

# DIOXINS FACT SHEET

## Contents

- General information about dioxins
- 2,4,5-T manufacture in New Zealand
- Dioxins and health
  - General information
  - Overall evaluation of all studies on dioxins
  - Occupational studies
  - Seveso studies
- Dioxins and breastfeeding
- Other studies of blood TCDD (pg/g (ppt) lipid) levels
  - Occupational studies
  - Non-occupational studies
  - Summary
- Paritutu soil study
- Other New Plymouth studies
- The health of the Paritutu population
- Serum dioxin testing
- Reducing dioxin exposure
- Pentachlorophenol
- Work in progress by the Ministry of Health
- References and bibliography

## General information about dioxins

The term “dioxins” refers to a group of environmentally persistent chemicals that share similar chemical structures and mechanism of toxicity. These compounds belong to three closely related families – the polychlorinated dibenzo-*p*-dioxins, polychlorinated dibenzofurans, and certain polychlorinated biphenyls. Dioxins exist in the environment as complex mixtures.

Dioxins in the environment are largely the result of formation as unintentional by-products of combustion and industrial processes. There are a few natural sources of dioxins, such as forest fires and volcanic activity, but generally these have emitted comparatively little dioxins into the environment compared with man made sources. Cigarette smoke also contains small amounts of dioxins.

Seventeen of the dioxins are thought to pose a health and environmental risk. Toxicity of the 17 varies; 2,3,7,8-tetrachlorodibenzo-*p*-dioxin, abbreviated as 2,3,7,8-TCDD or TCDD and commonly referred to as dioxin, is the most toxic.

Some exposure to dioxins is inevitable because of their persistence in the environment. For most New Zealanders about 90 percent of dioxin exposure is through diet, mainly from foods that contain animal fats such as meat, dairy products, eggs and fish. Dioxins enter the food chain via atmospheric deposition onto soil and plant surfaces and subsequent ingestion by grazing animals. Plants take up only very small amounts of dioxins via their roots. Small amounts of exposure occur from inhalation, skin absorption, and ingestion of contaminated soil or dust.

Advances in chemical and environmental management practices since the late 1980s have resulted in a reduction in dioxins emissions in New Zealand. Typically lower levels of dioxins are found in people from less industrialised countries and in younger people. Possible reasons for higher levels in older people include higher exposure several decades ago, differences in metabolism and amount of body fat, and ongoing accumulation. Levels for the New Zealand general population are at the low end of the range of levels reported internationally. Body burdens of dioxins are declining e.g. from 1988 to 1998 dioxins in breast milk of New Zealand women decreased by about 70 percent (Bates et al, 2001).

Once in the body dioxin accumulates in fat and persists for many years. The highest amounts are found in the liver and adipose tissue. In the blood dioxins bind to lipids and lipoproteins and serum TCDD levels are highly correlated with adipose tissue TCDD levels when both are expressed on a lipid weight basis. Dioxins are eliminated mainly in faeces with only small amounts eliminated in urine. Some is eliminated in breast milk. The half-life of TCDD in humans is uncertain but 7 -11 years is generally accepted. It is shorter in children than adults.

## **2,4,5-T manufacture in New Zealand**

The former Ivon Watkins-Dow (IWD), now Dow AgroSciences, chemical plant located in Paritutu, New Plymouth manufactured the herbicide 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) from 1962 to 1987. 2,4,5-T was used extensively in New Zealand to control gorse.

Trichlorophenol (TCP), which is an intermediate in 2,4,5-T manufacture, was manufactured on site from 1969. During TCP manufacture, dioxin is formed and remains as a contaminant in 2,4,5-T. Processing and regulatory changes from 1973 on significantly reduced the TCDD produced. TCDD was not a contaminant in other chemicals known to have been manufactured at the plant.

Incineration of liquid waste occurred on site from 1975 until 1979, and in 1985 and 1986. In 1981 a solid waste incinerator was established. Since 1986 this has operated on a non-continuous basis. Under the Clean Air Act 1972 (implemented in 1974 and replaced by the Resource Management Act 1991) air monitoring was undertaken by the Department of Health.

The Department of Scientific and Industrial Research, on behalf of the Department of Health, measured incinerator emissions for dioxins every six months from 1974 to 1979, and again periodically from 1983 to 1986. Available ambient air monitoring data for the peak years of liquid waste incineration (1975-79) are incomplete. What data are available on historical emissions from the waste incinerator cannot account for the total mass of TCDD present in the soil environment.

The solid waste incinerator continues to operate, but is regularly monitored by the Taranaki Regional Council. Current emissions are below their resource consent and accepted international emission standards. Whilst the incinerator would have contributed some of the exposure measured in the Paritutu population, the Paritutu serum dioxin study report (Fowles et al, 2005) suggests it is very unlikely to be the primary source.

Two chemical release incidents are known to have occurred. In November 1972 there was an explosion in the plant manufacturing the herbicide 4-(4-chloro-2-methylphenoxy) butanoic acid (MCPB); no TCDD was reported to have been released. In April 1986 a bursting disc failure in the TCP plant released TCDD.

In 1980 independent scientists, in association with a union representative, examined current work practices at the plant and found procedures to be satisfactory. However it was recommended that current procedures should be extended to include the pilot plant facility, the functions of which included clean up of plant wastes and recovery of usable materials (Department of Health, 1980).

During the 1970s there were a number of "clusters" of birth defects in New Zealand which were alleged to have been caused by 2,4,5-T. These were investigated by the Department of Health and no evidence was found to implicate 2,4,5-T as a causal factor (Department of Health, 1977).

Concerns relating to uncertainty over exposure to dioxin from the plant and whether health effects have occurred were the subject of a Ministerial inquiry in 1986. The inquiry found no substantiated evidence that the manufacture of 2,4,5-T had any adverse effect on residents' health (Brinkman et al, 1986).

## **Dioxins and health**

### ***General information***

Dioxins differ in toxic potential and the toxicity of individual dioxins is added in order to evaluate mixtures to which people are exposed. The term toxic equivalence (TEQ) refers to the amount of TCDD it would take to equal the combined toxic effect of all the dioxins in the mixture.

To assess health effects from dioxins, in general it is necessary to consider lifetime exposure rather than short-term (e.g. daily intake) exposure. Generally the most useful dose metric is total body burden.

Dioxins bind to a cellular protein, the Ah receptor. Whether adverse effects occur or not depends on what biological responses follow. These responses differ among and within species, and among tissues in individual species. Because of the potential diversity of biological responses to dioxins in the body it is currently not possible to state how, or at what levels, exposed individuals will respond. How much dioxin the person is exposed to and for how long is important as well as individual susceptibility.

Many studies have looked at how dioxins can affect health and much is still not completely understood. Dioxins can affect the growth and development of cells in ways that have the potential to result in a broad range of adverse effects. The most well studied dioxin is TCDD.

Low doses of dioxins produce biochemical changes such as enzyme induction (e.g. CYP1A1) in animals and humans, the clinical significance of which is uncertain (DeVito et al, 1995). At high doses TCDD can cause a severe acne-like skin condition known as chloracne, and cancer in some people. The range of TCDD body burdens that result in chloracne in humans is 436 to 13,600 pg/g lipid (DeVito et al, 1995). Based on a study of workers (Fingerhut et al, 1991) and a 10 year follow up study of the Seveso general population cohort (Bertazzi et al, 1993), DeVito et al (1995) estimated TCDD body burdens at the time of highest exposure associated with increased cancer incidence to be from 495 to 31,800 pg/g lipid.

Animal studies show immune, reproductive and developmental effects. Reproductive and developmental toxicity has been seen in all of the animal species tested and most of these species respond at similar doses. Although the evidence for these non-cancer effects in people is to date limited, these animal studies have been used internationally to establish health-based guidelines for exposure to dioxins in soil, air and food.

Most data indicate that TCDD is not genotoxic. There is some evidence it may have a small indirect genotoxic effect. In animals TCDD is a strong promoter and weak initiator of carcinogenesis. It is therefore plausible that a carcinogenic response to TCDD exposure in humans depends upon exposure to other initiators such as cigarette smoking.

The first evidence that dioxin caused cancer was an animal study published in 1978. Dioxin was not classified as a human carcinogen until 1997 by the International Agency for Research on Cancer (IARC) and the US National Toxicology Program in 1999.

The first epidemiological studies suggesting a cancer risk were a case report of three soft tissue sarcomas in phenoxy herbicide workers (1977) followed by a case control study on soft tissue sarcomas that showed a six-fold excess risk among workers exposed to phenoxy herbicides or chlorophenols (1979).

In the 1980s three large cohort studies were set up – two (US National Institute for Occupational Safety and Health (NIOSH) and IARC) involve chemical workers and workers involved in production or spraying of phenoxy herbicides and chlorophenols from many sites, and one involves people exposed to TCDD in Seveso, Italy following an explosion at a TCP plant in 1976.

There are differences observed among the epidemiological studies particularly for non-cancer effects. Some of these could be explained by differences in exposure levels and length of observation periods since exposure, and, in the case of occupational cohorts, concomitant exposure to other chemicals.

It is also reasonable to assume that the Paritutu residents may have been exposed to other chemicals at the same time as TCDD. Therefore there is the possibility of synergistic effects from multiple chemical exposure.

### ***Overall evaluation of all studies on dioxins***

As a result of the (US) Agent Orange Act of 1991, the Institute of Medicine (IOM) of the National Academy of Sciences has carried out reviews of scientific evidence about health effects of exposure to dioxin and other chemical compounds in herbicides used in Vietnam. This information is provided to the Department of Veterans Affairs and influences what diseases among Vietnam Veterans are recognised for compensation. The reviews include toxicological studies (cellular and animal) and epidemiological studies of three types of population (Vietnam veterans, occupationally exposed, and environmentally exposed). The most recent review was published in 2005.

Those conditions that have been accepted in the sufficient evidence of health effects category by the IOM are Hodgkin's disease (HD), non-Hodgkin's lymphoma (NHL), soft tissue sarcoma (STS), chronic lymphocytic leukaemia (CLL) and chloracne. There is limited or suggestive evidence that exposure to dioxins may cause respiratory cancers (lung, larynx and trachea), prostate cancer, multiple myeloma, acute and sub-acute transient peripheral neuropathy, porphyria cutanea tarda, Type II diabetes, and spina bifida in offspring. A number of other conditions, including other birth defects, have been suggested but there is insufficient or inadequate evidence to confirm these as being caused by exposure to dioxins (IOM, 2005).

In their 2000 review the IOM concluded that there was limited or suggestive evidence of an association between acute myeloid leukaemia (AML) in offspring and dioxin exposure. In 2002 this conclusion was rescinded and AML was moved to the inadequate or insufficient evidence category. The earlier conclusion had largely been based on an Australian study, the data from which were later found to be faulty. After data correction the study showed that children of Australian Vietnam veterans did not have an increased risk of AML. New evidence from German and Norwegian studies of AML in the children of parents who had occupational exposure to pesticides was also considered in the re-evaluation.

Since the last IOM review a meta-analysis of 22 studies of Agent Orange (50% 2,4-D and 50% 2,4,5-T) exposure in Vietnam has been published which shows an increased risk of birth defects (RR<sup>1</sup> 1.95; 95% CI 1.59-2.39) (Ngo et al, 2006). However the conclusions that can be drawn from this study are limited as more than 50% (13 of 22) of the studies included have not been published in any peer-reviewed journal. Eleven of the 13 Vietnamese studies included are unpublished. Commentary on this study by Schecter and Constable (2006) who have published research relating to dioxin exposure in Vietnam state:

*"However we are not convinced that Vietnamese investigations linking congenital malformations to dioxin are, as yet, more than suggestive. We know of no non-Vietnamese studies linking herbicide or dioxin exposure to congenital malformations other than spina bifida and anencephaly....This article and its novel approach confirm the need for continued rigorously controlled research to definitively answer the question [ has exposure to Agent Orange or its dioxin contaminant resulted in an increased incidence of birth defects in Vietnam?] To date the answer is, at best, scientifically equivocal and, at worst, without valid positive scientific evidence."*

### **Occupational studies**

Four highly exposed occupational cohort studies each show small increases in mortality from all cancers combined (SMR for the combined cohorts is 1.4; 95% CI 1.2-1.6) and lung cancer (SMR 1.4; 95% CI 1.1-1.7). All cancer mortality has been shown to increase with higher TCDD exposure and latency period of at least 20 years since exposure (Smith and Lopipero, 2001).

All cancer mortality for 2,187 Dow Chemical Company workers in the United States exposed to dioxins from 1940-83 and followed up to 1994 was the same as the background level (SMR 1.0; 95% CI 0.8-1.1). This cohort was the largest in the IARC cohort and has the longest follow up. Eleven percent of this cohort had developed chloracne but this group had lower than expected all cancer mortality (SMR 0.5; 95% CI 0.3-1.0) (Bodner et al, 2003).

In New Zealand production workers along with sprayers<sup>2</sup> were included in the IARC cohort study of about 22,000 workers in 12 countries exposed to phenoxy herbicides, chlorophenols and dioxins. This study found an association between exposure to phenoxy herbicides contaminated with TCDD or higher chlorinated dioxins with increased mortality from circulatory disease, particularly ischaemic heart disease, and possibly diabetes (Vena et al, 1998) and from STS and slight elevations from all cancers (SMR<sup>3</sup> 1.2; 95% CI 1.1-1.3), NHL and lung cancer. A 29% non-significant excess all cancer mortality was found when workers exposed to TCDD or higher chlorinated dioxins were compared to workers in the IARC cohort with no such

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<sup>1</sup> RR Relative risk

<sup>2</sup> The sprayers cohort comprised 703 sprayers on the chemical applicators register from 1973-1984 which was previously studied by Smith et al (1982) in a study of birth defects.

<sup>3</sup> SMR standardised mortality ratio

exposure (rate ratio 1.29; 95% CI 0.94-1.76) (Kogevinas et al,1997). New Zealand findings were not published separately because the short follow up time to 1990 meant relatively few deaths had occurred.

The two New Zealand cohorts that were part of the IARC cohort have been followed up for a further ten years. Follow up covered 1969-2000 for 813 production workers and 1973-2000 for 699 sprayers classified as exposed to TCDD, higher chlorinated dioxins, and phenoxy herbicides. A 24% non-significant excess all cancer mortality was found among the production workers (SMR 1.24; 95% CI 0.90-1.67). All cancer mortality was highest for synthesis workers (SMR 1.69; 95% CI 0.85-3.03) for whom it was significantly associated with duration of exposure. Lymphohaemopoietic cancer mortality was increased (SMR 1.65; 95% CI 0.53-3.85) particularly for multiple myeloma (SMR 5.51; 95% CI 1.14-16.1). All cancer mortality was reduced for workers handling the final products (SMR 0.83; 95% CI 0.40-1.53) and sprayers (SMR 0.82; 95% CI 0.57-1.14) (t Mannelje et al, 2005). A morbidity study, including analysis of serum dioxins, of former IWD workers is currently being undertaken by the Centre for Public Health Research, Massey University.

Dow AgroSciences is currently undertaking a mortality and morbidity study, including analysis of serum dioxins, of workers but has extended the IWD cohort to also include workers who worked at the Paritutu plant from 1985 to 2001.

A study of New Zealand male pesticide applicators using 2,4,5-T found the rate of birth defects among their children did not differ from the rate among male agricultural contractors. The rate for each group was similar to that reported in other New Zealand studies (Smith et al, 1981; Smith et al, 1982).

### **Seveso studies**

- Background

The Seveso incident resulted in the highest TCDD exposure known in a human residential population. However the exposure (as measured by blood TCDD levels) was in the order of 10 to 25 times less than reported in the occupational cohort studies. It is also unique in that the exposure was to TCDD alone and both genders and all ages are included in the exposed population.

- Chloracne

Chloracne (193 cases) was the only health effect established with certainty at the time of the incident. The majority of cases occurred in children and the highest prevalence was seen in the highest exposed zone in particular close to the factory.

- Cancer incidence

There was a non-significant excess (RR 1.2; 95% CI 0.7-2.1) in cancer incidence in the first ten years (1977-86) after the incident among all people aged 0-19 years living in any of the three exposure zones at the time of the incident. The three zones were grouped given the small size of the population aged 0-19 years in the two most exposed

zones and the rarity of the outcomes being studied in this age group (Pesatori et al, 1993).

- Mortality

After 20 years of follow up the Seveso cohort study has found increased all cancer (SMR 1.1; 95% CI 1.0-1.3), lung and rectal cancer mortality for men. Diabetes mortality was increased for women after 10 years since exposure. For men and women there is a moderate increase in lymphohaemopoietic (includes HD, NHL and leukaemia) cancer mortality. These results include the population from the high exposure as well as the mid-range exposure zones. Increased chronic cardiovascular and respiratory disease mortality occurred in the 5-10 years immediately after the incident in the most exposed zone residents which might be related in part to psychosocial stressors (Bertazzi et al, 2001). Results of the 25 year follow up are due to be published shortly.

- Reproductive health

A cytogenetic study in 1977 found no consistent evidence of chromosomal effects associated with TCDD exposure (Pesatori et al, 2003).

There was no evidence of birth defects attributable to TCDD in 34 cases of abortion which occurred in 1976 after the incident (Pesatori et al, 2003).

There was no increase in birth defects among live births and stillbirths to women who were living in the area at the time of the incident in any of the three exposure zones during 1977-82. The small number of exposed pregnancies in the two most exposed zones might have meant non-detection of a low risk and/or rare defects (Pesatori et al, 2003).

Children born to potentially exposed parents in the 20 years (1977-96) after the incident showed a significantly lower sex ratio (i.e. increased females) with increasing paternal serum TCDD levels. This effect occurred from about 100 pg/g. Fathers exposed when they were less than 19 years old had significantly more girls than boys (sex ratio 0.38; 95% CI 0.30-0.47) (Mocarelli et al, 2000).

The Seveso Women's Health Study (SWHS) was initiated in 1996 to study the effects of TCDD on reproductive health. The study cohort (n=981) comprises women who were one month to 40 years of age in 1976, lived in one of the most highly exposed zones and had blood taken and stored soon after the incident. For the endometriosis study (n=601) participants were not older than 30 years in 1976.

By 1998 15 women in the SWHS cohort had been diagnosed with breast cancer. Serum TCDD close to the time of the incident ranged from 13.1-1,960 pg/g (median 71.8 pg/g). Modelling of these results predicted a statistically significant two-fold increase (HR 2.1; 95% CI 1.0-4.6) in the hazard ratio for breast cancer associated with a 10-fold increase (e.g. from 10 to 100 pg/g) in serum TCDD. The authors consider this to be an early finding as the cohort is relatively young (average age at interview was 41 years) and the number of cases so far is small (Warner et al, 2002).

There was no evidence of an association between serum TCDD level close to the time of the incident and age of menarche (Warner et al, 2004).

A two-fold non-significant excess (RR 2.1; 90% CI 0.5-8.0) for endometriosis was found among women with serum TCDD levels greater than 100 pg/g close to the time of the incident but there was no clear dose-response relationship. Nineteen women in the SWHS cohort were diagnosed with endometriosis (surgically confirmed or ovarian endometriosis diagnosed by ultrasound). Serum TCDD ranged from 9.6-686 pg/g (median 77.3 pg/g). Study limitations include a small number of cases and the possibility of misclassification of disease status as it was not possible to confirm this surgically or by ultrasound for all the participants. Disease status was uncertain for 305 women (Eskenazi et al, 2002).

A retrospective study of pregnancy outcome in women from the two most exposed zones in Seveso found no significant findings in terms of birth outcomes such as birth weight, birth defects, spontaneous abortion and gestational age. Median serum TCDD level was 46.6 pg/g at the time of the incident (TCDD results are for blood taken shortly after the explosion and before conception). Associations for TCDD and lowered birth weight and gestational age were stronger though non-significant for pregnancies occurring within the first half-life (i.e. 8 years) after the incident. Within the first year after the incident about a third of all pregnancies ended in voluntary abortion but the rate did not vary by exposure. Some of these pregnancies could have resulted in an adverse outcome. The authors note that it is possible that the effects are yet to be observed since the most heavily exposed women were the youngest and the least likely to have had a pregnancy at the time of the study (Eskenazi et al, 2003).

### **Dioxins and breastfeeding**

An infant absorbs at least 95 percent of the dioxins in breast milk. Models indicate that the level of dioxins in a breastfed New Zealand infant reaches the mother's level after about six months of breastfeeding (Smith and Lopipero, 2001). Modelling done in the United States shows that by about 10 years of age the level of dioxins in breastfed children is similar to that of formula-fed children (US EPA, 2000).

### **Other studies of blood TCDD (pg/g (ppt) lipid) levels**

#### ***Occupational studies***

The blood TCDD levels estimated at the last time of exposure from three occupational cohorts that have shown increased all cancer mortality are 2,000 pg/g (average) up to 32,000 pg/g, 1,000 to 2,400 pg/g, and 345-3,890 pg/g (Smith and Lopipero, 2001).

The average serum TCDD level of 30 Dow Chemical Company workers exposed to chlorophenols was estimated to be 582 pg/g, assuming a 7-year half-life, and 1928 pg/g using a toxicokinetic model at the time workplace exposure ended (Collins et al, 2005).

A study of nine New Zealand 2,4,5-T applicators, with an average of 193 months spraying, found the average TCDD serum level (53.3 pg/g lipid) in 1988 was almost 10 times that for the matched control subjects (average 5.6 pg/g lipid). In general, the serum TCDD level increased with duration of 2,4,5-T exposure. These applicators had sprayed 2,4,5-T from 83 to 372 months. Given the half-life of TCDD, the findings suggest that the increase in TCDD would be about 3 pg/g among workers who only sprayed for one year (Smith et al, 1992).

A television news programme, TV One Close Up @ 7, had blood from three former IWD workers and one former IWD worker's spouse tested for TCDD in 2004 (TV One Close Up @ 7; 16 December 2004) which were compared with results from the 1996-7 Ministry for the Environment's (MfE) serum study.

		(MfE average)		
Male	50-64 yrs	5.4	(2.5)	(chemical process worker; 1977-1986)
Male	65+ yrs	2.3	(3.0)	(engineer; 1960-1967)
Female	65+ yr	11.6	(5.9)	(nurse; 1981-1996)
Female	50-64 yrs	34.8	(3.6)	(spouse of worker at IWD 1965-1992)

Two of the workers had been involved in 2,4,5-T manufacture, only one of whom had a TCDD level above the average expected for his age group and gender. Although the workers' TCDD levels were less than what would be expected based on overseas occupational studies the small number tested means no conclusions about the TCDD levels of former workers can be made.

### ***Non-occupational studies***

With the exception of Australia, the TCDD levels in the following table may not be representative of the general population of these geographical areas.

The USA average TCDD level of 1.9 pg/g is based on four studies totalling 588 blood samples collected from 1996-2001 from non-exposed people and, with the exception of one study, is not based on a population sample.

### Blood TCDD levels in certain countries

	TCDD (pg/g lipid)	
Germany	3.6 (n= 102; whole blood)	Schechter et al, 1994
Vietnam:		Schechter et al, 1994
Binh Hoa (sth)	28 (pooled n=50; whole blood)	
Dong Nai (sth)	12 (pooled n=33; whole blood)	
Ho Chi Minh City (sth)	3.4 (pooled n=50; whole blood)	
Hanoi (nth)	<2.4 (pooled n=32; whole blood)	
Australia	0.9	Harden et al, 2004
USA	1.9	Patterson et al, 2004

In some geographical areas other dioxins are a much greater contributor to total toxicity than TCDD e.g. TEQ for all dioxins for Germany are similar to USA and two areas (Binh Hoa, Dong Nai) in south Vietnam despite lower TCDD levels (Schechter et al, 1994).

Aerial spraying of Agent Orange occurred in parts of south Vietnam between 1962 and 1971 with the heaviest spraying occurring between 1967 and 1969. Blood samples were taken in 1999 from people living in three communes in central Vietnam where aerial spraying occurred from 1965-70. The amount of aerial spraying was least in Hong Van. Results of pooled whole blood samples from men and women at least 25 years old were:

Huong Lam	M	17 (n=31)
	F	5.3 (n=29)
Hong Thuong	M	21 (n=43)
	F	12 (n=37)
Hong Van	M	ND <sup>4</sup> (n=37)
	F	ND (n=27)

(Dwernychuk et al, 2002)

Following the Seveso incident three exposure zones were classified based on decreasing soil TCDD levels. Populations of the zones at the time of the incident were about 730 (zone A: highest exposure), about 5,900 (zone B: mid-range zone of exposure) and about 38,000 (zone R: low exposure). About 232,000 people from the

<sup>4</sup> ND=not detected

surrounding non-exposed area have been followed up to serve as the reference population.

At the time of the incident no methods were available to measure low TCDD concentrations in small blood samples. Therefore blood taken soon after the incident was stored and analysed in the 1980s or later.

TCDD concentrations for zone A ranged from 828-56,000 pg/g for 10 children with chloracne and from 1,770-10,400 pg/g for nine adults with no chloracne (Bertazzi et al, 1998).

In 1992-3 blood was also taken from randomly selected people over 20 years and TCDD levels back-calculated to 1976 assuming a half-life of 7.1 years.

Back-calculated TCDD results by zone

Exposure zone	average	median	N
A	333.8	388.7	6
B	111.4	76.6	52
R	5.3	5.5	52

(Bertazzi et al, 1998)

TCDD results close to the time of the incident for the SWHS cohort give a range of 3.2-56,000 pg/g (median 272.0 pg/g) for zone A and 2.5-3,140 pg/g (median 47.1 pg/g) for zone B. The youngest children at the time of the incident had the highest levels which decreased with age until about 13 years and then were constant. Zone of residence and age were the strongest predictors of serum TCDD. Other factors related to serum TCDD were chloracne, nearby animal mortality, being outdoors at the time of the incident and consumption of home-grown produce (Eskenazi et al, 2004).

In 1996 (i.e. 20 years later) the average TCDD results among randomly sampled exposed residents were 53.2 pg/g for those in zone A and 11 pg/g in zone B. This compares to 4.9 pg/g in the non-exposed zone. This study excluded people with severe medical illness and previous chloracne (Landi et al, 1998). Levels ranged from 1.0 to 62.6 pg/g in zone B (Landi et al, 1997).

A blood serum dioxin study in 1999 of 28 adult residents of a community in Louisiana, United States concerned about exposure from nearby chemical industries found a mean TCDD level of 7.6 pg/g. Study participants had an average age of 53 years and had lived in the area at least five years. Most reported eating locally caught fish and shellfish although a public health advisory limiting consumption because of chemical contamination had been issued (Orloff et al, 2001).

## **Summary**

The average Paritutu serum TCDD result of 6.5 pg/g in 2004 (i.e. 17 years after 2,4,5-T manufacture ceased) is lower than the mid-range exposed zone of Seveso 20 years after the incident, and lower than most reported results found in areas of central and south Vietnam where aerial spraying of Agent Orange is known to have occurred about 20 to 28 years previously. It is similar to that found in 1999 in a United States community close to chemical plants.

The average serum TCDD result of 14.7 pg/g in 2004 for those who lived in Paritutu at least 15 years from 1962 to 1987 is slightly higher than the mid-range exposed zone of Seveso 20 years after the incident and similar to some, but not as high as the highest, reported results found in areas of central and south Vietnam where aerial spraying of Agent Orange is known to have occurred about 20 to 28 years previously.

## **Paritutu soil study**

A residential soil study in 2002 by the MfE found TCDD at all sites investigated, but all but one result were below the most conservative residential guidelines set to protect people's health that exist internationally.

These soil findings are consistent with historical emissions from the plant as the source of TCDD in the area as the level of TCDD is normally low in relation to other dioxins when the primary source of dioxin is combustion. A previous MfE study, which was published in 1998, did not find TCDD in urban soils in any parts of New Zealand other than New Plymouth.

Concentrations tend to be highest close to the former IWD plant, and drop off rapidly within 800 to 1000 metres from the plant. Concentrations to the east of the plant, towards Mount Moturoa Domain, are higher than to the south of the plant. This is consistent with the prevailing winds in the area.

Dioxin is very stable under most environmental conditions, undergoing only very slow change in undisturbed soil over many decades. Consequently, it is considered unlikely that historical TCDD soil levels on residential properties would have been significantly higher than the levels measured in this study and would have exceeded the health-based soil guidelines.

## **Other New Plymouth studies**

In 1980 an independent clinical assessment of 45 current IWD workers (90% response rate) involved with 2,4,5-T manufacture found no evidence to indicate that their health had been adversely affected by their work. This included a comprehensive medical examination and routine laboratory tests. Three pregnancies among the partners of

workers during their time employed by IWD had resulted in miscarriages; in two cases there was a history of miscarriage, stillbirth or birth defects prior to employment at IWD (Department of Health, 1980).

A cancer mortality atlas published in 1982 by the Department of Health using 1974-78 mortality data found a higher rate of NHL and HD in New Plymouth compared to the national average. At that time there was no scientific evidence of an association between lymphatic cancer and dioxin.

From 1965 to 1971, 3.1 percent of babies born at Westown Maternity Hospital were reported by a former midwife to have had a birth defect. Her study recorded 48 of 167 birth defects as neural tube defects defined as including anencephaly, hydrocephaly, microcephaly and spina bifida (Carnachan, 2002). Neural tube defects are usually defined as including anencephaly and spina bifida, but not hydrocephaly, which may be caused by spina bifida, or microcephaly.

Dr Patrick O'Connor, former Medical Officer of Health, has carried out two studies (2001 and 2002) in response to public concerns about health effects associated with living near the former IWD plant. No difference in cancer registrations (1990-97), a lower rate of birth defects notifications (1988-99) and six percent (within the range of variation expected by chance) higher cancer mortality (1988-97) was found compared to the New Zealand population. The results do not exclude a small increased cancer risk. Data for multiple sclerosis<sup>5</sup> were insufficient to draw conclusions about comparative incidence rates of the disease.

The New Plymouth rate of neural tube defects (1965 -72) was slightly higher than the estimated national rate but the difference was not statistically significant. Three cases were identified from an area near IWD, which was two cases more than what was expected based on the New Plymouth rate. Although not a statistically significant difference this is uncertain given uncertainties with the data and the definition of the study area.

In late 2005 the Ministry of Health released the findings of a study of all cancer and HD, NHL, STS, and CLL incidence and mortality in New Plymouth from 1970 to 2001. This study found excess all cancer (SIR<sup>6</sup> 111, 95% CI 104-119), NHL (SIR 175, 95% CI 121-246) and CLL (SIR 251, 95% CI 144-408) incidence for 1970-74 compared to the rest of New Zealand. This is the only time period that shows an elevated cancer risk for all cancers and at least one of the four specific cancers associated with dioxin exposure. Assuming a 10-year minimum latency period and the cause was TCDD, the period of exposure would have been 1960-64, which is partially outside of the 2,4,5-T manufacturing period and before TCP was manufactured on site. Annual 2,4,5-T production was also low during 1962-64 compared to other years when the level of TCDD in 2,4,5-T was the same. Whilst TCDD exposure in the first few years of 2,4,5-T manufacture may have had a role, unknown exposure(s) before the start of 2,4,5-T

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<sup>5</sup> Multiple sclerosis had been raised as a concern by the community.

<sup>6</sup> SIR Standardised Incidence Ratio

manufacture and chance are also possible explanations. The study's limitations mean the possibility of an undetectable small elevation in cancer risk cannot be excluded (Read et al, in press).

### **The health of the Paritutu population**

To date there is no scientific evidence of increased disease rates in the New Plymouth population attributable to dioxins. However current data limitations mean the possibility of a small increased risk cannot be excluded.

It is possible that the TCDD levels found may have health consequences for individuals or may cause increased rates of disease, in particular cancer, on a population basis. The extent of the cancer risk is highly uncertain but based on the evidence to date from the more highly exposed IARC occupational cohort and the Seveso cohort it is estimated that it may be up to 10 percent above the national cancer mortality rate. A third of deaths in New Zealand are caused by cancer. Therefore if 100 people died in Paritutu every year about 33 deaths would be from cancer and up to an extra 3 cancer deaths may be attributable to dioxin. However these people would have had to have lived in the most exposed areas (i.e. 1 km to the east and about 400 m to the south) for at least 15 years from 1962 to 1987.

### **Serum dioxin testing**

Individual blood dioxin testing is not recommended. The results only indicate if the person has been exposed to dioxin and cannot be used to predict whether that person will develop health effects or not because of the exposure.

Tests for measuring dioxin levels in people are not routinely available. A blood dioxin test costs about \$2,500 and depending on the detection limit a large volume of blood is required e.g. 200 ml.

There are only a few laboratories in the world that are accredited by the World Health Organisation as capable of achieving low enough detection levels to detect blood dioxin levels that occur in the general population. If the detection limit is too high and various dioxins are not detected the scientific convention when calculating the TEQ is to assume that those dioxins are actually present at a level of half the detection limit value. Depending on the number of non-detectable dioxins this may result in an uninformative result.

### **Reducing dioxin exposure**

There is no generally accepted treatment to get rid of dioxins now in people. Everyone has some dioxins in their body although levels in the general population are decreasing.

Reduction in the amount of animal fat in the diet reduces dioxin exposure. It is not recommended that all fat is eliminated from the diet as a moderate amount is part of a healthy balanced diet.

## **Pentachlorophenol**

Pentachlorophenol (PCP) is another chemical that was widely used in New Zealand, primarily in the timber industry, which was contaminated with dioxins. Dioxins in PCP are mostly hexa-, hepta- and octa-chlorodibenzo-*p*-dioxins and some higher chlorinated furans. No PCP was manufactured in New Zealand, and its use in the timber industry ceased in 1988. One small study has shown that workers handling the substance have elevated serum dioxin levels. The Centre for Public Health Research, Massey University is undertaking a study of the health effects associated with PCP among timber treatment workers.

## **Work in progress by the Ministry of Health**

- Addition of 1980-1989 birth defect data from the New Zealand Birth Defects Monitoring Programme to the electronic database which began in 1990. Previously this data has only been available on paper files, which has limited analysis. This will assist the Ministry to answer questions about past birth defects in New Plymouth. One of the outcomes of entering the data will be to compare New Plymouth data with national data.
- Breast milk study

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