

**New Plymouth, Paritutu
Community Dioxin Exposure Assessment Study**

Prepared as part of a Ministry of Health
contract for scientific services

by

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March 2003

Client Report
FW0234

**New Plymouth, Paritutu
Community Dioxin Exposure Assessment Study:
Phase One Findings and Phase Two Methods**

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ACKNOWLEDGEMENTS

ESR wishes to thank the following people for their contribution:

- John Dempsey, Patrick O'Connor, and Vivian O'Leary of Taranaki Health for providing information and facilitating access to the Paritutu Community Health Liaison Group.
- Sue Connor (Toxics Campaigner, Greenpeace Aotearoa, New Zealand)
- Renee Lann of New Plymouth District Council for support, advice and assistance in organising mail outs.
- Other members of the Paritutu Community Health Liaison Group, for their time, support and constructive comments, namely:
 - Paddy Burt (Dioxin Investigation Action Group),
 - Dorothy Doeg (Paritutu Residents Association),
 - Andrew Gibbs, (Dioxin Investigation Network),
 - Peter Old (Multiple Sclerosis Society),
 - Margaret Scannell (Dioxin Legal Action Group), and
 - Andrys Underwood and Vienna Wegman (Ngati Te Whiti Hapu),
- Others, including Francis George and Roy Drake for their advice and guidance.
- Special thanks also go to Deborah Read (Ministry of Health) and Simon Buckland (Ministry for the Environment) for their guidance and expertise.

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SUMMARY

In October 2001 the Ministry of Health (MoH) contracted the Institute of Environmental Science and Research (ESR) to investigate non-occupational exposure to dioxins amongst residents of the New Plymouth suburb of Paritutu who were living, or had lived, close to the local Dow AgroSciences plant, formerly operating as Ivon Watkins-Dow Ltd (IWD). The MoH contract identified blood serum testing as the mechanism for assessing this exposure.

In view of the complex history of the issue it was agreed that, as far as practical, the proposed activities should encompass the perspectives of the key stakeholders particularly the local community, and that all aspects of the project be discussed and agreed prior to commencement.

This consultation [Phase I] 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 will include:

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

This will culminate in the preparation of a report detailing the findings.

1. INTRODUCTION

2. Background to Proposal

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

The purpose of this initial phase (Phase I), as described in the project document is to:

- a) “develop a plan for phase II of the study, namely, where appropriate testing and analysis of human and environmental dioxin residues and their relationship to human exposure, current and historical, and possible adverse (non-occupational) health effects arising from these, principally related to the activities of the Ivon Watkins-Dow Ltd chemical plant between the period of 1960 and 1987 and
- b) ensure that the plan reflects the needs and concerns of the affected residents and other key stakeholders.”

Paritutu is a seaside suburb in the south west of New Plymouth. The map below shows the relationship of the IWD plant to nearby residential areas, although it should be noted that the residential development occurred after the plant’s construction in 1960¹.

Figure 1: Aerial photograph of Paritutu, Spotswood, New Plymouth

¹ There is some variation on the actual date of the IWD relocation from Buller Street to Paritutu Road. The most recent PDP (2002) and Taranaki District Health Board (O’Connor; 2002) reports cite 1960 as the date of transfer of the IWD plant to Paritutu Rd, as does the Taranaki Regional Council’s (2001) investigation of alleged waste disposal. Previous government reports (Coster et al, 1986) cite this transfer occurred in 1962, and Pilgrim’s (1986) submission to the Brinkman et al (1986) Ministerial Committee of Inquiry points to the relocation occurring in 1961. These variances acknowledged, this study follows more recent inquiries and uses 1960 as the date of the IWD site relocation, and as a marker period for including Paritutu residents for further investigation.

(source; 2001 N.Z. Census Data)

Concerns about the measurement and possible impact of dioxins in the environment have been long standing in the Paritutu area. These concerns relate primarily to the activities of Dow AgroSciences, [formerly operating as Ivon Watkins-Dow (IWD)] who manufactured 2,4,5-T and other chemical products at a site in Paritutu from 1960 to 1987.

As a part of an ongoing 'whole of government' initiative, the MoH proposed measuring serum dioxin levels to assess dioxin exposure in selected residents. However, initial discussions with key community representatives on this proposal revealed a variety of concerns in relation to the proposed testing. It was decided therefore, prior to commencing testing to:

- a) clarify the nature of these community concerns;
- b) determine the extent to which serum testing could assist to resolve them;
- c) better communicate the science underpinning the proposed testing and, if appropriate,
- d) design a study.

3. Production of 2,4,5-T in New Zealand

As the cheapest and most effective means to control gorse and scrub, 2,4,5-T was an important contributor to New Zealand's pastoral and agricultural economy for many years to the extent that New Zealand was described as the 'heaviest user of 2,4,5-T in the world' (Brinkman et.al, 1986).

IWD began manufacture of 2,4,5-T in New Plymouth in 1948. In 1960 its manufacturing plant moved from Buller Street to Paritutu. Until 1969 the manufacture of 2,4,5-T was based on imported trichlorophenol (TCP), after 1969 TCP was manufactured locally. Dioxin, specifically 2,3,7,8-TCDD, is a by-product of TCP manufacture. Before 1969 dioxin was present in the imported TCP, and left the factory as a contaminant in the 2,4,5-T. After 1969 dioxin was likely released in air and liquid waste as a contaminant or by-product in the local manufacturing process, as well as leaving the factory as a contaminant in the 2,4,5-T.

From 1973, use of a solvent reduced dioxin levels in the 2,4,5-T from approximately 1 part per million (ppm) to 0.005 ppm. After use in the extraction process the solvent was stored and subsequently burned at a liquid waste incinerator on site between 1975 and 1979. In 1978/79 IWD introduced further changes to reduce the amount of dioxin produced and from 1980 waste was burned in a new solid waste incinerator. 2,4,5-T manufacture ceased in 1987.

The Department of Scientific and Industrial Research measured incinerator emissions for temperature and dioxins every six months from 1974 to 1979, and again periodically from 1983 to 1986. Incinerator emission monitoring has continued to the present time. Ambient air monitoring data for the peak years of liquid waste incineration (1975-1979) is however incomplete. Current Dow AgroSciences management maintain that their internal monitoring records for these periods show 'nothing exceeding the standards prevailing at the time'.

During the period of DSIR monitoring, the detection limits (e.g. 200 ng/m³ flue gas in 1976; [Pilgrim, 1986]) were, compared to current day standards simply a measure of "complete combustion" using

"best practicable means". In other words, there was apparently complete (100%) combustion, an artifact reflecting the relatively crude analytical technology available at the time. As the limits of analytical detection improved percentage limits were implemented on the amount of TCDD destroyed. In 1982, the standard was 99.9% destruction of TCDD.

The DSIR monitoring was done in accordance with the Clean Air Act (1972) with a licence first issued to IWD by the New Plymouth City Council (empowered by the Director General of Health) in 1974. Prior to 1972 there were no reported regulations, standards or monitoring of industry emissions to the environment, although some recourse was available under the nuisance section (s29) of the Health Act (1956). IWD would also have been required to get approval from the Medical Officer of Health and the local authority under the offensive trades section (s54) of the Health Act (1956). Other factors such as fugitive emissions of unknown quantity and the lack of data on dioxin contaminants in various production materials make exposures difficult to accurately assess.

SELECTED DIOXIN STUDIES & REPORTS

4. Dioxin in the New Zealand Environment

The Ministry for the Environment (MfE) Organochlorines Programme (OCP) published a series of reports from 1998 to 2001 (see Buckland et al, 2001) which provided national and regional figures for dioxin-like compounds in various parts of the environment, including food and soils. It is noteworthy that the OCP data showed soil samples taken from near Paritutu had comparatively high concentrations of 2,3,7,8-TCDD, a congener which is normally low in relation to other dioxin congeners when the primary source of dioxin is combustion.

Other soil sampling has been carried out by the Department of Health in 1985 and 1986, Taranaki Regional Council in 2001 and a local community action group in 2001. The results, indicating elevated levels of 2,3,7,8-TCDD in some sites are summarised in the latest MfE report (PDP, 2002). Whilst the results of previous studies and the current PDP samples point to historical exposure, the samples collected in May/June 2002 show that current levels do not exceed international guideline values².

Coster et al (1986) reviewed the manufacture, use, possible health effects and public perceptions of 2,4,5-T (including dioxin levels in the 2,4,5-T manufactured), concluding there was insufficient evidence to recommend banning 2,4,5-T, given its importance to farming and forestry, but recommending a precautionary approach, including further research and a moratorium on its manufacture.

The 1986 Report of the Ministerial Committee of Inquiry to the Minister of Health, known as the Brinkman report, advised on the 'impact on the health of the residents in New Plymouth from the manufacture of pesticides'. The report highlighted the dilemmas around the use of 2,4,5-T at that time, commenting that, for the control of brush weed and gorse in agriculture, there was 'at present no alternative available which is as effective and economic as the phenoxy herbicides' (Brinkman et al, 1986:6). This reflected the report's attempts to balance environmental effects, possible long-term human health concerns and economic considerations. Further research and a banning of spraying of 2,4,5-T in public places and built up areas was advised in the report, but it did 'not endorse the proposal for an immediate moratorium' (Brinkman et al, 1986:17).

² The exception to this is the Mt Moturoa domain sample result of 92 ng/kg for 2,3,7,8-TCDD. This is above the USEPA Region 6 and 9 and ATSDR guidelines, but below the German, USEPA (other regions) and New Zealand guidelines of 1000 and 1500ng/kg.

5. Dioxin and Health Effects in the New Zealand Population

Several occupational epidemiological studies have been conducted in New Zealand with a focus on exposure to dioxins and associated health effects.

Smith et al. (1984), in a case-control study of 82 people with soft tissue sarcoma and 92 controls with other types of cancer, derived relative risk estimates of 1.3 (90% CI= 0.6-2.5) and 1.5 (90% CI= 0.5-4.5) for exposure to phenoxy herbicides and chlorophenols respectively. The authors were unable to draw firm conclusions from this study about the relationship between phenoxyherbicide and chlorophenol exposure and soft tissue sarcoma (Smith and Pearce, 1986).

A subsequent international study by the International Agency for Research on Cancer (IARC) found a dose response relationship for 2,4-D, 2,4,5-T, TCDD, and any polychlorinated dibenzodioxin and non-Hodgkin's lymphoma or soft tissue sarcoma (Kogevinas et al., 1995). Excess risk for exposure to 2,4,5-T, TCDD, and polychlorinated dibenzodioxins or furans was approximately twofold for these diseases.

Smith et al., (1992) found the average TCDD serum level for nine professional 2,4,5-T applicators was almost 10 times that for the matched controls, although the average levels of all other dioxin congeners and isomers did not differ significantly. The variation in TCDD levels among the applicators was related to the duration of their 2,4,5-T exposure. It was concluded that increased risks of cancer from brief exposure to phenoxy herbicide reported in other countries are probably not attributable to the TCDD that contaminates 2,4,5-T. The data from these studies are reviewed in the international context by Vena and Kogevinas (Vena et al, 1998; Kogevinas et al, 1997). The current study however, focuses only on possible non-occupational exposure.

With regard to non-occupationally exposed people, an MfE national survey of dioxin levels in blood has been conducted through the OCP. It examined composite blood samples from around the country to derive biological indices of exposure of the general population to dioxins. The serum samples were collected between December 1996 and November 1997 from 2925 individuals³. The subsequent report (Buckland et al, 2001) comments that 'the NZ mean blood level of 12.8 ng/kg toxic equivalents (TEQ), (lipid-adjusted TEQ basis [excluding dioxin-like PCBs]) is at the low end of international values'.

A wider New Zealand health risk assessment (Smith and Lopipero, 2001) concluded that "the current background exposures to dioxin-like compounds for the New Zealand population has, in our opinion, an insufficient margin of safety and steps should be taken to further reduce exposure". However, Bates et al (1999) comment that the serum dioxin TEQ of non-occupationally exposed New Zealanders, derived from the OCP data (Buckland et al, 2001), is lower than other countries and that the relatively low body burden of dioxins is consistent with levels found in the New Zealand diet and environment. Such population studies do not however exclude the possibility of isolated higher exposures in localised areas.

The Ministry of Health has commissioned the testing of dioxin levels in breast milk on two occasions, with samples taken in 1988 and 1998 (ESR, 2001). Consistent with the serum data the results suggest that dioxin body burdens in New Zealand are low by international comparisons, and, again consistent with international trends, have declined over the past 10 years.

The Brinkman report (1986), referenced above, found no substantiated evidence that the manufacture of TCP and 2,4,5-T had any ill effect on the health of residents of New Plymouth. The choice of 2,4,5-T, rather than 2,3,7,8-TCDD for analysis in body tissue, reflected the limitations of scientific knowledge and technique at that time.

³ 2497 samples were eligible for inclusion on the basis of no evidence of occupational exposure.

A supplementary report to the Ministerial Committee of Inquiry was published in 1987 (Brinkman et al, 1987). This was an assessment of the levels of 2,4,5-T in the urine and blood in a small randomly selected sample of Spotswood residents, IWD employees, farmers, spray contractors and their families. The report findings were that 2,4,5-T was not measurable in the blood of the Spotswood residents, but detectable in the other groups despite no significant recent exposure. Further examination of these individuals found that farmers, sprayer contractors, and their families were likely exposed to the herbicide through physical contact with contaminated clothing. The dermal uptake from contact with these clothes was significant, and had likely been ongoing in some individuals for many years.

The report concluded that under worst case calculations, the amount of TCDD that would be absorbed after 30 years was 2.96 ng/kg, assuming none of the TCDD was eliminated over that time. The report evaluated the data on health effects of dioxin at the time and concluded that there was no evidence that "...dioxin, or 2,4,5-T itself in minimal doses over many years has produced any ill effect on human health" (Brinkman et al, 1987).

6. Dioxin in the Taranaki Environment

In 2001 the Taranaki Regional Council (TRC) investigated dumpsites allegedly used by IWD to dispose of chemical wastes. The council conducted interviews with 80 informants to identify possible locations resulting in the investigation of 36 suspected sites.

The investigations included Ground Penetrating Radar Surveys (GPRS) followed by analysis of a range of soil, sediment, leachate, marine biota, surface and groundwater samples. Sampling protocols were reviewed by the Dioxin Information Network (DIN) and the Dioxin Information Action Group (DIAG). Representatives from these groups also accompanied TRC and consultant staff on the fieldwork.

The study found no evidence of disposal of agricultural wastes beyond those sites already known to the council, no evidence of environmental risk at any site, and recommended no further action.

There are however a number of events [based on a variety of sources] pertinent to possible environmental exposure of residents (and others), some of which it has not been possible to fully investigate and assess:

- a) a 'fire' or 'explosion' at the plant on 3.11.1972;
- b) an 'explosion' or 'emission release' on 15.4.1986 [this event prompted immediate investigation (see PDP, 2002; Appendix B, p2) and provided the impetus for the Brinkman inquiry];
- c) waste drums being damaged in transit on the docks;
- d) damage to garden foliage on different occasions in the early 1970s.

7. Dioxin and Health Effects in the Taranaki Population

The Taranaki District Health Board responded to public concerns about alleged health effects, principally cancers, birth defects and multiple sclerosis, associated with residence near the former IWD plant by publishing an analysis of cancer registrations (1990-97), cancer mortality (1988-97) and birth defect notifications (1988-99) for Moturoa, which includes Paritutu (O'Connor, 2001). The report found no difference in cancer registrations; a lower rate of birth defects notifications and a six percent (within the range of variation expected by chance) higher cancer mortality for Moturoa, compared to the overall New Zealand population. Information on multiple sclerosis was also

obtained from a variety of sources. The data were insufficient to draw conclusions about comparative incidence rates of the disease.

The report commented that the results do not exclude a small increased cancer risk, nor could all historic exposures and possible health effects be assessed given the available data.

An unpublished personal study of congenital malformations at Westown Maternity Hospital between 1965-71 by a local midwife, described 167 birth defects out of a total of 5392 deliveries. No comparison was made to national rates. It is of note that the Westown Maternity Hospital had a large rural catchment, making it difficult to ascribe any rates specifically to the suburb of Paritutu or New Plymouth.

A further study by Taranaki District Health Board released in August 2002 (O'Connor, 2002), covering the years 1965-72 commented that the rate of neural tube defects at Westown Hospital was higher than the estimated national rate, but the difference was not statistically significant. The report describes three cases of neural tube defects whose home addresses were near the IWD factory and comments that, based on the New Plymouth rate, the expected number is about one, but that this difference is of uncertain statistical significance.

The report comments that it was not possible to extend the case search beyond 1972 because of incomplete records and changes in the way records were kept. It concluded that it is not possible from present data, to link neural tube defects at Westown Maternity Hospital and the three cases of neural tube defects in Paritutu, to any particular cause.

8. Dioxin and Health Effects – International

2,3,7,8-TCDD is considered by the International Agency for Research on Cancer (IARC) to be a Group 1 carcinogen (i.e. known to be carcinogenic to humans). It is also considered a known human carcinogen by the U.S. National Toxicology Program.

As a result of the Agent Orange Act of 1991 the Institute of Medicine (IOM) of the National Academy of Sciences of America has carried out reviews (IOM, 1994 et seq) of the scientific evidence about the health effects of exposure to dioxin and other chemical compounds in herbicides used in Vietnam.

The reviews include toxicological (cellular and animal) studies, and information about three epidemiological study populations (Vietnam veterans, and occupationally and environmentally exposed populations). Toxicological studies are reviewed to update information on toxicokinetics, mechanism of action, biological plausibility of toxic effects, and disease outcomes.

Conclusions are reached by evaluating the strengths and limitations of the epidemiological evidence published since the previous reviews, and interpreting the evidence in the context of all the published scientific literature. Specific methodological issues considered include bias, confounding, consistency of findings, statistical power, adequacy of exposure assessment, and length of the observation period. Each disease outcome is assigned to one of four categories based on statistical association (see Table 1 below).

Some diseases have been shifted from one category to another over time e.g. porphyria cutanea tarda from “inadequate/insufficient” to “limited/suggestive evidence” and skin cancers from “limited/suggestive evidence of no association” to “inadequate/insufficient evidence” in 1996, Type 2 diabetes from “inadequate/insufficient” to limited/suggestive evidence” in 2000, and acute myeloid leukaemia (AML) in offspring from “inadequate/insufficient” to “limited/suggestive” in 2000 and to

“inadequate/insufficient” again in 2002. The current associations, with their respective strength of association, are as follows:

Table 1: Strength of Association of Diseases and Herbicide Exposure

Hierarchy by Strength of Association	Disease
Sufficient evidence	Chronic lymphocytic leukemia (CLL) Soft tissue sarcoma Non-Hodgkin's lymphoma Hodgkin's disease Chloracne
Limited/Suggestive evidence	Respiratory cancers (lung, larynx, trachea) Prostate cancer Multiple myeloma Acute and subacute transient peripheral neuropathy Porphyria cutanea tarda Type 2 diabetes Spina bifida (in offspring)
Inadequate/Insufficient evidence	Hepatobiliary cancers Nasal/nasopharyngeal cancer Bone cancer Breast cancer Cancers of the female reproductive tract Renal cancer Bladder cancer Testicular cancer Leukaemia including acute myeloid leukaemia (in offspring) Skin cancers Spontaneous abortion Birth defects (other than spina bifida) Neonatal/infant death and stillbirths Low birthweight Childhood cancer in offspring including AML Abnormal sperm parameters and infertility Cognitive and neuropsychiatric disorders Motor/coordination dysfunction Chronic peripheral nervous system disorders Gastrointestinal, metabolic and digestive disorders Immune system disorders Circulatory disorders Respiratory disorders AL-type primary amyloidosis
Limited/suggestive evidence of NO association	Cancer of the gastrointestinal tract (colon, rectal, stomach and pancreatic tumours) Brain tumours

(Source: Institute of Medicine, 2002)

Other major summaries of the human health effects of dioxin are: the Human Health Reassessment of TCDD and Related Compounds by the U.S.A. Environmental Protection Agency; the International Agency for Research on Cancer (IARC) Monograph, and the Agency for Toxic Substances and Disease Registry (ATSDR), (USEPA 2000, IARC 1997, ATSDR 1998).

CONSULTATION PROCESS

Consultation was primarily with an existing working group representing key community interests. This group, the Paritutu Community Health Liaison Group, was established by Taranaki Health in April 2001 to address ongoing dioxin concerns. Members comprised representatives from Taranaki Health, Ngati Te Whiti Hapu, the Paritutu Residents Association, the Multiple Sclerosis Society, New Plymouth District Council, the Dioxin Investigation Network (DIN), the Dioxin Investigation Action Group (DIAG) and the Dioxin Legal Action Group (DLAG). This group is hereafter referred to as the community consultation group.

Other stakeholders with previous involvement e.g. Te Puni Kokiri were approached at the beginning of the consultation and, although they did not attend the community consultation group meetings, indicated a wish to be informed of the outcomes. Dow AgroSciences similarly were approached early in the consultation process, they also preferred to be kept informed rather than attend the community meetings in person.

The community consultation group forum was also open to interested community members who attended as observers, on the understanding that any comments were to be made on completion of the formal proceedings. The meetings were not advertised publicly, relying on the integrity of the networks of the community representatives. A total of three community consultation group meetings were held between October 2001 and March 2002.

Outside this community consultation group forum, a number of smaller group meetings between ESR staff, individual members and their advisors were also held. At the request of DIN, DIAG and DLAG further background information and expertise was sought, including the New Zealand and Australian Vietnam Veterans Associations, Greenpeace, Dr Pollack (Macquarie University, Australia), and Hatfield Consultants Ltd (Canada).

In addition to the individual meetings, there were also frequent telephone discussions with some group members, concerned community members within these networks, and advisors to DIN and DIAG. Expectations of some people regarding the actions needed to resolve the issue contrasted with the potential of the 'science' proposed to help meet these ends, and were recurrent themes in discussions.

Final consultation to review and amend a draft of this report was conducted in early May 2002 through individual meetings with members of this community consultation group, their wider advisory networks, and other community members who had specifically requested involvement in the process. The Phase II study, as currently proposed, reflects as far as practical the key issues arising in the final consultation.

9. Limits of the Consultation

Given the longstanding, fluid and informal nature of the various groupings, it was difficult to ascertain the extent to which the people involved in the consultation fully represented the interests of the previous and current residents of Paritutu. Whilst the interests of the current Paritutu property owners were clearly represented through the Paritutu Residents Association, it is difficult to ascertain the adequacy of representation of previous residents. Furthermore, DIN, DIAG, DLAG and other such groups were not prepared to share information on membership details and representation to assist in this process.

It should also be emphasised that this was a 'community' rather than 'public' consultation, due largely to the complexity and contested nature of the issues. Consultation was extended through the existing community group and their wider networks. Members of the community consultation group felt they adequately represented the principal concerns of the general public and key stakeholder groups within the community with a keen interest in dioxin and knowledge of the relevant local history.

This report, whilst focusing on investigating dioxin exposure of residents through serum measurement, comments on other issues raised in discussions which, although outside of the remit of the project will be important in maximizing the contribution of any blood testing programme to resolution of the wider issue. These are outlined in Appendix One.

10. FINDINGS

Issues raised in initial discussions were followed up both individually and with others in DIN's and DIAG's wider advisory network. Scientific literature searches were done to complement the information held by these groups and their advisors, and the findings subsequently disseminated to the community consultation group at individual and group meetings.

Four broad sets of concerns were articulated:

- Ill health (resulting from possible historic dioxin exposure).
- Dioxin exposure (in the current residential environment).
- Information access.
- Testing procedures.

Majority agreement was reached as to the way forward, in particular the majority supported the testing of blood serum as a reasonable next step in assessing the body burden of dioxin in those most likely to have been highly exposed. Some background to the four broad sets of concerns is outlined below.

11. Ill Health

Health effects claimed by some local groups e.g. DIN, DIAG would appear, based on the limited information available, to be very varied in nature and encompass a broad spectrum of morbidity and mortality. Many of the effects appear in a small number of families. The same groups cite anecdotal information of historical effects, particularly birth defects in the 1960s and 70s. The latter have subsequently been followed up by Taranaki District Health Board (O'Connor, 2002).

Some community members also perceive insufficient recognition or attention has been given to the concerns of individuals claiming ill health from dioxin exposure. It was difficult to substantiate these claims. Further consideration of these issues will only be possible with full information disclosure by DIN, DIAG, and related groups.

The issue of a more systematic population based study of possible health effects merits consideration, a decision which will be based on collation and evaluation of the results of this and other relevant studies.

12. Dioxin Exposure

Concerns were expressed that previous environmental testing had focused on suspected chemical dumpsites, not specifically on residential properties. The primary concern of the majority of residents is the impact of possible environmental contamination on housing and land value and the risk to health of current and future generations. A recently completed study by MfE has, in major part, addressed the issue of safety for residents living in the area. (PDP, 2002 and MfE/MoH Environment and Health Statement, 2002).

13. Information Access/Representation

Anecdote and other informal networks and mechanisms have become the principal local communication mechanisms. One of the consequences of this is the development of distrust and suspicion from certain sectors of the population, some of which could be lessened through improving access to relevant information.

Full resolution of the issues will only be possible however if there is acceptance by all parties of the need to share information and participate in the democratic process. Monopolisation of proceedings by a vocal minority, especially those without clear evidence of constituency, is contrary to the process.

14. Testing Procedures

Concerns were raised about the appropriateness of the use of blood serum as the optimum body tissue for testing and its validity as a measure of body burden.

The U.S. Centers for Disease Control and Prevention (CDC) have measured dioxin in human specimens since 1985 and have undertaken a number of method validation studies. Prior to 1986 adipose tissue (involving the surgical removal of about 10g of adipose tissue via a needle biopsy) was the sample of choice. Subsequent studies using serum include: Vietnam veteran ground troops, US Air Force Ranch Hand Vietnam veterans, NIOSH workers, residents exposed to contaminated food, and residents of Seveso, Italy (Patterson et al, 1990).

A study of paired serum and adipose tissue samples for TCDD from Missouri residents exposed to TCDD-contaminated waste oil used to control dust, found that the partitioning coefficient of TCDD between serum and adipose tissue on a lipid weight basis is approximately one. This is a very high correlation. Levels were compared over almost three orders of magnitude of concentration (from about 3 to 1000 parts per trillion). TCDD is associated almost completely with lipoproteins in blood. Fasting has no effect on serum concentrations when concentrations are corrected for serum lipid content (Needham et al, 1989). A similar partitioning coefficient was reported between adipose tissue and whole blood (Papke et al, 1988, cited in Turner et al, 1992), and adipose tissue and plasma (Schechter et al, 1990).

A study of paired samples from 20 Vietnam veterans suggested higher chlorinated PCDD/Fs may not partition equally between blood and adipose tissue however despite the individual congener differences the toxic equivalency (TEQ) values for blood and adipose tissue were similar (Schechter et al, 1990). However partitioning has not been well investigated for congeners other than TCDD because a population of sufficient size to have a wide range of concentrations for these congeners has not been found (Turner et al, 1992). Since TCDD is the contaminant associated with 2,4,5-T, the partitioning relationship between serum and adipose for TCDD is the most relevant. Any uncertainty about the relationship for other congeners is not critical for this study.

In the New Zealand serum study mentioned previously organochlorine concentrations in the serum of women on a lipid weight basis were in accordance with results from the New Zealand breast milk study (Buckland et al, 2001). Samples for the two studies were collected close to one another in time (serum: December 1996 to November 1997 and breast milk: October 1998 to May 1999).

CDC states that there is no analytical justification for the use of adipose tissue rather than serum for lipophilic compounds such as PCDD/Fs even if exposure occurred years ago. CDC is also not aware of any information that serum is not a suitable matrix when exposure was in the past.

The long half-life of TCDD permits reliable measurement and interpretation of serum concentrations in people who were excessively exposed more than 30 years ago (Sampson et al, 1994).

A synopsis of this evidence was presented to the community consultation group, and after due consideration it was decided that blood serum was a far more practical proposition (less invasive, more likely to encourage people to participate) for use in the first instance than any other body tissue.

15. SERUM STUDY METHODS

16. Objective

To identify a group of Paritutu residents and others most likely to have been non-occupationally exposed to dioxin from the local manufacture of 2,4,5-T between 1960 and 1987 and compare their serum dioxin levels with a New Zealand population group from the 1998 MfE serum study (Buckland et al, 2001).

5.2 Exposure Assessment and Participant Selection

The process for identification and selection of a group of Paritutu residents most likely highly exposed to dioxin is as follows:

1. Use soil data to model environmental concentration gradients in the vicinity of the IWD plant.
2. Delimit the geographical boundary of the study area by overlaying MfE and other soil data with multi-pathway and plume dispersal estimates to a distance where levels are indistinguishable from New Plymouth background.
3. Based on availability and access, obtain records for the specific geographic area to determine residents of the area over the estimated peak exposure period.
4. Identify these individuals using all practical means including:
 - Past and current electoral rolls
 - Advertisements in newspapers, local newsletters and like publications
5. Send a preliminary questionnaire including cover letter, information leaflet, and stamped addressed envelope to respondents, plus those previously registering an interest with the MoH or Taranaki Health in participating in this study.
6. Collate, assess, and, where necessary, clarify the questionnaire responses.
7. Using the multi-pathway model combined with data on duration of residence and the soil levels, identify a group of individuals for more detailed questioning (nb. the model will need to assume input parameters for fugitive emissions as constant throughout the time period).
8. Send out a second, more detailed questionnaire to individuals meeting screening criteria.
9. Use appropriate Geographic Information Systems (GIS) and related methods to clarify the geographic locality and representativeness of the respondent group compared to the total Paritutu population.

10. Collate data and select the group of people for possible testing.
11. Communicate with all individuals returning questionnaires to advise of the outcome of the selection process, and negotiate informed consent for those selected.
12. Arrange with those selected for an additional informed consent check at the blood collection point.

Accurate exposure assessment in such a situation, i.e. when exposure of an undetermined extent took place many years previously, is very difficult and there are many potential sources of error.

Similarly the time elapsed presents difficulties in tracing historical residents. Depending on access and availability, information sources eg local electoral rolls, rating rolls, Wises and Post Office directories, electricity and telephone records as well as local Plunket and school records will be reviewed to identify the people residing in Paritutu during the relevant time. Participants will be sought through advertising in national and local newspapers, as well as notices in public places in the Paritutu area, and local newsletters including appropriate fora through Te Puni Kokiri and local Maori health groups.

Additionally, those who have previously registered an interest with the MoH or Taranaki Health, (contingent upon the provision of additional confirmatory information) will be forwarded the requisite documentation and information. The questionnaire will be self administered with contact details provided for assistance if needed.

Depending on the number responding to the study publicity who meet the screening criteria, individuals residing in the area for over one year during key exposure times may be traced through current electoral records and subsequently contacted.

Completed questionnaires will be collated and individuals will be selected for testing based on their estimated exposure, which is a function of duration, time frame, soil concentration, and specific lifestyle determinants. Depending on the age breakdown of the individuals, those over the age of 65 may be excluded because this age group has the greatest variability in serum dioxin levels nationally (Buckland et al, 2001) (see Table 2).

Given the history of this issue and recognising the logistical and ethical constraints, people will be given the opportunity to list any other specific concerns including those relating to their personal health. Although self reported morbidity has significant limitations, it does provide an opportunity for people to voice specific concerns. These will not be factored into the exposure assessment, as they are not relevant to the specific objective of the testing programme.

It is proposed that potential participants are ranked using a multi-pathway exposure assessment. This model is outlined below. It will include questionnaire information (see Appendix 2) identifying significant individual behaviours influencing exposure e.g. home grown food, and/or roof rainwater consumption, and/or residence at times of IWD plant 'accidents', plus data on the principal environmental determinants of exposure.

17. Multi Pathway Exposure Assessment & Plume Dispersal Modelling

A plumedispersion model will be used to forecast ground level dioxin concentrations taking into account the local topography, emission parameters and atmospheric data. It should be emphasised that while published sources (Brinkman et al, 1986) cite the dominant source of dioxin emissions from IWD as the liquid and solid waste incinerators, fugitive emissions, by

their very nature cannot be assessed and may have been significant components of the total environmental exposure.

The model will identify gradients of exposure density on a map of Paritutu, which can then be overlaid with the duration of the residence of individuals in the area. This will then be used in a multi-pathway exposure assessment, which will include consideration of the special circumstances of individuals as discussed above.

Benefits of using the plume dispersal model include:

- Accounts for topography and temporal changes in wind direction and climate to estimate areas of relative contamination/exposure.
- Specific events can be quantitatively factored into the exposure criteria, e.g. the 1986 release and the 1972 fire.
- Assists in understanding if the soil levels measured can be explained by incinerator emissions or need other explanation.
- Feeds into a multi-pathway exposure model, which aims to identify individuals at greatest exposure considering a number of special circumstances, such as ingestion of homegrown produce.

The principal alternative i.e. using soil levels and residence as the only criteria is problematic in that soil levels themselves are simply a cumulative reflection of historical emissions and do not provide information about when peak periods of exposure may have occurred. A preliminary analysis of the combination of soil levels with plume dispersal modelling suggests that incinerator emissions may not explain all the TCDD levels seen in the soil (AES, 2002; PDP 2002). Therefore, a greater focus on earlier factory practices that may have led to fugitive emissions may be necessary, thus providing greater information on peak exposure times. Attempts will be made with the appropriate authorities to clarify the likely nature and extent of fugitive emissions.

Fugitive emissions can be estimated indirectly by subtracting the estimated soil dioxin levels in the plume dispersal model from the total soil dioxin concentrations measured in 2002. The remainder being likely to have arisen from fugitive emissions, although not fully explaining the timing of these emissions.

5.3 Bias and Confounding

There are a number of possible sources of bias and confounding. The questionnaire will identify some potential exposure routes and confounders, such as smoking, occupation etc and these can be corrected for in the analysis. Others, many of which will arise during the initial phases of the study, will remain and these will have to be accepted as a limitation of the study. The principal form of bias will be selection bias, which will occur due to a number of factors. Although the model will minimise some aspects, others (e.g. non-participation of people now resident overseas) will still occur, given the resource constraints. Every effort to include ex-residents now resident in other parts of NZ will be made through the placement of advertisements in various national newspapers.

5.4 Blood Collection Procedures and Protocols

These will be established using appropriate informed consent and guidelines, as well as the use of appropriately accredited organisations, namely the New Zealand Blood Service. Included in the consent process, information will be provided about the limitations of the testing, in particular the inability of the results in aggregate and/or individually to be correlated to individual health effects.

5.5 Maori Responsiveness

Staff at the local Te Puni Kokiri office have indicated their willingness to help develop protocols for the collection, storage and disposal of the blood samples. They have also offered to advise on advertisement placement to optimise potential Maori awareness, participation and subsequent communication.

5.6 Data Analysis

The test results will be compared with the relevant data from the MfE serum study (Buckland et al, 2001). In order to maximise the generalisability of any observations or conclusions to the wider community, the focus of the analysis will be on the comparison of the two groups, not individuals within them. However the samples will not be pooled and individuals will have access to their individual results.

Statistically, this presents some challenges since individuals in the MfE serum study were stratified by age, gender, ethnicity, and geographical region, for which analytical tests were undertaken on pooled blood samples within each stratum. There were only a few strata where multiple pooled samples, up to a maximum of three, were analytically tested.

The MfE data showed significant trends by age group but not by geographic region, gender or ethnicity. It is possible therefore to calculate estimates of population variance for age strata by combining the results of multiple pooled samples across gender, geographic region, and ethnicity. This will give an estimate of variance that can be used to calculate the statistical significance of the deviation of the Paritutu mean from the MfE mean.

The original MoH brief for the serum study provided for up to 100 tests based on estimated sample costs and total budget. After consultation it is suggested that the sample number be reduced, given the high cost per unit analysis. For example, the statistical benefit of testing 100 versus 50 samples suggests the ability to discern an approximately 30% smaller difference in mean levels. In part dependent upon the age and exposure groups targeted for the analytical testing, this magnitude of difference is unlikely to translate into any meaningful difference in risk assessment, and, as such, a smaller number than 100 is recommended.

Table 2 outlines several different scenarios in order to estimate the minimum mean analyte level required from the Paritutu residents to obtain a significantly different result from the MfE data should one exist in the wider population of that age group in Paritutu. Levels and variability in the analyte results increase with age group for the MfE data and as such the different age groups need to be examined separately. For each age stratum, several different sample sizes are each examined to identify the minimum mean analyte level required to ensure significantly different results from the MfE survey at 80% power and 95% confidence.

For example, in the 35-49 age stratum, if 10 residents aged 35-49 are tested, then their mean analyte level would need to be 9.8 ng/kg lipid weight basis or greater in order to achieve 80% power and 95% confidence. Equal variance is assumed in the absence of any other information, it is conceivable that the analyte levels for the Paritutu residents could be either more or less variable than those from the MfE survey. The sample size (or power) calculations use the different sample sizes in the two groups in the calculation of the combined effects of variability in the two groups. As the total sample size for the Paritutu residents is likely to be much smaller than that for the MfE survey the variance for the MfE data will be the dominant factor in any statistical analysis.

In addition to some of the statistical questions discussed above there are two other methodological questions which merit further discussion. The first of these relates to understanding what might constitute 'significant' evidence of TCDD exposure above background in the Paritutu community? In this analysis, the focus will be on 2,3,7,8-TCDD levels and their relationship to background estimates of serum TCDD from New Zealand and overseas population data.

The USEPA (2000) defines "background" serum levels for the late 1990s as about 25 pg/g lipid (as dioxins/furans and PCBs as total TEQ). This is for USA only, and this level has declined somewhat since the 1980s when it was estimated to be about 55 pg/g. This reduction could be partially due to improved limits of detection, which would tend to lower the overall estimate in the event of non-detected results⁴. The USEPA also concluded that at intakes of 1 pg/kg body weight/day from all sources, the blood levels would be approximately 7-8 pg TEQ/g lipid, assuming 50% absorption from the diet. For comparison, the New Zealand estimated daily intake is around 1.4 pg TEQ/kg body weight/day (Smith & Lopipero, 2001).

There is the ancillary question of what do these raised levels mean in terms of risk, in particular to human health. It should be re-emphasised that it is not the intent of the project to attempt to link health outcomes and serum dioxin levels. It is however useful to have an understanding of what serum levels are in studies where human health outcomes have been assessed. That said, it should be recognised that there is no solid threshold value for this assessment, but qualitative statements may be made regarding the relationship between the measured TEQ and reported meta-analyses for recognised health effects.

According to the USEPA reassessment of the exposures and health effects of dioxins (2000), adverse effects may not be detectable until body burdens increase to a level 10 to 100 times that of background. In the case of the USA, this translates into blood levels of about 250 – 2500 pg TEQ/g lipid (USEPA 2000). The possibility of adverse effects occurring at lower body burdens (but still statistically above background) could not be excluded, but would be difficult to establish as having a causal relationship.

In addition to the above, there are a number of practical and inter-related issues in this study which merit mention prior to testing as challenges in any analysis/consideration of the significance of exposure and effect using the serum data. These relate primarily to the number of people tested, the size of the comparison sample from the MfE study, and the costs of testing.

⁴ By convention, environmental contaminants that are ubiquitous, including the chlorinated dioxins, are assigned an arbitrary value of one-half the limit of detection (LOD), in the event that the analytical measurements are unable to detect them (i.e. instead of assuming that the true value in that sample is zero). Therefore, a lower LOD tends to be more precise and exert less influence on the estimate of chlorinated dioxins in the environment. This convention has been used by the Ministry for the Environment's Organochlorines Programme, as well as by the WHO, and a number of governmental agencies worldwide.

Table 2: Approximate sample size calculations for 80% power, 95% confidence intervals (assuming a one sided test (ie testing only for a significant increase of 2, 3, 7, 8 -TCDD)

Age group strata	National serum survey sample size	National serum survey mean analyte levels (ng/kg lipid weight basis)	Minimum significant result for the Proposed sample size per strata			
			n=10*	n=25*	n=50*	n=100*
15-24	325	1.0	5.1	3.7	3.0	2.5
25-34	500	1.4	7.8	5.5	4.4	3.6
35-49	550	2.0	9.8	7.0	5.6	4.7
50-64	350	3.1	19.8	13.9	11.0	9.0
65+	275	4.6	39.9	26.9	21.0	17.1

* Assuming similar variability to the national serum survey.

NB the minimum significant result is the mean analyte level in the Paritutu blood samples that would be significantly different from the national serum survey results, for the proposed sample size for that age group stratum.

There needs to be consideration of the optimal selection of individuals across the five strata. In part that is dependent upon those that volunteer for testing and their levels of potential exposure. Clearly younger individuals would be at risk with a lower analyte level, however they are also likely to have lower total exposure. Other issues needing consideration include the fact that 15-24 year olds are unlikely to have had direct exposure with plant emissions. Also the MfE survey results for 65+ year olds are high and highly variable indicating that persons in this age group from Paritutu will need to have very high analyte levels in order to show significant differences from the national results.

In conclusion the age groups 25-34 and 35-49 are the optimal strata for testing, however, the final sample size and selection criteria will be dependent on the response to the invitations for participation.

Whilst direct exposure ceased upon termination of 2,4,5-T production in 1987, the kinetics of dioxin indicate that if significant exposure did occur, dioxins should still be present in blood fat and measurable.

The blood samples collected will not be pooled for analytical testing. Each individual sample will be tested for the seventeen 2,3,7,8-TCDD-like congeners that are of greatest toxicological and environmental significance. This will derive an estimate of relative body burden described as TEQ (toxic equivalent). Possible exposure to 2,3,7,8-TCDD in relation to the other congeners of human significance will be explored. The other analytes also help eliminate the possibility of exposure to other organochlorines from other sources, both local to the plant site, but also the possibility of either work or residential exposure at sites distant from the plant eg people who have resided elsewhere before or after their time in Paritutu.

There is the option of simultaneously testing for non-ortho and mono-ortho PCBs. PCBs have been found to contribute approximately 35% to the measured serum dioxin TEQ in New Zealanders (Smith and Lopipero, 2001). While we have no reason to believe that PCB serum levels in Paritutu will turn out to be elevated compared to New Zealand population norms, we require the analyses to be done in order to fully characterise the total TEQ for the individuals in Paritutu. Therefore we will require analyses of the 12 non-ortho and mono-ortho PCBs in addition to the analysis of chlorinated dioxins and furans. The added cost of including PCBs is approximately 10%. It should be highlighted that the testing for dioxins is expensive. An internationally accredited laboratory will charge up to NZ \$2602.00 per sample. This includes the cost of PCBs and will vary according to current New Zealand dollar exchange rates.

Laboratory advice is that 80 mls of whole blood would normally yield 40 mls of serum, of which 0.2 g is lipid. Based on the lowest level calibration, if TCDD/F is detected at 0.2 pg absolute, then the detection limit for a 40ml serum sample is 1pg/g (lipid basis). The detection limits can be lowered by increasing the blood sample size. For example, if 200ml of whole blood is collected, resulting in 100mls of serum, then the resulting detection limit would be 0.4 pg/g lipid.

18. Study Safeguards

Standard safeguards will be used including the use of peer reviewers and other standard analytic quality assurance procedures e.g. international laboratory accreditation.

Additional peer reviewers nominated by some members of the community included Hatfield Consultants Ltd (Canada) and Greenpeace (Science Unit). Both were given the opportunity to review and comment on drafts of this report, and Hatfield Consultants Ltd have indicated they would be willing to peer review subsequent work. Substantive comments from Greenpeace have not yet been received, but are expected. Greenpeace have declined to be a formal peer reviewer but will be kept informed and have the opportunity for further input.

Individual names and results will remain confidential, however the aggregate results of the study will be published and disseminated through local and scientific fora.

Individuals may also wish to store their serum, at their own expense, for future testing or disposal. ESR can advise on options and costs of this in communication with the participants selected.

6. COMMUNICATING RESULTS AND SUBSEQUENT ACTIONS

In addition to the results of the group as a whole, individuals may wish to know more about their own results. Particularly in the case of an elevated level, the results will need to be communicated with care by an appropriate health professional. Such individuals will be referred initially to their medical practitioner for further discussion. It is recommended that the MoH consider communicating with the relevant local groups at an appropriate time.

In the final consultation process several people raised concerns about the “what if” and the “where to next”. A number of possible scenarios can be envisaged depending on the aggregate environmental and blood results. These can be most usefully explored when all the data are available.

The final consultation also involved asking people to rank three priorities for future action, with the objective of enabling community members to articulate additional issues not covered in the report. Suggestions made are outlined in Appendix One together with ideas mooted in discussion which are not central to the body of the report.

It should be emphasised that the vast majority of the stakeholders and community consultation group felt that while the exercise was useful, consideration of further actions should be deferred until after the results of the study were known.

Scientific Literature Consulted in Preparation of Report

AES (Air and Environmental Sciences) (2002) **Multipathway Assessment of Exposures from Dioxin Releases in the Paritutu Area – unpublished report for ESR** Auckland : Sept 2002.

ATSDR [Agency for Toxic Substances and Disease Registry] (1998) **Toxicological profile for chlorinated dibenzo-p-dioxins**. Atlanta: Agency for Toxic Substances and Disease Registry

Archibeque-Engle, S. L., Tessari, J.D., Winn, D.T., Keefe, T.J., Nett, T.M., Zheng, T. (1997) Comparison of Organochlorine pesticide and polychlorinated biphenyl residues in human breast adipose tissue & serum, in **Journal of Toxicology & Environmental Health**, vol. 52, pp 285-293

Aust, S.D., Millis, C.D., and Holcomb, L. (1987) Relationship of basic research in toxicology to environmental standard setting: the case of polybrominated biphenyls in Michigan, in **Archives of Toxicology**, vol. 60, pp 229-237

Bates M., Buckland, S.J., Ellis, H., Garrett, N., Needham, L., Patterson, D., Turner, W., Russell, D., Wilson, N., Duncan, A. (1999). PCDDs and PCDFs in the serum of the non-occupationally exposed New Zealand population, in **Organohalogen Compounds** vol. 44, pp17-21

Bertazzi, P.A., Consonni, D., Bachetti, S., Rubagotti, M., Baccarelli, A., Zocchette, C., Pesatori, A.C. (2001) Health Effects of Dioxin Exposure: A 20-Year Mortality Study, in **American Journal of Epidemiology**, vol. 153, no. 11, pp 1031-1044

Bertazzi, P.A., Consonni, D., Bachetti, S., Rubagotti, M., Baccarelli, A., Zocchette, C., Pesatori, A.C. (2001) Bertazzi et al. Respond to Smith and Lopipero, in **American Journal of Epidemiology**, vol. 153, no. 11, pp 1048-1049

Blair A., Hoar Zahm S. (1990) Methodologic Issues in Exposure Assessment for Case-Control Studies of Cancer and Herbicides in **American Journal of Industrial Medicine**, vol. 18, pp 285-293

Bond, G.G., Bodner, K.M., Cook, R.R. (1989) Phenoxy Herbicides & Cancer: Insufficient Epidemiologic Evidence for a Causal Relationship, in **Fundamental & Applied Toxicology**, vol. 12, pp 172-188

Brinkman, G.L., Matthews, R.E.F., Earl, W.B. (1986) **Possible health effects of manufacture of 2,4,5-T in New Plymouth**. Report of Ministerial Committee of Inquiry to the Minister of Health (October 1986)

Brinkman, G.L., Matthews, R.E.F., Earl, W.B. (1987) **Possible health effects of manufacture of 2,4,5-T in New Plymouth**. Ministerial Committee of Inquiry Supplementary Report to the Minister of Health (October 1987)

Buckland, S.J., Bates, M.N., Garrett, N., Ellis, H. (2001) **Concentrations of selected organochlorines in the serum of the non-occupationally exposed New Zealand population**. Wellington : Ministry for the Environment, May 2001

Bullman, T.A., & Kang H.K. (1994) The effects of Mustard gas, ionizing radiation, herbicides, trauma and Oil Smoke on US Military Personnel: The Results of Veteran Studies **Annual Review of Public Health** vol. 15, pp 69-90

Carlo, G.L., Lee, N.L., Sund, K.G., Pettygrove, S.D. (1992) The Interplay of Science, Values, and Experiences among Scientists Asked to Evaluate the Hazards of Dioxin, Radon and Environmental Tobacco Smoke in **Society for Risk Analysis**, vol. 12, no.1, pp 37-43

Chess, C., Tamuz, M., Saville, A., Greenberg, M. (1992) Reducing uncertainty and increasing credibility: the case of Sybron Chemicals Inc. in **Industrial Crisis Quarterly**, vol. 6, pp 55-70

Cohen, F.L. (1986) Paternal Contributions to Birth Defects, in **Nursing Clinics of North America**, vol. 21, no. 1, pp 49-65

Coster, A.P., Edmonds, A.S., Fitzsimons, J.M., Goodrich C.G., Howard, J.K., & Tustin, J.R. (1986) **The use of 2,4,5-T in New Zealand: a report to the Environment Council**. Wellington : Commission for the Environment (August 1986)

ESR [Institute of Environmental Science and Research Ltd] (2001) **Investigation of Organochlorine Contaminants in the Milk of New Zealand Women**. Report to the Ministry of Health, April 2001

Fessenden-Raden, J., Fitchen, J., Heath, J.S. (1987) Providing risk information in Communities: Factors influencing what is Heard and Accepted, in **Science, Technology & Human Values**, vol. 12, Issues 3 & 4, pp 94-101

Friedman, J.M. (1984) Does Agent Orange cause Birth Defects?, in **Teratology**, vol. 29, pp 193-221

Giri, A.K. (1986) Mutagenic and Genotoxic effects of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin: A Review, in **Mutation Research**, vol. 168, pp 241-248

Gismondi, M. & Sherman, J. (1996) Pulp Mills, Fish Contamination, and Fish Eaters: A Participatory Workshop on the Politics of Expert Knowledge, in **Central Nervous System** 7 (4), Dec 1996, pp 127-137

Goetz, C.G., Bolla, K.I., and Rogers, S.M. (1994) Neurologic health outcomes & Agent Orange: Institute of Medicine Report, in **Neurology**, vol. 44, pp 801-809

Goodman P.G., Vaughan, C.E., Gill D. (1992) Relocation or Proximity? Major Factors Associated with Prolonged Impact following Dioxin Contamination and Flooding in Missouri, in **International Journal of Mass Emergencies and Disasters**, vol. 10, no. 1, pp 115-132

Heyworth, J. & McCaul, K. (2001) Prevalence of non-specific health symptoms in South Australia, in **International Journal of Environmental Health Research** vol. 11, pp 291-298

Hoppin, J.A. (2000) Pancreatic Cancer & Serum Organochlorine Levels, in **Cancer Epidemiology, Biomarkers & Prevention**, vol. 9, pp 199-205

IARC [International Agency for Research on Cancer] (1997) **Polychlorinated dibenzo-*para*-dioxins and polychlorinated dibenzofurans** .vol. 69

Institute of Medicine (1994) **Veterans and Agent Orange: Health effects of herbicides used in Vietnam**. Washington, DC: National Academy Press

Institute of Medicine (1996) **Veterans and Agent Orange: Update 1996**. Washington, DC: National Academy Press

Institute of Medicine (1999) **Veterans and Agent Orange: Update 1998**. Washington, DC: National Academy Press

Institute of Medicine (2000) **Veterans and Agent Orange: Herbicide/dioxin exposure and Type 2 diabetes**. Washington, DC: National Academy Press

Institute of Medicine (2001) **Veterans and Agent Orange: Update 2000**. Washington, DC: National Academy Press

Institute of Medicine (2002) **Veterans and Agent Orange: Update 2002**. Washington, DC: National Academy Press.

<http://books.nap.edu/books/0309086167/htm/7.htm#pagetop>

Kimbrough, R.D. (1985) Laboratory & Human Studies on Polychlorinated Biphenyls (PCBs) & Related Compounds, in **Environmental Health Perspectives**, vol. 59, pp 99-106

Kocan, A., Petrik, J., Drobna, B., and Chovankova, J. (1994) Levels of PCBs and some organochlorine pesticides in the human population of selected areas of the Slovak Republic, in **Chemosphere**, vol. 29, nos. 9-11, pp 2315-2325

Kogevinas, M., Beccher, H., Benn, T., Bertazzi, P.A., Bofetta, P., Bueno-de-Mesquita, H.B. Coggon, D. Colin, D. Flesch-Janys D., Fingerhut, M., Green, L., Kauppinen, T., Littorin, M. Lynge, E., Matthews, J.D. Neuberger, M., Pearce, N., Saracci, R. (1997) Cancer mortality in workers exposed to phenoxy herbicides, chlorophenols, and dioxins: An expanded and updated international cohort study, in **American Journal of Epidemiology**, vol.145, pp1061-1075

Kogevinas, M., Kauppinen, T., Winkelmann, R., Becher, H., Bertazzi, P.A., Bueno-de-Mesquita, H.B., Coggon, D., Green, L., Johnson, E., Littorin, M., Lynge, E., Marlow, D.A., Mathews, J.D., Neuberger, M., Benn, T., Pannett, B., Pearce, N., Saracci R. (1995) Soft tissue sarcoma and non-Hodgkins lymphoma in workers exposed to phenoxy herbicides, and dioxins: Two nested case control studies, in **Epidemiology**, Vol. 6, No. 4, pages 396-402

McNabb, S. (1993) Contract and Consulting Anthropology in Alaska, in **Human Organization**, vol. 52, pp 216-224

Needham, L., Patterson, D., Pirkle, J., Henderson, L., and Burse, V.W. (1989) The basis for measuring 2,3,7,8-tetrachlorodibenzo-p-dioxin in serum, in **Chemosphere**; vol.18, pp425-430

Needham, L., Patterson, D., Houk, V. (1991) Levels of TCDD in selected human populations and their relevance to human risk assessment, in **Banbury Report 35: Biological basis for risk assessment of dioxins and related compounds**. Cold Spring Harbour Laboratory Press, pp229-257

Needham, L.L., Patterson, D.G., and Turner, W.E. (2002) Assessing levels of 2,3,7,8-tetrachloro-p-dioxin in selected populations. National Institute of Environmental Health (USA)
http://www.niehs.nih.gov/external/usvcrp/conf2002/abs_pdf/diox-013.pdf

Novey, L. (1988) Collective judicial management of mass toxic tort controversies: lessons and issues from the Agent Orange litigation, in **Social Science and Medicine** vol. 27, no 10, pp 1071-1084

O'Connor, P. (2001) **Rates of reported illness in Paritutu and Moturoa**. A report to the Taranaki District Health Board, August 2001

O'Connor, P. (2002) **Neural Tube Defects At Westown Maternity Hospital, 1965-72**. A report to the Taranaki District Health Board, August 2002

Patterson, D., Turner, W., Isaacs, S., Alexander, L. (1990) A method performance evaluation and lessons learned after analyzing more than 5,000 human adipose tissue, serum, and breast milk samples for polychlorinated dibenzo-p-dioxins (PCDDs) and dibenzofurans (PCDFs), in **Chemosphere** vol. 20, pp829-836

PDP [Pattle Delamore Partners Ltd] (2002) **Dioxin Concentrations in Residential Soil, Paritutu, New Plymouth** Wellington : Sept 2002

Pilgrim, R. (1986) **Submission to the Committee of Enquiry into Possible Health Effects of Manufacture of Agricultural Chemicals in New Plymouth.** Wellington: July 1986

Sampson, E., Needham, L., Pirkle, J., Hannon, W., Miller, D.T., Patterson, D.G., Bernert J.T., Ashley, D.L., Hill, R.H., Gunter, E.W., Paschal, D.C., Spierto, F.W., Rich, M.J. (1994) Technical and scientific developments in exposure marker methodology, in **Clinical Chemistry** vol.40, pp1376-1384

Schecter. A., Kassis, I., Papke, O. (1998) Partitioning of dioxins, dibenzofurans, and coplanar PCBs in blood, milk, adipose tissue, placenta and cord blood from five American women, in **Chemosphere** vol 37 (9-12), pp1817-23

Schecter, A., Ryan, J., Constable, J., Baughman, R., Bangert, J., Fuerst, P., Wilmers, K., and Oates, R.P. (1990) Partitioning of 2,3,7,8-chlorinated dibenzo-p-dioxins and dibenzofurans between adipose tissue and plasma lipid of 20 Massachusetts Vietnam veterans, in **Chemosphere** vol. 20, pp951-958

Schecter, A., Ryan, J.J., Masuda, Y., Brandt-Rauf, P., Constable, J., Cau, H.D., Dai, L.C., Quynh, H.T., Phuong, N.T.N., Phiet, P.H. (1994) Chlorinated & Brominated dioxins and Dibenzofurans in Human Tissue Following Exposure, in **Environmental Health Perspectives Supplements**, vol. 102 suppl. 1, pp 135-147

Shan, G., Leeman, W.R., Gee, S.J., Sanborn, J.R., Jones, A.D., Chang, D.P.Y., Hammock, B.D. (2001) Highly sensitive dioxin immunoassay and its application to soil and biota samples, in **Analytica Chimica Acta**, vol 1, pp 169-178

Sharp, D., Eskenazi, B., Harrison, R., Callas, P., & Smith, A.H. (1986) Delayed Health Hazards of Pesticide Exposure, in **Annual Review of Public Health** vol. 7, pp 441-471

Silbergeld, E.K., Mattison, D.R. (1987) Experimental & Chemical Studies on the Reproductive Toxicology of 2, 3, 7, 8-Tetrachlorodibenzo-*p*-Dioxin, in **American Journal of Industrial Medicine**, vol. 11, pp 131-144

Smith, A.H., Lopipero, P. (2001) **Evaluation of the toxicity of dioxins and dioxin-like PCBs: A health risk appraisal for the New Zealand population.** A report for the Ministry for the Environment, February 2001.

Smith, A.H., and Lopipero, P. (2001) Invited Commentary: How do the Seveso findings affect conclusions concerning TCDD as a human carcinogen ?, in **American Journal of Epidemiology**, vol. 153, no. 11, pp 1045-1047

Smith, A.H., Patterson, D.G., Warner, M.L., MacKenzie, R., Needham, L.L. (1992) Serum 2, 3, 7, 8-Tetrachlorodibenzo-*p*-dioxin Levels of New Zealand Pesticide Applicators and their Implication for Cancer Hypotheses, in **Journal of the National Cancer Institute**, vol. 8, no. 2, pp 104-108

Smith, A.H., and Pearce, N.E. (1986) Update on soft tissue sarcoma and phenoxyherbicides in New Zealand, in **Chemosphere** vol 15, pp 1795-1798.

Smith, A.H., Pearce, N.E., Fisher, D.O., Giles, H.J., Teague, C.A., Howard, A.K. (1984) Soft tissue sarcoma and exposure to phenoxyherbicides and chlorophenols in New Zealand, in **Journal of the National Cancer Institute** 73(5): 1111-7

- Steele, E. J., Bellet, A.J., McCullagh, P.J., and Selinger, B. (1990) Reappraisal of the findings on Agent Orange by the Australian Royal Commission, in **Toxicology**, vol. 51, pp 261-268
- Sterling, T. D., and Arundel, A.V. (1986) Health Effects of Phenoxy Herbicides, in **Scandinavian Journal of Work, Environment and Health**, vol. 12, pp 161-173
- Stern, P. C. (1991) Learning through conflict: a realistic strategy for risk communication, in **Policy Sciences**, vol. 24, pp 99-119.
- Sturgeon, S. R., Brock, J.W., Potischman, N., Needham, L.L., Rothman, N., Brinton, L.A., and Hoover, R.N. (1998) Serum concentrations of organochlorine compounds and endometrial cancer risk (United States), in **Cancer Causes and Control**, vol. 9, pp 417-424
- Taranaki Regional Council (2001) **Investigation of alleged agrichemical waste disposal sites in New Plymouth**. Technical Report 2001-42. Stratford : Taranaki Regional Council
- Taylor, S.E., and Repetti, R. L. (1997) Health Psychology: What is an unhealthy environment and How does it get Under the Skin?, in **Annual Review Psychology** vol. 48, pp 411-447
- Turner, W., Patterson, D., and Needham, L. (1992) Factors influencing the measurement and interpretation of serum and adipose tissue levels of PCDDs and PCDFs. **Journal of Exposure Analysis and Environmental Epidemiology** vol. 2: 137-154.
- USEPA [United States Environmental Protection Agency] (2000) Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds. Part III: Integrated Summary and Risk Characterization for 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds.
EPA/600/p-00/001Ag, www.epa.gov/ncea
- Vellinga, A. and Van Loock, F. (2002) The Dioxin Crisis as Experiment to Determine Poultry-Related Campylobacter Enteritis, in **Emerging Infectious Diseases**, vol. 8, no. 1, January, pp 19
- Vena, J., Boffetta, P., Becher, H., Benn, T., Bueno-de-Mesquita, H.B., Coggon, D., Colin, D., Flesch-Janys, D., Green, L., Kauppinen, T., Littorin, M., Lynge, E., Mathews, J.D., Neuberger, M., Pearce, N., Pesatori, A.C., Saracci, R., Steenland, K., Kogevinas, M.,(1998) Exposure to dioxin and nonneoplastic mortality in the expanded IARC international cohort study of phenoxyherbicide and chlorophenol production workers and sprayers, in **Environmental Health Perspectives**. vol.106 Suppl 2, pp645-653
- World Health Organization. (1998) **Assessment of the Health Risk of Dioxins: Re-evaluation of the Tolerable Daily Intake (TDI)**. World Health Organization, European Centre for Environment and Health; International Programme on Chemical Safety. WHO Consultation, May 25-29, 1998, Geneva, Switzerland.
- Zimmerman, R. (1995) When Studies Collide*: Meta-analysis and Rules of Evidence for Environmental Health Policy – Applications to Benzene, Dioxins, and Formaldehyde, in **Policy Studies Journal**, vol. 23, no. 1, pp 123-140

Appendix One: Community Concerns Arising during Consultation

1. Concerns articulated and in part or whole addressed by proposed study or other current studies:

- a) The safety of current residents as a result of possible environmental contamination;
- b) Possible health risks for future generations living in the area;
- c) Depreciating real estate values;
- d) The validity of blood testing as a method for assessing exposure.

2. Concerns articulated, but largely historical and difficult to address through this study:

- a) Distrust of government, science and industry.
- b) Allegations of conflict of interest in key stakeholders.
- c) Government support of 2,4,5-T production.
- d) Perceived lack of timely and appropriate recognition and health care assistance for those claiming adverse health effects from dioxin exposure.
- e) Unreliable data on adverse health effects (especially birth defects).
- f) Disputed facts relating to historical events.
- g) Inclusion of occupational exposure groups in the study, in addition to residents.
- h) Lack of follow up of Brinkman and other report recommendations.
- i) Linkage of claimed health effects and dioxin exposure.

3. Outcomes suggested:

- a) Immediate closure of the Dow AgroSciences Paritutu plant
- b) Soil sterilisation or evacuation of residential areas in proximity to IWD site
- c) Further independent research.
- d) Memoranda of understanding between appropriate organisations.
- e) A forum for dialogue and apologies from industry and government.
- f) Compensation and health care provision for claimed adverse effects.
- g) Longitudinal study of the health of current and ex-Paritutu residents
- h) Hazardous chemical inventory, usage and import and safety data publicly available
- i) More research on women, reproductive health and birth defects in offspring in relation to dioxin exposure.

Appendix Two: Questionnaire

A cover letter will be provided to individuals registering an interest in participating in the study to explain the purpose of the self-administered questionnaire. The letter and

questionnaire will be sent by post. This will include contact numbers for explanations or assistance, and a stamped addressed envelope for return of the completed questionnaire.

DRAFT ONLY (these questions will be asked at different stages of the selection process, rather than in the single questionnaire form shown here)

FOR INITIAL QUESTIONNAIRE

If you lived in Paritutu for at least one year from 1960 to 1987 please answer the following questions

A. Your contact details:

1. Title: (please tick one box only)

Mr Mrs Ms Dr Miss

2. Name: (please put your last name in capitals)

3. Current Address:

4. Contact Telephone number: (__) _____

B. Background information:

Date of Birth: _____

Sex: Male Female

Ethnicity: Which ethnic group do you belong to ?

(mark the spaces or space that applies to you)

N.Z. European

Maori

Samoan

Cook Island Maori

Tongan

Niuean

Chinese

Indian

Other Please state _____

C. Residential History:

(please give your full residential history, that is, everywhere you have lived, what year, and for how long - a separate page is attached if you need more space)

Area	Dates (approx)	Duration (years or months)	Full address if New Plymouth
Eg. Wellington	e.g. 1960-64	e.g 4yrs	

If you have lived outside New Zealand, please state where, when, and for how long?

D. Work History:

(please give your full working history, that is, everywhere you have worked, what year, and for how long - a separate page is attached if you need more space)

Job title	Activity	Year(s) of employment	Duration (yrs or mths if less than one year)	Employer and site
Eg. Forklift driver	Stocking printing warehouse	1978 - 81	3 years, 20hrs per week.	EPI Printers Tawa, Wellington

E. Other Factors:

a) When living in Paritutu, did you raise chickens or other poultry at home for eggs or meat that you ate ?

Yes No

If YES, over what time period ?

b) When living in Paritutu, did you eat fruit or vegetables grown on your property?

Yes No

If YES, please tick how often ?

More than once a week

Once a week

Once or twice a month

Less than once a month

And, over what time period ? (e.g . from 1976-78)

If yes, what sort of homegrown fruit or vegetables did you eat ?

Were the fruit or vegetables peeled ?

Yes, most of the time

Yes, sometimes

No never

FOR INCLUSION IN SUBSEQUENT QUESTIONNAIRES

1. Have any of your jobs involved the use or handling of TCP or 2,4,5-T ? (These may have been in products such as Aero 72, Scrub Dessicant, Stantox 2,4,5-T, and Weedone).

Yes

No

Don't know

If YES, please specify which products

2. Have you been an employee of Dow AgroSciences (formerly DowElanco or Ivon Watkins-Dow) ?

Yes

No

If YES, when and for how long ?

3. Have you worked on the Dow AgroSciences site in the past as a contractor or in some other role ?

Yes

No

If YES, please state what role, when and for how long?

Hobbies or Leisure Activities:

1. Please could you list any hobbies or leisure activities (this is to help determine any opportunities for non-occupational, non-residential exposure to dioxins)

2. Have you ever sprayed the herbicide 2,4,5-T on your property ?

Yes

No

Don't know

Lifestyle:

a. Smoking:

Do you smoke ? Yes No

If YES, how many years have you smoked for ? _____

How many cigarettes per day ? _____

If NO, have you ever smoked regularly in the past? _____

How many cigarettes per day ? _____

b. Dietary Habits:

a) Have you eaten shell fish collected from the Paritutu shoreline ?

Yes No Don't Know

If YES, please tick how often ?

More than once a week

Once a week

Once or twice a month

Less than once a month

And, over what time period ?
(e.g.. from 1976-78)

c) When living in Paritutu, did you drink water collected from the roof ?

Yes No

If YES, over what time period ?
(e.g. 1970-72)

d) Are you a vegetarian or vegan ?

Yes No

If YES, over what time period ?
(e.g. 1970-72)

e) Have you regularly eaten animal fat ?

Yes No

If YES, please tick how often ?

More than once a week
Once a week
Once or twice a month
Less than once a month

If YES, over what time period ?
(e.g. 1970-72)

c. Weight: (rapid weight gain or loss can effect the breakdown of dioxins in the body)

What is your current weight? (please specify kilograms or pounds)

What is your current height ? (please specify metres or feet)

Breastfeeding history ? (If relevant) (dioxin levels decrease with the number of breast fed children and the length of the nursing period)

How many children have you breast fed ?

What was the average length of the nursing period for each child ?

Medication: (medication to lower lipids for high cholesterol (such as Bezalip, Colestid, Fibalip, Gemizol, Lescol, Lipex, Lipitor, Nicotinic Acid, Olbetam, Questran) might interfere with the result)

Please list any long term medications you have taken for the treatment of high cholesterol;

Other activities: Are there any other activities that you are concerned may have increased your chances of exposure to dioxins ?

Yes No

If YES, please specify;

Previous Tests for Dioxin

Have you been tested for dioxin in the past?

Yes No

If YES, what year were the tests done, who did them, and what was the result ?

Additional health information:

Have you been diagnosed as having any of the following conditions:

soft tissue sarcoma	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
non-Hodgkin's lymphoma	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
Hodgkin's disease	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
chloracne	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
chronic lymphocytic leukaemia	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>

(This list is compiled from the *sufficient* evidence category from the USA Institute of Medicine 2002 update of health effects associated with exposure to dioxin)

Have your children, parents or close relatives suffered any of the conditions listed above ?

Yes No Don't know

If YES, please specify what condition:

Blood collection and disposal:

Have you ever donated blood?

Yes No

If YES, when was your last donation ?

Yes No

Do you have any specific requirements for the disposal of your blood ?

Yes No

If YES, please specify:

Informed Consent:

The information you have provided will be entered into a database to guide selection of a group of Paritutu residents most likely to have been at risk of exposure to possible dioxin emissions from the Ivon Watkins Dow factory during the period of operation 1960-1987.

This questionnaire and any further information provided will be kept secure and confidential. This data will be managed so that you remain anonymous throughout the selection process.

You will be informed by writing as to whether or not you have been selected for blood testing.

Please let us know if your contact details change.

(If you are selected to give blood, an additional written informed consent will be required at the time of blood collection)

If you are selected to give blood, are you willing to give up to 300ml blood as part of this study?

Yes No

Are you willing for the *results* of your individual blood test to be combined anonymously with others and for this information to be shared with the local and scientific community ? (Please note you will NOT be personally identified)

Yes No

Full name: _____

Signature: _____

Date: _____

Thank you for your assistance.

Please could you place the completed questionnaire in the stamped addressed envelope provided and post it to ESR as soon as possible.

Detachable information and duplicate informed consent sheet for participant - will include information on issues such as;

Not everyone who completes the questionnaire will have blood taken.

When you will know if you have been chosen to give blood and what will then happen.

Explanation as to why so much blood is needed, that it will be taken by a trained person, and about how long it will take.

Why the work is being done?

Who is funding the work?

Who is running the study?

How does this relate to other studies recently done?

How will people be selected?

How will dioxin levels in my body be measured?

How accurate are these measurements?

What will happen to blood results?

What will happen to the blood?

Contact details for ESR / local health provider: