (Appendix I). Three quality control samples were sent either to the US Centers for Disease Control in Atlanta, Georgia, USA or to the Axys Analytical Services. One sample was split between Axys and USCDC, while two others were repeat blind samples sent to Axys in the second round of testing. QC results were within normal interlaboratory variation between Axys and USCDC, and the repeated samples were very consistent (less than 20% variance) between the two sampling time points.

The Axys laboratory used high-resolution gas chromatography coupled with high-resolution mass spectrometry to analyse for the full spectrum of chlorinated dioxins and furans and PCBs relevant to characterising an individual dioxin TEQ according to the WHO 1998 TEF scheme. Detection limits for TCDD were typically 0.1 pg/g lipid. Serum lipids were measured by a sub-contracting laboratory in Canada, and results were very closely matched (> 95% concordance) to that by USCDC.

It was not possible to calculate serum lipid concentrations standardised by age and gender due to the small number of observations in each category.

3. RESULTS

3.1. Serum TCDD Concentrations

The serum TCDD concentrations for Part I, Part II, and the combined group of 52 individuals are shown in Tables 2-4. In comparison to estimated national background levels in 2004 (Table 5), TCDD was seen to be elevated in all age groups and in both genders in the study. Across the group, the arithmetic mean value was 6.5 pg TCDD/g lipid, compared with an expected mean TCDD concentration in a similar group in 2004 of 1.7 pg/g lipid, or an increase of 3.8-fold.

Table 6 shows dioxin and PCB TEQ results, using TEFs from WHO (WHO 1998). The TEQ was not significantly elevated overall across the 52 participants in the study (16.6 pg/g lipid observed vs 13.7 pg/g lipid expected). However, for the 15 long-term residents (greater than 15 years residence between 1962 and 1987), the TEQ was significantly elevated two-fold compared to 1997 values for a group of similar age (TEQ = 31.6 pg/g lipid observed vs 16.9 pg/g lipid expected). The TEQ elevations in this group of 15 individuals became non-significant when TCDD was subtracted from the total TEQ (TEQ = 16.9 pg/g lipid observed vs 13.3 pg/g lipid expected). Therefore, across all groups TCDD is the major driving factor in total dioxin TEQ differences from national mean values.

Table 2: Mean serum TCDD levels: Part I

Age group	Paritutu Sample size (Part I)	TCDD (pg/g lipid)	
		Arithmetic Mean [95% CI]	Geometric Mean
Male			
35-49	1	**1.3**	**1.3**
50-64	6	**9.8** [1.3 - 18.3]	**7.5**
64+	4	**14.6** [0 - 35.4]	**10.9**
Subtotal	11	**10.8** [0.8 – 20.8]	
Female			
35-49	5	**6.2** [0.6 - 11.8]	**5.1**
50-64	4	**7.1** [0 - 14.4]	**5.9**
64+	4	**17.8** [5.0 - 30.6]	**16.2**
Subtotal	13	**10.0** [2.5 – 17.6]	
All ages	**24**	**10.4** [6.9 - 13.8]	**7.5**

95% CI = lower and upper 95% confidence interval around the mean.

Table 3: Mean serum TCDD levels: Part II

Age group	Paritutu Sample size (Part II)	TCDD (pg/g lipid)	
		Arithmetic Mean [95% CI]	Geometric Mean
Male			
25-34	2	**1.7** [0.7 – 2.7]	**1.6**
35-49	2	**2.1** [1.7 – 2.5]	**2.1**
50-64	6	**2.6** [2.0 – 3.2]	**2.5**
64+	1	**11.8**	**11.8**
Subtotal	11		
Female			
19-24	4	**1.7** [1.1 – 2.3]	**1.6**
25-34	4	**1.3** [1.1 – 1.6]	**1.3**
35-49	2	**3.3** [0 – 6.7]	**2.9**
50-64	7	**5.7** [1.7 – 9.7]	**4.6**
64+	-		
Subtotal	17		
All ages	**28**	**3.2** [1.6 – 5.0]	**2.5**

95% CI = lower and upper 95% confidence interval around the mean.

Table 4: Mean serum TCDD levels: all samples (N=52)

Age group	Paritutu Sample size (all samples)	TCDD (pg/g lipid)	
		Arithmetic Mean [95% CI]	Geometric Mean
Male			
25-34	2	**1.7** [0.7 – 2.7]	**1.6**
35-49	3	**1.9** [1.3 – 2.5]	**1.8**
50-64	12	**6.1** [2.3 – 10.0]	**4.3**
64+	5	**14.0** [4.1 – 24.0]	**11.1**
Subtotal	22	**6.9** [3.5 – 10.3]	**4.3**
Female			
19-24	4	**1.4** [0.8 – 2.1]	**1.6**
25-34	4	**1.3** [1.0 – 1.6]	**1.3**
35-49	7	**5.3** [2.3 – 8.3]	**4.3**
50-64	11	**6.0** [3.1 – 8.9]	**5.0**
64+	4	**17.8** [9.9 – 25.7]	**16.2**
Subtotal	30	**6.2** [3.8 – 8.6]	**4.1**
All ages	**52**	**6.5** [4.6 – 8.6]	**4.2**

95% CI = lower and upper 95% confidence interval around the mean.

Table 5: Background mean serum TCDD levels: MfE samples and projected concentrations in 2004.

Age group	Sample size (MfE)	MfE TCDD in 1997 (pg/g lipid) Mean [95% CI]	Projected mean TCDD in 2004 (pg/g lipid) Mean [95% CI]
Male			
25-34	145	**1.2** [1.1-1.4]	**0.6** [0.5-0.7]
35-49	199	**1.8** [1.6-2.0]	**1.1** [1.0-1.2]
50-64	170	**2.5** [2.3-2.7]	**1.5** [1.4-1.7]
64+	139	**3.0** [2.8-3.3]	**1.9** [1.7-2.1]
Female			
15-24	114	**1.1** [1.0-1.2]	**0.6** [0.5-0.7]
25-34	224	**1.5** [1.3-1.7]	**0.9** [0.8-1.1]
35-49	368	**2.1** [1.9-2.4]	**1.4** [1.3-1.6]
50-64	255	**3.6** [2.8-4.3]	**2.4** [1.9-2.8]
64+	242	**5.9** [5.1-6.7]	**4.1** [3.5-4.6]
Expected mean for this study group		**2.7** [2.4 – 3.0]	**1.7** [1.5 – 1.9]

95% CI = lower and upper 95% confidence interval around the mean.

Table 6. Mean serum total dioxin and PCB TEQ: All Paritutu participants and Ministry for the Environment 1997 Organochlorines Programme survey.

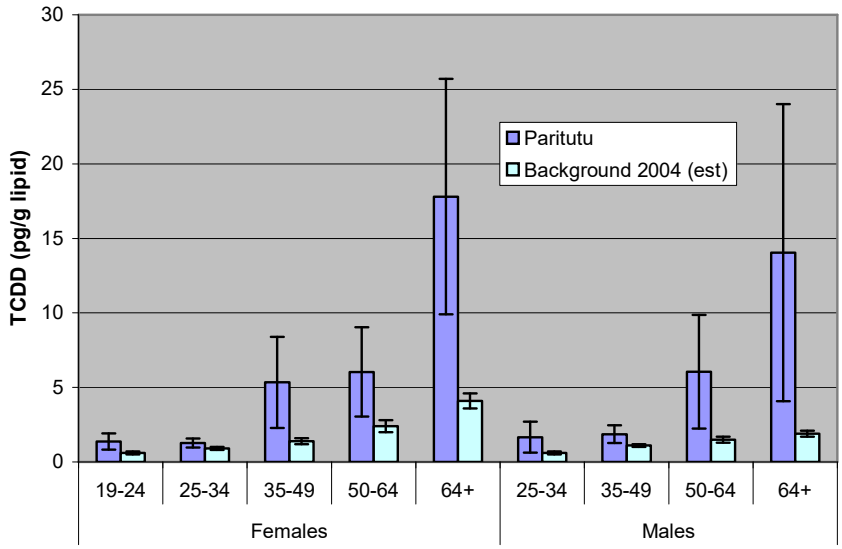
Age group	Paritutu dioxin TEQ (pg/g lipid) Mean [95% CI]	1997 MfE dioxin TEQ (pg/g lipid) Mean [95% CI]	Paritutu PCB TEQ (pg/g lipid) Mean [95% CI]	MfE PCB TEQ (pg/g lipid) Mean [95% CI]
Male				
25-34	**5.9** [0-11.4]	**7.4** [6.4-8.4]		
35-49	**8.0** [0-17.0]	**10.2** [9.4-11.0]		
50-64	**17.5** [5.3-22.7]	**13.9** [12.9-14.9]	**7.6** [3.1-12.1]*	**6.2** [6.1-6.3]
65+	**32.5** [1.7-63.3]	**14.8** [12.9-16.7]	**12.5** [9.0-16.0]*	**8.0** [7.9-8.1]
Female				
15-24	**4.8** [0-10.2]	**6.7** [5.7-7.7]		
25-34	**4.0** [0.1-7.9]	**8.5** [7.6-9.4]		
35-49	**13.8** [2.3-25.3]	**12.7** [11.8-13.6]	**5.5** [1.7-9.4]*	**6.5** [6.5-6.7]
50-64	**16.8** [3.8-29.8]	**16.7** [15.3-18.1]	**7.5** [0.5-14.5]*	**7.1** [7.0-7.2]
65+	**35.6** [0.2-71.0]	**23.7** [22.0-25.4]	**9.7** [3.0-16.4]*	**10.0** [9.9-10.1]
All ages	**16.6** [13.1 – 20.2]]	**13.7**	**8.1** [6.4 – 9.8]	**7.1**

95% CI = lower and upper 95% confidence interval around the mean
* PCBs were only measured in the first 24 participants

As shown in Tables 2 and 3, the overall increase in serum TCDD was less in Part II due to younger average age of participants, and residence in the area for fewer years.

Participant age and exposure duration were significantly associated with TCDD levels in generalised linear regression at $p<0.01$, but no other variables were. Figure 3 shows the arithmetic mean serum levels of TCDD and expected background for each age group of participants.

Figure 3. Arithmetic mean serum TCDD in all subgroups tested (N=52) by age and gender compared to background (means and 95% confidence intervals are shown)



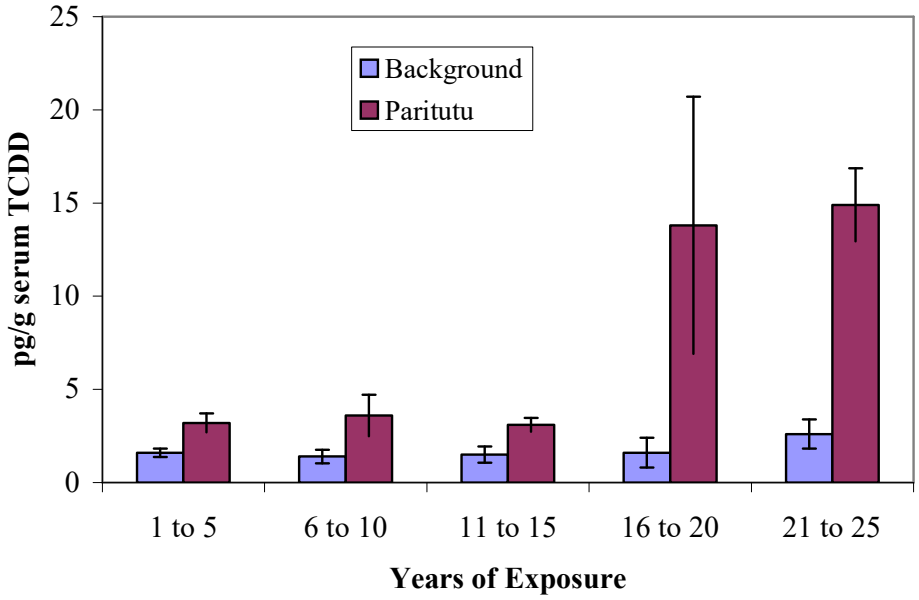
3.2 Role of Timing of Residence

Duration of residence was a key factor in the TCDD elevations found. Clear time periods of particular concern were not evident across the 25-year period of the 2,4,5-T production. Of the 37 people who had lived in the area for less than 15 years from 1962-1987 only one was demonstrably elevated (17.9 pg/g), and the next highest serum result in this group was only moderately elevated at 7.1 pg/g. The mean serum TCDD level in the 37 participants living less than 15 years in the area was 3.2 pg/g. In contrast, those 15 people having lived at least 15 years in the area from 1962-1987 had a mean serum TCDD level of 14.6 pg/g lipid. Figure 4 shows mean serum TCDD levels by the number of years of residential exposure, compared to background values expected for each group of residents (based on the age and gender composition of the group).

For participants living in the area for less than 15 years, the average age-adjusted increase in TCDD was 2.6 pg/g lipid among those living in the area prior to 1974, vs 1.5 pg/g lipid, for those with less than 15 years residence living there only after 1974. This difference was not statistically significant.

The need for a minimum 15-year residence time may also indicate a span of time necessary for exposure to multiple episodic releases of TCDD from the plant, but this cannot be ascertained with certainty with the current limited data.

Figure 4. Effect of years of exposure between 1962-1987 on arithmetic mean TCDD levels in sera of Paritutu residents and expected background (means and standard errors are shown).

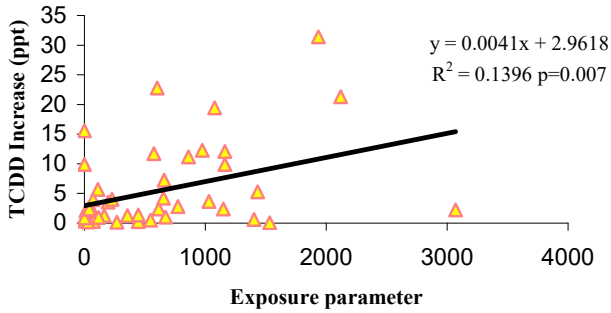


3.3 Role of Home Grown Produce as a Route of Exposure

The questionnaires collected information on the home produce consumption history of each study participant, including the type and extent of edible vegetation grown above and below ground, and also home grown poultry and egg consumption in the relevant years of residence. While all but five of the participants described some level of home vegetable/fruit gardening, only 13 of the 52 (25%) raised chickens for eggs, and only one for poultry meat *and* eggs.

There appeared to be a weak but statistically significant contribution of home gardening of 'exposed fruits and vegetables' (including rhubarb, apples, pears, grapes, silverbeet, cauliflower, cabbages, etc) to the level of serum TCDD in the participants ($p < 0.007$, for correlation, $p < 0.03$ for Spearman's non-parametric test, and $p < 0.014$ for simple regression) (Figure 5). The relationship was strongest when limited to analysis of participants who were over the age of 35 in 1997 (i.e. excluding children and adolescents from the exposure period). However, no relationship was seen for root vegetables, 'protected' fruits (i.e. citrus), or poultry eggs.

Figure 5. Contribution of consumption of exposed fruits and vegetables (i.e. silverbeet, lettuce, cabbages, apples, pears, etc) to TCDD exposure



In Figure 5, the formula used to describe the exposure parameter is:

TCDD increment

$$\propto \%ConsumptionRate * SoilConc * \frac{1}{k}\left[1 - e^{-kdt}\right]e^{-kt_2}$$

The incremental increase in blood lipid level is **proportiona**l to the above equation, **not equal** to it. Assumptions regarding elimination rates, intake and body composition are listed in Appendix P.

Where:

- k is the TCDD elimination rate (= Ln(2)/(half life)). For this calculation, an elimination half-life of 11 years was assumed, but the relationship holds for any assumed value in the published range (7.1 – 11.2 years).
- t_2 = years since last dioxin exposure in Paritutu
- dt is the number of years of exposure at Paritutu
- *%ConsumptionRate* is the percentage of the diet as home-grown produce, and
- *SoilConc* is the soil TCDD level predicted in 2002.

The weak but significant relationship seen with exposed fruit and vegetable garden produce consumption was not seen for 'protected' produce (i.e. citrus), root vegetables (i.e. potatoes, kumaras, carrots), or poultry/eggs. This supports air inhalation and direct deposition onto foods as significant routes of exposure, while indicating that ongoing exposure from soil uptake is not likely to have occurred. Therefore, it is concluded that while home gardening of exposed fruits and vegetables contributed to TCDD increases, generally this contribution was small compared to that from inhalation, and that there is no evidence of significant exposure of an ongoing nature (i.e. through the soil).

3.4 Spatial Analysis of Paritutu Soil Dioxin Levels and the Role of Waste Incinerators

The spatial analysis of measured soil TCDD concentrations in 2002 (and previous samples) showed that the TCDD in the soils around Paritutu most likely originated from the IWD plant (Appendix C). A Krige function using Geospatial Analyst (ArcGIS, ESRI®, Redlands, CA, USA) software showed a strong degree of spatial autocorrelation of soil TCDD concentrations, the highest occurring at the IWD plant, with a rapid decline south of the plant. The highest residential TCDD soil concentration predicted by the Krig function was 106 pg/g, with a total of 37 addresses predicted to be above 40 pg/g. The highest modelled soil concentration at a residence for which we were able to obtain a serum sample in this study was 42.9 pg/g soil.

The predicted soil TCDD concentrations from air dispersion modelling (at a 5 cm soil depth) emanating from the liquid waste incinerator emissions over the 1975-79 period were, maximally, in the range of 0.2-0.6 ng TEQ/kg (Appendix B). In contrast, the actual measured soil TCDD concentrations are in the range 100-300 ng TEQ/kg over the same area. The measured concentrations of TCDD in soil are, therefore, between 150 and 1500-times higher than those predicted by air dispersion and multipathway modelling.

The spatial analysis of the 2002 soil testing data is broadly consistent with a plume of TCDD emanating from the IWD plant, and extending to approximately 1000 meters; predominantly to the East, and approximately 400 meters to the South. The geostatistical model (Figure 6) illustrates this pattern, showing the highest concentrations outside the plant immediately east of the plant boundary.

This pattern of soil concentrations is not consistent with the dispersion/deposition modelling of emissions from the incinerator stacks. The model predicts much lower concentrations overall, and the highest concentrations in soils on Mt Moturoa, with relatively low concentrations immediately east of the IWD plant.

The principal conclusion is that the soil TCDD most likely originated predominantly from emissions that took place in years prior to the incineration operations as a result of one or more airborne releases from the site, or possibly from fugitive emissions from routine operations.

The modelled soil concentrations shown in Figure 6 and Table 7 indicate that there are over 500 addresses in the study area that are predicted to have soil TCDD concentrations in excess of 3.4 pg/g which was the lowest soil concentration in the current study that was associated with elevated serum TCDD after long term residence.

Figure 6. Prediction of soil TCDD concentrations in Paritutu. Areas above background for New Zealand are lightest yellow and background for New Plymouth is one shade darker.

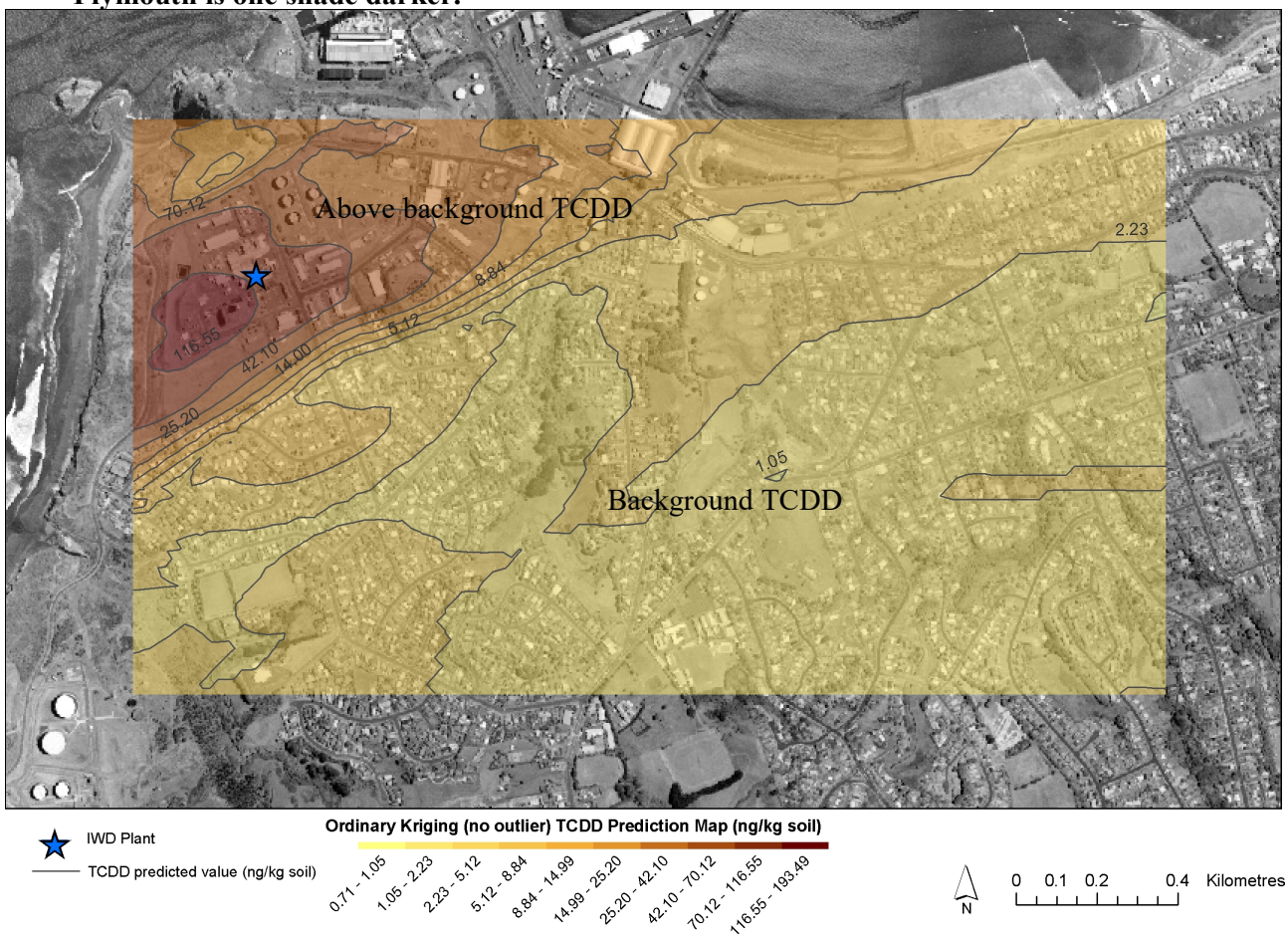


Table 7. Areas of modelled 2,3,7,8-TCDD soil contamination.

Soil TCDD (estimated – 2002 values)	Number of addresses that occur in study area
0 – 3.39	1,679
3.4 – 10	444
10 – 20	52
20 +	41

3.5 Evaluation of the Toxicokinetic Model

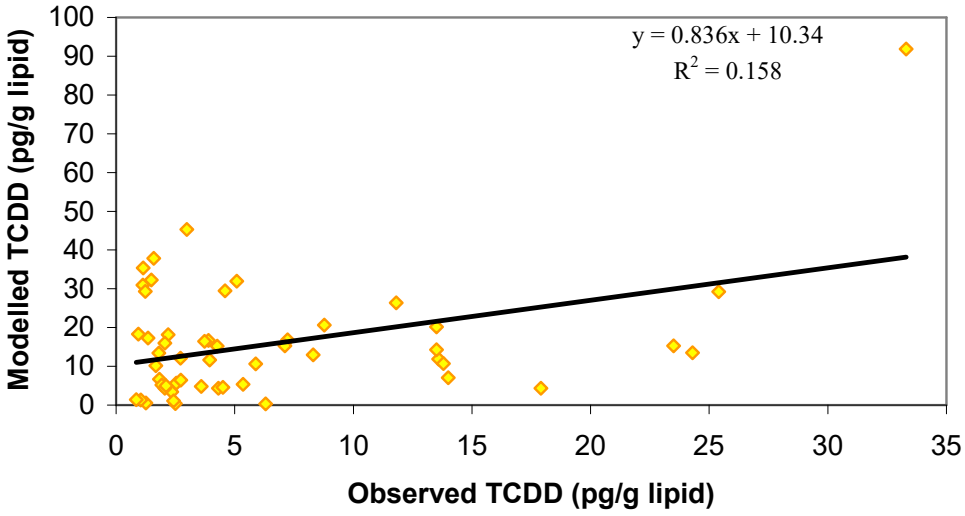
The toxicokinetic model developed for TCDD, estimated the expected magnitude of TCDD retention in subgroups, and helped inform the selection of individuals most likely to be able to show a significant elevation in 2004 (Figure 7; Appendices E, F). For the purposes of participant selection, this model included age and gender-

dependent TCDD background intake and half-life functions. Figure 7 illustrates the relationship between predicted and observed TCDD levels based on the assumed TCDD emission profile between 1962-1975. The y-intercept of 10.34 pg/g lipid indicates the tendency of the model to overpredict observed TCDD values. This may be partly explained by the assumed deposition rate used in the model overestimating ambient air TCDD concentrations and hence inhalation and exposed produce intakes during the emission period which are predicted to be a significant exposure pathways.

The toxicokinetic model can also be used to help back-calculate the extent of historical exposure, based on the individual serum TCDD in 2004, as it includes parameters that affect the elimination half-life of TCDD in the body, such as body fat content, breastfeeding, dietary patterns, and sudden weight loss. These parameters were collected from individuals via questionnaire before testing. The model encounters difficulties in estimating TCDD half-life in obese individuals; there is virtually no reliable information in the international literature on TCDD half-life in persons over the age of 70.

It should be noted that the uncertainties in estimating half-life for obese and elderly individuals was only a potential complication for forecasting the precise degree of serum TCDD elevation in 2004. These issues would not be expected to result in an increase in the probability of a false positive result, but could result in a false negative, or introduce such variability that a very large sample size would be needed to detect a statistically significant difference from background. This did not turn out to be the case in the current study.

Figure 7. Modelled vs observed TCDD in Paritutu participants



3.6 Exposure Reconstruction

An attempt was made to ascertain any significant variations in exposure through the 1962-1987 period. However, due to limited data, we were unable to identify confidently any clear time periods as being critical, or to rule out any particular time period within the 25-year 2,4,5-T production history of the plant. The most important variable observed to predict an increased TCDD was duration of residence. This was particularly evident in people who had lived in the area for a minimum of 15 years.

Ideally, identification of critical time periods of exposure would enable a back-calculation of peak body burden for each individual. If it is assumed that exposures were predominantly airborne, then it is reasonable to use either 1987 as the cut-off point for significant TCDD exposures or an earlier year if the resident moved away from the area prior to 1987.

It is also necessary to select an elimination half-life for TCDD, which varies from person to person, depending on age, gender, and body fat content.

A simplistic calculation of past peak TCDD levels in the test participants gives a range of increased TCDD between 0 (i.e. for those people tested who were at or below expected background in 2004) and 225 pg/g lipid (for the individual with the highest TCDD level), using a half-life value of 7.1 (USEPA 2000). The maximum past peak increase in TCDD was 98 pg/g lipid in the individual with the highest serum TCDD level when using an 11-year half-life (van der Molen et al 1998). These back-calculations assume that exposures ceased in 1987 or earlier if the residence ended before that date. A conservative estimate of peak values suggests an overall average increase in TCDD (above background) for the group of 52 participants of between at least 17 and 35 pg/g lipid. Among participants who lived in the area for more than 15 years, the peak increase above background was estimated to have been at least between 39 and 77 pg/g lipid.

These estimates are conservative in that higher levels of TCDD would have occurred if the exposures ceased earlier than 1987, and there is the possibility that including children in this calculation biases the estimated peak toward lower levels, since the elimination rate of TCDD in small children appears to be faster than adults.

4. DISCUSSION

This study has demonstrated elevations in serum TCDD in selected residents of Paritutu, significantly above that of the general New Zealand population. .

The mean measured TCDD serum concentration for all 52 participants was 6.5 pg/g lipid. The expected national mean for a similar group would be approximately 1.7 pg/g lipid (based on 1997 data extrapolated to 2004). For those 15 participants living in the area for more than 15 years, the average TCDD concentration in 2004 was 14.6 pg/g lipid, whereas the expected mean for the same group was 2.4 pg/g lipid.

Mean elevations in serum TCDD ranged up to 7.3 fold, with older people showing greater elevations than those in younger age groups (see figure 2). Older people have

been shown in overseas studies to have similarly elevated dioxin levels, most likely due to higher exposures in the past (Orloff et al., 2001). The mean elevation in serum dioxin TEQ compared to the 1997 OCP mean was 1.2-fold, due exclusively to the elevation in TCDD. Subtracting TCDD from the total TEQ removed elevations in TEQ among both women and men. Serum PCB levels among the first 24 participants were not significantly elevated by comparison with national background values.

International evidence suggests that TCDD body burdens are falling; for example, lipid-adjusted TCDD levels in the USA, Canada, Germany, and France were estimated to be approximately 2 pg/g lipid in 2000, and are likely to be less than that in 2004 (Aylward and Hayes 2002)[1]. Therefore, the use of 1997 OCP data for comparison is likely to underestimate the true relative magnitude of TCDD elevation in the study group over the general population, and the adjusted values used reflect the lower values expected in 2004.

The elevation in serum TCDD was usefully characterised by multipathway exposure and toxicokinetic modelling, most especially when using the air/soil TCDD deposition rate assumptions from McLachlan (1997). Inhalation of TCDD in air, and, to a lesser extent, uptake of TCDD through 'exposed' fruits and vegetables (silverbeet, apples, cabbages, etc) accounted for the elevated TCDD seen in the study group. There was no significant increase in TCDD for people who indicated regular consumption of seafood from the Paritutu shoreline. There was no evidence for soil uptake of TCDD as evidenced from the lack of association between protected and root vegetables and elevations in TCDD blood levels.

The geographic distribution of TCDD in soil is consistent with prevailing wind patterns and identifies the IWD plant as the source. However, the air dispersion and multipathway exposure modelling based on available data (i.e. incinerator operations and estimates of TCDD released from the 1986 'bursting disc failure') underestimate the observed soil TCDD concentrations by 150-1500 fold. In addition, the dioxin congener profile in soils and sera indicate that TCDD is the only consistently elevated compound, in contrast to the expected diverse profile of congeners arising from incineration. Therefore, one can reasonably conclude that the elevated TCDD in soil and sera is not a result of combustion processes associated with incineration.

Although participants in this study were chosen to optimise the chance of detection of serum TCDD elevations from a previous exposure, the soil spatial modelling indicates that there could be individuals with greater exposures than those represented by the current study group.

The following can reasonably be concluded, based on the data and the information currently available:

- Selected individuals in Paritutu have been exposed to 2,3,7,8-TCDD.
- The resulting (statistically significant) elevations in serum TCDD are correlated to soil TCDD, duration of residence from 1962 to 1987, age and gender.

[1] It it useful to note that TCDD levels are strongly influenced by the age distribution of the population tested.

- The mean dioxin TEQ was elevated, but this was not statistically significant except in those people living in the area for more than 15 years.
- TCDD was responsible for all elevations seen in TEQ above national means.
- Inhalation was the primary route of exposure. However, there is evidence for some additional exposure through 'exposed' (leafy) vegetables and fruits from home-gardening.
- Exposures occurred throughout the period 1962 – 1987.
- Exposures were not the result of a single release of material, but a continual release throughout the production period.

The following can reasonably be excluded, based on the data and the information currently available:

- Incineration as the source of exposure for the study population.
- Inhalation exposure to people born after 1987.
- Soil contamination as a source of significant serum TCDD elevations.

The following remain unanswered by the study:

- Characterising the exposure to residents not included in this study
- Serum TCDD levels in individuals who resided in areas where soil TCDD exceeded those in this study.
- The possibility of people raising poultry residing at addresses estimated to have the highest TCDD soil contamination, and whether some additional ongoing TCDD exposure is occurring in people living at these addresses.
- Characterising exposure to workers at the IWD plant
- Characterising the potential health effects attributable to TCDD exposure for people who were significantly exposed.
- The specific variation in exposures between 1962 and 1987.
- Whether there was a contribution to TCDD exposure from production of chlorinated phenolic products *other than* 2,4,5-T.

The purpose of this study was to characterise exposure of the Paritutu community to dioxins rather than to study the health risks associated with that exposure. A recent study of IWD plant workers by t'Manetje et al., (2005) provides an estimate of excess cancer deaths for those who had occupational exposures at the plant. However, since quantitative measures of exposure in these workers were not obtained, and significant differences between the populations and methods of exposure likely exist, these data are not directly applicable to the residential community surrounding the plant.

Having established dioxin exposure in this community, a logical next step is to establish the feasibility of an epidemiological study using geospatial analysis to determine whether or not the exposed Paritutu community demonstrates evidence of health effects as have been observed previously in other exposed communities (Bertazzi et al 2001, Pesatori et al 2003).

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