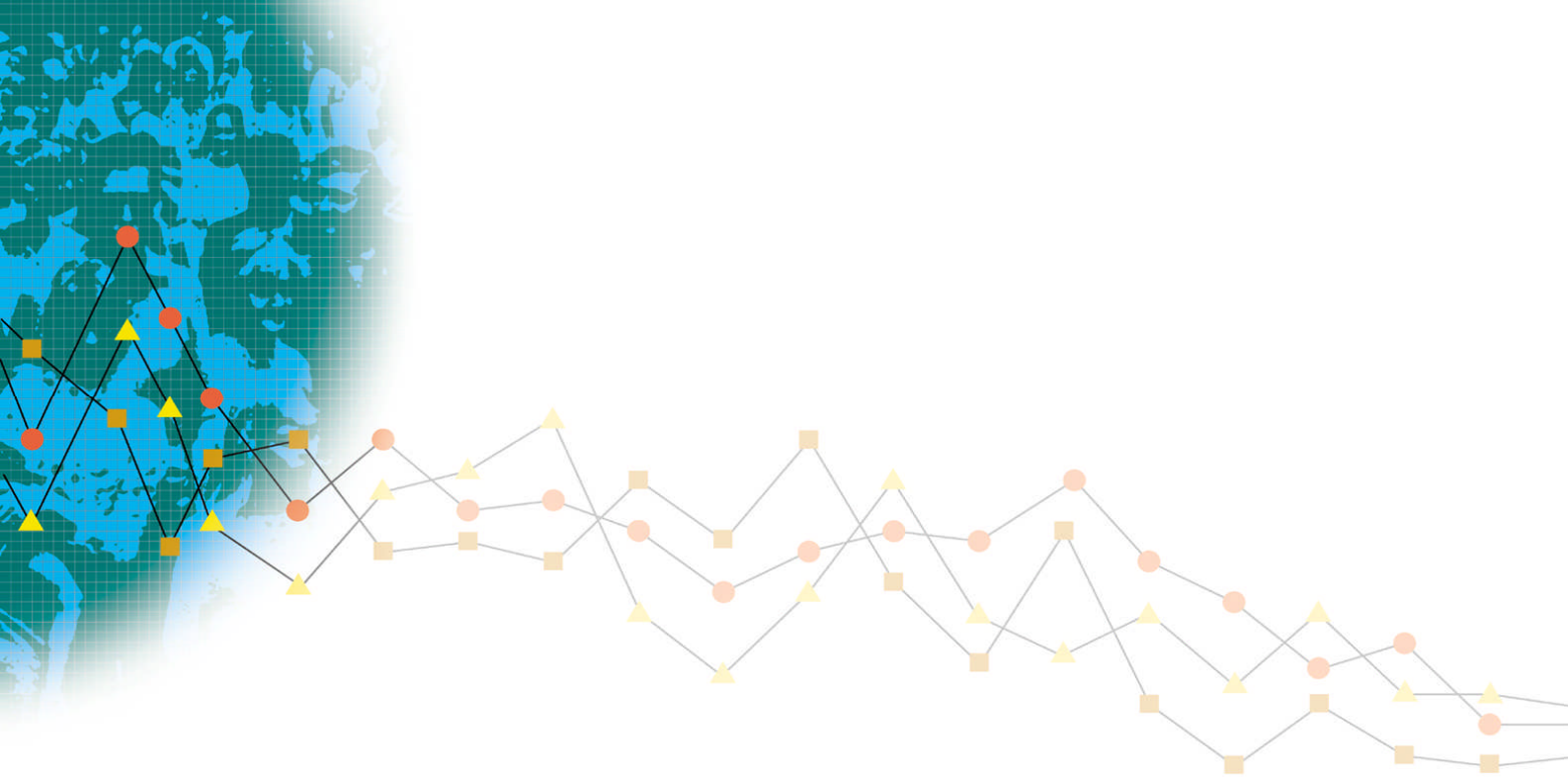


Birth Defects in the New Plymouth District

Barry Borman and Deborah Read

CPHR Technical Report no 32

Centre for Public Health Research
Massey University Wellington Campus
Private Box 756
Wellington
Phone: 64-4-801 5799 ext 62180
Fax: 64-4-380 0600
E-mail: b.borman@massey.ac.nz

ACKNOWLEDGEMENTS

The authors would like to thank Dr Patrick O'Connor, Medical Officer of Health, Wanganui DHB and Dr John Harris, former Director, California Birth Defects Monitoring Program for peer reviewing the document and Mr Andrew Gibbs for providing additional data.

CONTENTS

EXECUTIVE SUMMARY

Background

The herbicide 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) which contained the dioxin 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) as a contaminant was used extensively in New Zealand to control gorse. There has been longstanding community concern over the possibility of health effects, including birth defects, on the local population from its manufacture between 1962 and 1987 at the former Ivon Watkins-Dow (IWD) chemical plant in Paritutu, New Plymouth.

Methods

During 1965-71, a midwife at Westown Maternity Hospital (WMH) in New Plymouth, collected data on the occurrence of the birth defects in births in the hospital. These unpublished data have been used to compare the prevalences of birth defects in New Plymouth with those reported in other New Zealand hospital and population based national and local studies for the period prior to the establishment of the New Zealand Birth Defects Monitoring Programme (NZBDMP). From 1980 to 1989 data from the NZBDMP were used to compare the prevalences of birth defects in New Plymouth to those reported in other health districts. Prevalence rates and 95% confidence intervals were calculated.

Results

During 1965-71, the overall rate of birth defects, and specifically talipes and congenital dislocation of the hips (CHD), at the WMH was significantly higher that the rates reported from some other studies in New Zealand at that time.

However, there was no difference between the rates of spina bifida, Down syndrome, congenital heart defects, and facial clefts. An association has been reported between TCDD and spina bifida (Institute of Medicine, 2009).

Data from the NZBDMP showed the rate of birth defects in New Plymouth was consistently higher than the national average and many other areas during 1980-89. The difference was likely due to an ascertainment bias with exceptionally high rates of CHD and talipes in New Plymouth.

Since 1964, an orthopaedic surgeon in the Taranaki region, had 'dedicated himself to ensuring every baby born in Taranaki Base Hospital's maternity unit has been, and will continue to, be tested for displaced hips'. Taranaki was also reported to have the highest number of orthopaedic surgeons per head of population in New Zealand. A previous study found that the rate of CHD was more than three times the rate at National Women's Hospital, the major referral centre in Auckland.

Conclusion

These data provide no evidence of an effect on the rates of birth defects from 2,4,5-T/TCDD exposure in New Plymouth.

INTRODUCTION

The herbicide 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) which contained the dioxin 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) as a contaminant was used extensively in New Zealand to control gorse.

Sara and Forbes (1972) made the first suggestion of dysmorphogenetic effects of 2,4,5-T usage in New Zealand. In a letter to the editor of the New Zealand Medical Journal the authors reported two babies born with myelomeningocele (a variant of spina bifida) within a month of each other from adjacent Waikato farms 'where for several years aerial spraying has been carried out with a chemical called 2,4,5-T.' They added 'we cannot prove that the two women concerned here did in fact drink water contaminated by dioxin' but 'believe that 2,4,5-T should be treated with grave suspicion.'

The Department of Health reviewed the toxicology and epidemiology of 2,4,5-T and three alleged 'clusters' of neural tube defects (NTDs) in Waikato, Northland and Taranaki. The Taranaki cluster comprised five NTDs, four which occurred in Opunake (three in one road) in 1974-76 and one in Normanby in 1977. No evidence was found to implicate 2,4,5-T as a causal factor in any of these 'clusters' (Department of Health 1977a, 1977b). In Taranaki three of the mothers had no exposure to pesticides in the first trimester of their pregnancy (the period during which NTDs arise), while the fourth had contact with Tordon 50D (2,4-D and picloram) and possibly to 2,4,5-T. The Department of Health also carried out an investigation in response to a medical practitioner linking the birth of two babies with fatal congenital abnormalities to 2,4,5-T exposure. One baby had biliary atresia and the other had cardiac defects. It was not established that either woman was significantly exposed to 2,4,5-T at any time during her pregnancy (Department of Health 1980b).

Hanify et al (1980, 1981a, 1981b) compared all birth defects in Northland maternity hospital catchment areas during 1960-77 with densities of aerial

2,4,5-T spraying in the same areas and time period. Spraying was significantly associated with talipes, independent of ethnicity, but not with spina bifida, anencephaly, cleft lip with or without cleft palate, isolated cleft palate, cardiac defects or hypospadias/epispadias.

Smith et al (1981, 1982) found the rate of birth defects among the children of New Zealand male pesticide applicators using 2,4,5-T 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.

From 1962-87, 2,4,5-T was manufactured at the former Ivon Watkins-Dow (IWD), now Dow AgroSciences, chemical plant in Paritutu, New Plymouth. In 1980 an independent clinical assessment, including a comprehensive medical examination and routine laboratory tests, was carried out on 45 current IWD workers (90% response rate) involved with 2,4,5-T manufacture. No evidence was found to indicate that their health had been adversely affected by their work. 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 1980a).

In 1986 a Ministerial inquiry was carried out over concerns relating to uncertainty over exposure to dioxin from the IWD plant and possible health effects (Brinkman et al 1986). The inquiry noted that ‘Submissions from individuals claiming that the pesticides [2,4,5-T and 2,4-D] had caused congenital defects, excessive miscarriages and soft tissue tumours were mainly anecdotal accounts.’ Conversely, several commercial sprayers and farmers, who had used ‘these chemicals for years and are often soaked to the skin when spraying and yet they have stated that they and their families had not suffered any ill health. Many stated that they had produced several healthy children with no miscarriages or congenital defects among their progeny.’

The inquiry concluded there was ‘no substantiated evidence that the manufacture of these pesticides [2,4,5-T and 2,4-D] has had any ill effect on the health of the residents of New Plymouth’ (Brinkman et al 1986).

Carnachan (2002) reported that a former midwife, Hyacinth Henderson, at Westown Maternity Hospital, New Plymouth, had recorded that 3.1 percent of babies born at the institution during 1965-71 had a birth defect. Her study (the 'Henderson study’) recorded 48 of 167 birth defects as NTDs including anencephaly, hydrocephaly, microcephaly and spina bifida (Carnachan 2002). O’Connor, a Medical Officer of Health, carried out two studies in response to public concerns about health effects associated with living near the former IWD plant. The first (O’Connor 2001) found a lower rate of birth defects notifications (1988-99) compared to the New Zealand population.

In the second, O’Connor (2002) investigated the prevalence of NTDs at Westown Maternity Hospital for the period 1965-72, using the historically available labour ward records. The focus on NTDs was because the ward records often only mentioned major defects and since 1996 the US Institute of Medicine (IOM) has concluded that there is suggestive evidence that paternal exposure to TCDD and herbicides used in Vietnam such as 2,4,5-T is associated with spina bifida in veterans’ children and insufficient or inadequate evidence for any other birth defect (IOM 2009).

A table of the data collected by Henderson was also presented (Table 1) and compared to data O’Connor had collected. Henderson noted 28 cases of NTDs, anencephaly and spina bifida, but O’Connor could only identify 24 cases. The difference could have been due to the way in which the cases and defects were classified. O’Connor found the New Plymouth rate of NTDs (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 were two cases more than what was expected based on the New Plymouth rate. The difference was labelled of ‘uncertain statistical significance’ given the ‘uncertainties of our data, and the fact that much depends on the definition of the study area’ (O’Connor 2002).

Table 1: New Plymouth Hospital records of congenital malformations delivered at Westown Maternity Hospital – the 'Henderson study'. 'Reconstruction of a table provided by the CN [charge nurse Hyacinth Henderson] based on her personal data collection' (O'Connor, 2002)

Defect as recorded	**1965-66**	**1966-67**	**1967-68**	**1968-69**	**1969-70**	**1970-71**	**Total**
Anencephalic	3	0	4	2	1	2	12
Hydrocephaly	2	2	6	1	0	4	15
Microcephaly	1	1	2	0	1	0	5
Meningocele	2	0	5	3	1	3	14
Spina bifida	0	0	0	0	0	2	2
Congenital Heart	1	2	6	3	0	0	12
1 Artery Cord	6	1	1	1	5	2	16
Cystic Kidney	0	0	0	0	0	1	1
Renal Agenesis	0	0	1	0	1	0	2
Atresia of Bowel	0	1	1	1	1	2	6
Tracheo-oesophageal Fistula	0	1	0	0	0	0	1
Diaphragmatic Hernia	0	1	0	0	0	0	1
Exomphalos	3	0	0	0	0	0	3
Circulatory Defect	0	0	0	1	0	0	1
Liver Defect	0	0	1	0	0	0	1
Mongols	0	1	1	0	0	2	4
Trisomy (5)	0	1	0	1	0	0	2
Hare Lip	1	0	3	2	3	1	10
Cleft Palate	1	1	2	0	2	1	7
Talipes	12	6	8	9	0	6	41
Extra Digits	0	0	0	0	0	3	3
Hypospadias	2	2	1	2	3	6	16
Web Digits	1	1	0	2	2	0	6
Lung Deformity	0	0	0	0	0	1	1
Eye Abnormality	1	0	0	0	0	0	1
Ear Abnormality	1	0	0	0	0	0	1
Angioma	0	1	0	0	1	0	2
Tumour of Face	0	0	0	0	1	0	1
Rare Blood Cord	0	1	0	0	0	0	1
Stenosis of Larynx) Diagnosed before leaving hospital	0	0	1	0	0	0	1
Plyoric Stenosis) Diagnosed before leaving hospital	0	0	1	0	0	0	1
Teratoma	0	0	0	1	2[(1)]	0	3[(1)]
Achondroplasia	0	0	0	1	2	0	3
Oxycephaly	0	0	0	0	1	0	1
Lymphangiestasia	0	0	0	1	0	0	1
Nasal Bone Defect	0	0	0	1	0	0	1
Other Joint Defects	5	3	3	1	0	2	14
Imperforate Anus	0	0	1	0	1	0	2
Total	42	26	48	32[(2)]	27[(2)]	38	215[(2)]
Number of Babies Affected	*37*	*24*	*29*	*27*	*22*	*28*	*167*
C.D.H. Hips Splint	29	12	9	4	11	9	74

Notes: (1) data provided by A Gibbs (2009) shows that the 'Henderson study' only reported 1 case of teratoma (in 1968-69) not 3 as shown in table above as reconstructed by O'Connor (2002).
(2) This original table, taken from O'Connor (2002) has two errors of addition. For 1968-69 the sum of all defects is 33 not 32 as shown and in 1969-70, 28 not 27. Allowing for the further correction provided by A Gibbs (2009) (see

note above) the 'correct' number of defects noted in 1969-70 is 26 and therefore the total number for the complete study is 213. No additional evidence has been provided to indicate that the number of affected babies (167) has changed. Assuming the correct number of teratoma cases is 1 not 3 as shown by O'Connor (2002), it is likely these will be single cases and therefore, the number of babies affected is 165. However, the total number of babies (167) affected as given by O'Connor (2002) has not been disputed by either A Gibbs (2009) or Carnachan (2002). Source: O'Connor 2002, Gibbs 2009.

The international studies of most relevance to New Plymouth are those relating to Seveso, Italy. An explosion at a trichlorophenol plant in Seveso, Italy in 1976 resulted in the highest TCDD exposure known in a human residential population.

A cytogenetic study in 1977 found no consistent evidence of chromosomal effects associated with TCDD exposure (Pesatori et al 2003). There was also no evidence of birth defects attributable to TCDD in 34 cases of abortion which occurred in 1976 after the incident and 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).

A meta-analysis of 22 studies of Agent Orange (50% 2,4-D and 50% 2,4,5-T) exposure in Vietnam shows an increased risk of birth defects (RR 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 percent (13 of 22) of the studies included have not been published in a peer-reviewed journal and 11 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 states:

> '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.'

AIMS

The current study arose from specific concerns about a possible link between the exposure to 2,4,5-T and the occurrence of birth defects in the New Plymouth area.

There were two aims. Firstly, to determine if the prevalence of birth defects in Westown Maternity Hospital (WMH) reported by Henderson (Table 1) and O'Connor (2002) were different from those reported in other New Zealand studies of birth defects. Secondly, to use data from the New Zealand Birth Defects Monitoring Programme (NZBDMP) to compare the rates of birth defects in the New Plymouth district with other areas in New Zealand during the period 1980-89.

SECTION I

Westown Maternity Hospital and Other New Zealand Studies of Birth Defects

Despite differences in ascertainment, time periods, and in some instances definitions, the prevalence rates from other New Zealand studies of birth defects provide a context and comparison for the rates reported from the Westown Maternity Hospital in New Plymouth during 1965-71 (the 'Henderson study' cited in Carnachan 2002 and O'Connor 2002 (Table 1).

Howie and Phillips (1970) conducted a study of birth defects among live births and stillbirths born in 1964-67 at the major metropolitan and perinatal referral centre of National Women's Hospital (NWH) in Auckland. Babies were routinely examined by hospital staff within the first 24 hours after birth and again before discharge at an average age, in term babies of eight days. About 40 percent were examined at followup clinics usually six weeks after discharge.

Buckfield (1973), reporting on the prevalence of birth defects among total births at Queen Mary Hospital (QMH) in Dunedin during 1967-70, acknowledged the study would have included some births to mothers referred antenatally to the hospital from other parts of the Otago province. The accuracy of the diagnosis and classification was believed to be high as the 'great majority of the babies were examined personally and the remainder by a paediatric registrar' (Buckfield 1973).

In the course of carrying out a cost benefit analysis of screening for spina bifida, Baker (1981) reported the prevalence of the NTDs, anencephaly and spina bifida, at NWH during 1968-77. Legge (1982) ascertained live and stillborn cases of NTDs and other defects of the central nervous system at Christchurch Women's Hospital (CWH) in 1970-75. Because CWH is also a

major referral centre, Legge adjusted the rates to account for the likely effect of admitting high risk maternity patients. Howie (cited in Baker 1981) had previously made a similar adjustment to his initial rates at NWH. No details of the adjustment methodology are available.

Hanify et al (1980, 1981a, 1981b) ascertained live and stillborn cases from the maternity registers of Northland hospitals during 1967-77. Babies born in hospitals outside Northland to Northland-domicile parents were excluded, but babies born in Northland to non-Northland parents were included.

Foster (1984) and Borman et al (1986, 1993) presented data from national studies based on a multiple source case ascertainment (eg, death and perinatal death certificates, hospital admissions, post-mortem reports). Foster (1979, 1982) previously reported early data from the New Zealand Birth Defect Monitoring System (NZBDMP), which was based on the reporting of cases with birth defects from the medical notification of birth scheme, established in 1975.

For the current study, all prevalence rates are per 1000 total births (ie, live births and stillbirths), except where otherwise noted (usually when stillbirths have not been included in the study). Where the original data were available from a study, 95% confidence intervals (95%CI) for the prevalence rates have been calculated. In some instances, rates have been estimated from the original data.

All Birth Defects

During the period 1965-70, 167 cases of birth defects among 5,392 births (both live births and stillbirths) were recorded at the WMH in New Plymouth. The prevalence rate of 30.97/1000 (95%CI 26.42-36.09), was significantly higher than the rates reported from the Dunedin referral centre of QMH (19.35/1000, 95%CI16.03-23.16) and across the hospitals in Northland (15.62/1000, 95%CI 14.04-17.32) and nationally for 1978 (27.62/1000, 95%CI 26.16-29.13) and the period 1980-89 (22.39/1000, 95%CI 21.96-22.83) (Table 2). The rate at WMH was not significantly different from the rate at NWH, the major maternity hospital and referral centre in Auckland, for 1964-67.

Table 2: The prevalence of birth defects in New Zealand

Place of Study	Years	Cases	Births	Rate/1,000	95% CI	
National Women's Hospital (1)	1964-67	387	16,103	24.03	21.65	26.61
Westown Maternity Hospital (2)	1965-71	167[(a)]	5,392	30.97	26.42	36.09
Northland (3)	1966-77	374	23,951	15.62	14.04	17.32
Queen Mary Hospital, Dunedin (4)	1967-70	119	6,151	19.35	16.03	23.16
New Zealand (5)	1978	1,440	52,137	27.62	26.16	29.13
New Zealand (6)	1980-89	11,298	504,567	22.39	21.96	22.83
National Women's Hospital – Maori (1)	1964-67	58	2,355	24.63	18.82	31.72
National Women's Hospital – NonMaori (1)	1964-67	313	12,920	24.23	21.57	27.12
Northland – Maori (3)	1966-77	144	7,587	18.98	16.00	22.37
Northland – nonMaori (3)	1966-77	228	16,253	14.03	12.24	16.00

Notes: (a) see note for Table 1 regarding the total number of possible cases.
Sources: (1) Howie and Phillips 1970; (2) Henderson study quoted in Carnachan 2002 and presented in O'Connor 2002; (3) Hanify 1980, 1981a, 1981b; (4) Buckfield 1973;(5) Foster 1984; (6) NZBDMP 1980-89.

Anencephaly

Henderson (Table 1) recorded 12 cases of anencephaly at WMH (2.23/1000, 95%CI 1.24-3.73), but O'Connor (2002) only identified 10 cases (1.85/1000, 95%CI 0.98-3.25). Irrespective, neither rate is significantly different from the rate reported from the other single hospital based studies at NWH (2.48/1000, 95%CI 1.80-3.36), QMH (1.30/1000, 95%CI 0.64-2.41), and CWH (1.16/1000, 95%CI 0.75-1.76) or across Northland (1.04/1000, 95%CI 0.69-1.52). All these rates, based on hospital data, are higher than the national rate ascertained from multiple sources in the late 1970s (Table 3).

Table 3: The prevalence of anencephaly in New Zealand

Place of study	Years	Cases	Births	Rate/1,000	95% CI	
National Women's Hospital (1)	1964-67	40[a]	16,103	2.48	1.80	3.36
Westown Maternity Hospital (2)	1965-71	12	5,392	2.23	1.24	3.73
Westown Maternity Hospital (3)	1965-71	10	5,392	1.85	0.98	3.25
Northland (4)	1966-77	25	23,951	1.04	0.69	1.52
Queen Mary Hospital, Dunedin (5)	1967-70	8	6,151	1.30	0.64	2.41
National Women's Hospital (6)	1968-77	[b]	[b]	1.93	[c]	[c]
National Women's Hospital (7)	1968-77	[b]	[b]	2.70	[c]	[c]
Christchurch Women's Hospital (8)	1970-75	21[d]	17,913	1.16	0.75	1.76
New Zealand (9)	1978	51	52,143	0.98	0.74	1.29
New Zealand (10)	1978-82	205	261,150	0.78	0.68	0.90
National Women's Hospital – Maori (1)	1964-67	4	2,355	1.70	0.65	3.86
National Women's Hospital – nonMaori (1)	1964-67	35	12,920	2.71	1.92	3.73
Northland – Maori (4)	1966-77	3	7,587	0.40	0.13	0.99
Northland – nonMaori (4)	1966-77	21	16,253	1.29	0.83	1.93
New Zealand – Maori (10)	1978-82	19	35,185[e]	0.54	0.34	0.82
New Zealand – nonMaori (10)	1978-82	186	226,829[e]	0.82	0.71	0.95

Notes: (a) Howie and Phillips reported 40 cases of anencephaly (table IV), but in their table VII only classified 4 as Maori and 35 as European; (b) original data not available; (c) could not be calculated; (d) estimated from original data; (e) some births had no ethnicity indicated.
Sources: (1) Howie and Phillips 1970; (2) Henderson study presented in O'Connor 2002; (3) O'Connor 2002, (4) Hanify et al 1980, 1981a, 1981b; (5) Buckfield 1973; (6) Howie 1978; (7) Baker 1981; (8) Legge 1982; (9) Borman et al 1986; (10) Borman and Cryer 1993.

Spina bifida

The rates of spina bifida at WMH reported by both Henderson (16 cases, rate of 2.97/1000, (95%CI 1.79-4.68), and O'Connor (2002) (14 spina bifida, rate of 2.60/1000, 95%CI 1.51-4.21), are significantly higher than the rate reported from NWH (Table 4). They are not significantly higher than the rates in other single hospital studies at QMH and CWH, or in the multiple hospital study across Northland.

.

Table 4: The prevalence of spina bifida in New Zealand

Place of study	Years	Cases	Births	Rate/1,000	95% CI	
National Women's Hospital (1)	1964-67	18	16,103	1.12	0.69	1.72
Westown Maternity Hospital (2)	1965-71	16	5,392	2.97	1.79	4.68
Westown Maternity Hospital (3)	1965-71	14	5,392	2.60	1.51	4.21
Northland (4)	1966-77	36	23,951	1.50	1.07	2.06
Queen Mary Hospital, Dunedin (5)	1967-70	10	6,151	1.63(a)	0.86	2.85
National Women's Hospital (6)	1968-77	(b)	(b)	0.93	(c)	(c)
National Women's Hospital (7)	1968-77	(b)	(b)	2.06	(c)	(c)
Christchurch Women's Hospital (8)	1970-75	58(d)	17,913	3.25	2.47	4.17
New Zealand (9)	1978	53	52,143	1.02	0.76	1.31
New Zealand (10)	1978-82	247	261,150	0.94	0.83	1.07
National Women's Hospital – Maori (1)	1964-67	1	2,355	0.42	0.09	1.66
National Women's Hospital – nonMaori (1)	1964-67	17	12,920	1.32	0.80	2.05
Northland – Maori (4)	1966-77	3	7587	0.40	0.13	0.99
Northland – nonMaori (4)	1966-77	32	16,253	1.97	1.37	2.75
New Zealand – Maori (10)	1978-82	18	35,185(e)	0.51	0.32	0.79
New Zealand – nonMaori (10)	1978-82	229	226,829(e)	1.01	0.88	1.15

Notes: (a) Buckfield (1973) reported the rate as 1.62; (b) original data not available; (c) could not be calculated; (d) estimated from original data; (e) some births had no ethnicity indicated
Sources: (1) Howie and Phillips 1970; (2) Henderson study presented in O'Connor 2002; (3) O'Connor 2002, (4) Hanify et al 1980, 1981a, 1981b; (5) Buckfield 1973; (6) Howie 1978; (7) Baker 1981; (8) Legge 1982; (9) Borman et al 1986; (10) Borman and Cryer 1993.

Neural tube defects

NTDs comprise the specific defects anencephaly and spina bifida, but the classification has not always been consistently applied (Borman and Cryer 1990).

The prevalence rates at WMH, despite Henderson reporting 28 cases and O'Connor finding only 24, are generally not significantly different from the rates reported from any of the other hospital studies in New Zealand. The only exception is that the rate for Henderson's study (5.19/1000, 95%CI 3.53-7.40) is significantly higher than the rate reported for multi-hospital ascertainment in Northland (2.55/1000, 95%CI 1.96-3.26). All hospital based rates are higher than the national rate for 1978-82 (1.73/1000, 95%CI 1.57-1.90) using a multiple source ascertainment (Table 5).

NonMaori have higher rates of NTDs than Maori (Table 5).

Table 5: The prevalence of neural tube defects in New Zealand

Place of study	Years	Cases	Births	Rate/1,000	95% CI	
National Women's Hospital (1)	1964-67	58	16,103	3.60	2.75	4.64
Westown Maternity Hospital (2)	1965-71	28	5,392	5.19	3.53	7.40
Westown Maternity Hospital (3)	1965-71	21[a]	5,392	3.89	2.50	5.83
Northland (4)	1966-77	61	23,951	2.55	1.96	3.26
Queen Mary Hospital, Dunedin (5)	1967-70	18	6,151	2.93[b]	1.81	4.51
Christchurch Women's Hospital (6)	1970-75	79[b]	17,913	4.51	3.50	5.49
New Zealand (7)	1978-82	452	261,150	1.73	1.57	1.90
National Women's Hospital – Maori (1)	1964-67	5	2,355	2.14	0.89	4.53
National Women's Hospital – nonMaori (1)	1964-67	52	12,920	4.02	3.03	5.25
Northland – Maori (4)	1966-77	6	7,587	0.79	0.35	1.59
Northland – nonMaori (4)	1966-77	53	16,253	3.26	2.46	4.25
New Zealand – Maori (7)	1978-82	37	35,185[c]	1.05	0.75	1.44
New Zealand – nonMaori (7)	1978-82	415	226,829[c]	1.83	1.65	2.02

Notes: (a) O'Connor (2002) recorded 21 NTD cases: 10 anencephaly, 14 spina bifida, with three having both defects; (b) calculated from original data; (c) some births had no ethnicity indicated.
Sources: (1) Howie and Phillips 1970; (2) Henderson study presented in O'Connor 2002; (3) O'Connor 2002, (4) Hanify et al 1980, 1981a, 1981b; (5) Buckfield 1973; (6) Legge 1982; (7) Borman and Cryer 1993.

Talipes

The reported rate of talipes at WMH during 1965-71 was 7.60/1000 (95%CI 5.52-10.24), significantly higher than that found at NWH (3.66/1000, 95%CI 2.81-4.71) and nationally in 1978 (4.32/1000, 95%CI 3.76-4.93) (table 6).

A number of studies have found Maori to have the highest prevalence of talipes with a rate reported between 6 to 7 per 1000 (Beals 1978; Cartlidge 1983, 1984; Chapman et al 2000). Although no ethnic specific data were available from the Henderson study, the rate at WMH (7.60/1000, 95%CI 5.52-10.24) is only slightly lower, but not significantly different from the Maori rates at NWH (8.14/1000, 95%CI 5.10-12.41) and Northland (8.96/1000, 95%CI 6.99-11.33).

Table 6: The prevalence of talipes in New Zealand

Place of study	Years	Cases	Births	Rate/1,000	95% CI	
National Women's Hospital (1)	1964-67	59	16,103	3.66	2.81	4.71
Westown Maternity Hospital (2)	1965-71	41	5,392	7.60	5.52	10.24
Northland (3)	1966-77	124	23,951	5.18	4.31	6.18
New Zealand (4)	1978	225	52,137	4.32	3.76	4.93
National Women's Hospital (1) – Maori	1964-67	19	2,335	8.14[(a)]	5.10	12.41
National Women's Hospital (1) – nonMaori	1964-67	38	12,920	2.94	2.11	4.00
Northland – Maori (3)	1966-77	68	7,587	8.96	6.99	11.33
Northland – nonMaori (3)	1966-77	55	16,253	3.38	2.57	4.39

Notes: (a) Howie & Phillips (1970) reported the rate as 8.07 (their table VII).
Sources: (1) Howie and Phillips 1970; (2) Henderson study presented in O'Connor 2002; (3) Hanify et al 1980, 1981a, 1981b; (4) Foster 1984.

Down syndrome

The rate of Down syndrome at WMH during 1965-71 (0.74/1000, 95%CI 0.28-1.68), was not significantly different from the rates at NWH (1.30/1000, 95%CI 0.84-1.95) and QMH in Dunedin (0.81/1000, 95%CI 0.34-1.72).

Based on followup from enrolments at schools, contact with medical superintendents at psychopedic and mental health hospitals, Morris (1971) estimated that the rate of surviving Down syndrome cases born between 1 January 1953 and 31 December 1960, was 0.73/1000 (95%CI 0.66-0.82). For the period 1979-81 the national rate for total births was little changed at 0.89/1000 (95%CI 0.34-1.06).

NonMaori have higher rates than Maori (Table 7).

Table 7: The prevalence of Down syndrome in New Zealand

Place of study	Years	Cases	Births	Rate/1,000	95% CI	
New Zealand (1)	1953-60	339[(a)]	462,590[(a)]	0.73[(a)]	0.66	0.82
National Women's Hospital (2)	1964-67	21	16,103	1.30	0.84	1.95
Westown Maternity Hospital (3)	1965-71	4	5,392	0.74	0.28	1.68
Northland (4)	1966-77	11	23,951	0.46	0.25	0.79
Queen Mary Hospital, Dunedin (5)	1967-70	5	6,151	0.81	0.34	1.72
New Zealand (6)	1978	37	52,137			
New Zealand (7)	1979-81	126	141,742	0.89	0.74	1.06
New Zealand – Maori (1)	1953-60	16[(a)]	51,237[(a)]	0.31[(a)]	0.19	0.49
New Zealand – nonMaori (1)	1953-60	323[(a)]	411,353[(a)]	0.79[(a)]	0.70	0.88
National Women's Hospital (2) – Maori	1964-67	0	2,335	-	-	-
National Women's Hospital (2) – nonMaori	1964-67	20	12,920	1.55	0.98	2.34
Northland – Maori (4)	1966-77	0	7,587	-	-	-
Northland – nonMaori (4)	1966-77	11	16,253	0.68	0.37	1.16

Note: (a) live births only.
Sources: (1) Morris 1971; (2) Howie and Phillips 1970; (3) Henderson study presented in O'Connor 2002; (4) Hanify et al 1980, 1981a, 1981b; (5) Buckfield 1973; (6) Foster 1984; (7) Foster 1982.

Congenital heart defects

Congenital heart defects comprise the largest single group of birth defects among live births. The rate at WMH during 1965-71 was 2.23/1000 births (95%CI 1.24-3.73) significantly lower than the rates in two studies at NWH (5.34/1000, 95%CI 4.21-6.71; 4.72, 95%CI 3.73-5.90), but similar to those in Northland (1.46/1000, 95%CI 1.03-2.01), and at QMH (2.93/1000, 95%CI 1.81-4.51). The 1978 New Zealand rate from a national register of birth defects was 3.50/1000 live births (95%CI 3.00-4.05) (Table 8).

There is little difference between the rates for Maori and nonMaori (Table 8).

Table 8: The prevalence of congenital heart defects in New Zealand

Place of study	Years	Cases	Births	Rate/1,000	95% CI	
National Women's Hospital (1)	1960-63	73[a]	13,653[a]	5.34[a]	4.21	6.71
National Women's Hospital (2)	1964-67	76[b]	16,103	4.72[b]	3.73	5.90
Westown Maternity Hospital (3)	1965-71	12	5,392	2.23	1.24	3.73
Northland (4)	1966-77	35	23,951	1.46	1.03	2.01
Queen Mary Hospital, Dunedin (5)	1967-70	18	6,151	2.93[c]	1.81	4.51
New Zealand (6)	1978	181[a]	51,777[a]	3.50[a]	3.00	4.05
National Women's Hospital (1) – Maori	1964-67	7	2,335	3.00	1.42	5.75
National Women's Hospital (1) – nonMaori	1964-67	20	12,920	1.55	0.98	2.34
Northland – Maori (3)	1966-77	11	7,587	1.45	0.79	2.48
Northland – nonMaori (3)	1966-77	23	16,253	1.42	0.93	2.08

Notes: (a) live births only; (b) Howie and Phillips (1970) reported 80 malformations among 76 babies; 4 babies had multiple congenital heart defects and the rate was 4.7 (see Howie and Phillips (1970), table IV; (c) Buckfield (1973) reported the rate as 2.92 in table 16 .
Sources: (1) Rowe et al 1981; (2) Howie and Phillips 1970; (5) Henderson study presented in O'Connor 2002;(4) Hanify et al 1980, 1981a, 1981b; (5) Buckfield 1973; (6) Borman et al 1987.

Congenital dislocation of the hip

The rates of congenital dislocation of the hip (CDH) recorded by Henderson at WMH (13.72/1000, 95%CI 10.81-17.20) and Hadlow (1988) among live births in New Plymouth, (16.33/1000, 95%CI 13.96-18.99) were three times higher than the rate reported from NWH (3.54/1000, 95%CI 2.70-4.57) and twice the national rate in 1978 (6.16/1000, 95%CI 5.49-6.88). Howie and Phillips (1970) found no significant difference in the rate of CDH between Maori and nonMaori at NWH (Table 9).

Table 9: The prevalence of congenital dislocation of the hip in New Zealand

Place of study	Years	Cases	Births	Rate/1,000	95% CI	
New Plymouth (1)	1976-85	172[a]	10,534[a]	16.33	13.96	18.99
National Women's Hospital (2)	1964-67	57	16,103	3.54	2.70	4.57
Westown Maternity Hospital (3)	1965-71	74[b]	5,392	13.72	10.81	17.20
New Zealand (4)	1978	321	52,137	6.16	5.49	6.88
National Women's Hospital (1) – Maori	1964-67	6	2,335	2.57[c]	1.15	5.15
National Women's Hospital (1) – nonMaori	1964-67	50	12,920	3.87	2.90	5.08

Notes: (a) live births only; (b) reported as 'CHD Hip Splints' – see Table 1;(c) Howie and Phillips (1970) give the rate as 2.55 (table VII).
Sources: (1) Hadlow 1988;(2) Howie and Phillips 1970; (3) Henderson study presented in O'Connor 2002; (4) Foster 1984

Facial clefts

Facial clefts comprise all cleft lip and cleft palate. At WMH the rate of facial clefts (3.15/1000, 95%CI 1.93-4.91) was high, but not significantly different from that at QMH in Dunedin (2.11/1000, 95%CI 1.21-3.48), NWH (1.68/1000, 95%CI 1.13-2.40), in Northland (1.54/1000, 95%CI 1.10-2.11), or in an extensively ascertained study in Auckland over 1960-76 (1.83/1000, 95%CI 1.65-2.02) (Table 10).

Maori have higher rates of facial clefts than nonMaori (Table 10).

Table 10: The prevalence of facial clefts in New Zealand

Place of study	Years	Cases	Births	Rate/1,000	95% CI	
Auckland (1)	1960-76	396[a]	216,836[b]	1.83	1.65	2.02
National Women's Hospital (2)	1964-67	32[c]	16,103	1.99	1.38	2.77
National Women's Hospital (2)	1964-67	27[d]	16,103	1.68	1.13	2.40
Westown Maternity Hospital (3)	1965-71	17[e]	5,392	3.15	1.93	4.91
Northland (4)	1966-77	37	23,951	1.54	1.10	2.11
Queen Mary Hospital, Dunedin (5)	1967-70	13	6,151	2.11	1.21	3.48
National Women's Hospital (1) – Maori	1964-67	7	2,335	3.00[f]	1.42	5.75
National Women's Hospital (1) – nonMaori	1964-67	20	12,920	1.55	0.98	2.34
Northland – Maori (3)	1966-77	19	7,587	2.50	1.57	3.82
Northland – nonMaori (3)	1966-77	16	16,253	0.98	0.59	1.55

Notes: (a) estimated from original data;(b) live births only; (c) in Howie and Phillips (1970) table IV, 32 cases are reported including 4 cases with Pierre-Robin syndrome; (d) in Howie and Phillips (1970) table VII 27 cases of facial clefts are reported, excluding the four cases with Pierre-Robin syndrome; the implication is that 1 case had no ethnicity; (e) includes 'hare lip' – 10 cases and 'cleft palate' – 7 cases; (f) the rate given by Howie and Phillips (1970) was 2.97.
Sources: (1) Chapman 1983; (2) Howie and Phillips 1970; (3) Henderson study presented in O'Connor 2002; (4) Hanify et al 1980, 1981a, 1981b; (5) Buckfield 1973

Hypospadias

Estimates of the number of male births had to be made because most of the studies did not specify the gender of the births. Assuming 50% of the births in Westown were male (the same proportion as at NWH), the rate of 5.95/1000 males (95%CI 3.57-9.36) was not statistically significantly different from that at NWH (2.60/1000, 95%CI 1.67-3.89) (Table 11). The WMH rate was, however, significantly higher than the rate in Northland (1.60/1000 males, 95%CI 0.99-2.47).

If the proportion of males in the WMH birth population was the same as in Northland (47%), the rate at WMH would be 6.31/1000 male births (95%CI 3.80-9.96), not significantly different from the NWH rate.

Table 11: The prevalence of hypospadias in New Zealand

Place of study	Years	Cases*	Births*	Rate/1,000*	95% CI	
National Women's Hospital (1)	1964-67	21	8,076[(a)]	2.60	1.67	3.89
Westown Maternity Hospital (2)	1965-71	16	2,696[(b)]	5.93	3.57	9.36
Westown Maternity Hospital (2)	1965-71	16	2,534[(c)]	6.31	3.80	9.96
Northland (3)	1966-77	18	11,250[(d)]	1.60	0.99	2.47
New Zealand (4)	1979-81	150	70,871[(d)]	2.12	1.79	2.49

Notes: (a) males only; (b) estimated at 50% male births;(c) estimated at 47% male births; (d) estimated from original data.
Sources: (1) Howie and Phillips 1970; (2) Henderson study presented in O'Connor 2002; (3) Hanify et al 1980, 1981a, 1981b; (4) Foster 1982.

Discussion

Comparison of rates from studies of birth defects is fraught with difficulty in interpretation (Borman and Cryer 1990). There are often differences in the methods of case ascertainment, definitions of the defects, and the period in which the data are collected. In the current study, Carnachan (2002) stated that the Henderson study 'recorded 48 out of the 167 birth deformities as neural-tube defects'. However, this included 12 anencephaly, 14 meningocele and two spina bifida, defects which are considered to be NTDs, but also 15 hydrocephaly and five microcephaly, which are not classified as NTDs (Table 1). In most epidemiological studies of NTDs, births with coincident anencephaly and spina bifida will be classified as anencephaly, and those with spina bifida and hydrocephaly, as spina bifida. However, in most of the New Zealand based studies there is no definition given as to the classification of the specific defects. Therefore, it is possible, for one child with anencephaly and spina bifida to have been recorded as having two specific defects rather than one (anencephaly).

Carnachan (2002) reported that 'between 1964 and 1971, the number of birth defects reported in New Plymouth nearly doubled'. However, the Henderson study showed that the defects reported at WMH were 42 in 1964-66, 26 in 1966-67, 48 in 1967-68, 32 in 1968-69, 27 in 1969-70 and 38 in 1970-71 (Table 1). In these respective periods, the number of 'affected' babies were 37, 24, 29, 27, 22, 28 for a total of 167 babies (Table 1).

The Henderson study (O'Connor 2002) includes some conditions which are not considered 'birth defects' such as 'tumour of face' (one case, 1969-70), 'rare blood cord' (one case, 1966-67), 'teratoma' (one case, 1967-68), and 'lymophangietasia' (one case, 1968-69) (Table 1). If it is assumed these defects are excluded from the overall number of infants reported with defects (167), and they each occurred in only one infant, the overall number of infants with defects in Westown is 163 and the rate is 30.23/1000 (95%CI 25.74-35.20). The prevalence rate based on 167 cases was 30.97/100 (95%CI

26.42-36.09). Therefore, a decrease of four cases does not alter the overall patterns of birth defect prevalence found in the comparison of the various studies.

The Henderson study (O'Connor (2002)) only gives the total number of babies (5,392) for 1965-71 and not the annual number of births. To estimate the possible number of births each year at WMH, the proportion at which the national births occurred each year was applied to the total WMH total of births (Table 12). The highest estimated prevalence rate of birth defects occurred in 1965-66 (42.28/1000, 95%CI 30.20-57.74) and the lowest in 1969-70 (24.26/1000, 95%CI 15.71-36.02). There was no statistically significant change in the prevalence rate over the entire period (Table 12).

Table 12: Estimated annual rate of cases with birth defects at Westown Maternity Hospital, 1965-71

Years	Estimated births	Number of cases*	Estimated rate/1,000	95% CI	
1965-66	875	37	42.28	30.20	57.74
1966-67	882	24	27.20	17.94	39.78
1967-68	898	29	32.30	22.11	45.78
1968-69	907	27	29.75	20.09	42.66
1969-70	907	22	24.26	15.71	36.02
1970-71	922	28	30.36	20.64	43.27
1965-71	*5392**	*137*	*30.97*	*26.42*	*36.09*

** as per Table 1*

Although there were differences in the numbers of NTDs recorded in the Henderson study (28, Table 1) and those found by O'Connor (2002), 24, both show a similar pattern of rates over 1965-71 (Table 13). The highest rate occurred in 1967-68 and the lowest in the preceding year. However, because of the small numbers involved (and the accompanying wide confidence intervals), no discernible trend is evident in the estimated rates.

Table 13: Estimated annual rate of neural tube defects at Westown Maternity Hospital, 1965-71

	Henderson study (see table 1)				O'Connor (2002)			
Years	Cases	Rate/1000[(a)]	95% CI		Cases	Rate/1000[(a)]	95% CI	
1965-66	5	5.71	2.39	12.09	4	4.57	1.75	10.38
1966-67	0	0.00	0.02	0.00	0	0.00	0.02	0.00
1967-68	9	10.03	5.14	18.02	7	7.80	3.68	14.97
1968-69	5	5.51	2.30	11.66	6	6.61	2.96	13.25
1969-70	2	2.21	0.63	6.43	1	1.10	0.24	4.31
1970-71	7	7.59	3.59	14.57	6	6.51	2.91	13.04
1965-71	*28*	*5.19*	*3.53*	*7.40*	*24*	*4.45*	*2.94*	*6.51*

Note: (a) rates based on estimated annual number of births.

The estimated rate of talipes was highest in the first period with 12 defects reported. There is some suggestion of a decline in the rate over time (Table14).

Table 14: Estimated annual rate of talipes at Westown Maternity Hospital, 1965-71

Years	Cases	Rate/1,000[(a)]	95% CI	
1965-66	12	13.71	7.66	23.01
1966-67	6	6.80	3.04	13.63
1967-68	8	8.91	4.40	16.51
1968-69	9	9.92	5.09	17.82
1969-70	0	0.00	0.02	0.00
1970-71	6	6.51	2.91	13.04
1965-71	*41*	*7.60*	*5.52*	*10.24*

Note: (a) rates based on estimated annual number of births.

The rate of congenital dislocation of hips was highest in 1965-66, but declined thereafter (Table 15).

Table 15: Estimated annual rate of congenital dislocation of hips at Westown Maternity Hospital, 1965-71

Years	Cases	Rate/1,000[(a)]	95% CI	
1965-66	29	33.13	22.68	46.96
1966-67	12	13.60	7.60	22.82
1967-68	9	10.03	5.14	18.02
1968-69	4	4.41	1.69	10.01
1969-70	11	12.13	6.61	20.76
1970-71	9	9.76	5.01	17.54
1965-71	*74*	*13.72*	*10.81*	*17.20*

Note: (a) rates based on estimated annual number of births.

Assuming that annually throughout 1965-71, 50% of all the births (5,392) were male, the highest rate of hypospadias occurred in 1970-71 (13.01/1000 male births, 95%CI 5.82-26.08) (Table 16). It was not significantly different from the rate in other years.

Table 16: Estimated annual rate of hypospadias at Westown Maternity Hospital, 1965-71 (assuming 50% of all births were males)

Years	Cases	Rate/1,000[(a)]	95% CI	
1965-66	2	4.57	1.30	13.33
1966-67	2	4.53	1.29	13.22
1967-68	1	2.23	0.48	8.72
1968-69	2	4.41	1.25	12.86
1969-70	3	6.62	2.24	16.57
1970-71	6	13.01	5.82	26.08
1965-71	*16*	*5.93*	*3.57*	*9.36*

Note: (a) rates based on estimated annual number of births.

The form of case ascertainment remains a fundamental issue in comparing rates from different studies. The rate reported from hospital based studies, which include most of the New Zealand studies, will depend to the extent to which mothers with complications of pregnancy and other features associated with a higher risk of a baby having a birth defect are preferentially admitted (Borman and Cryer 1990). The study at QMH by Buckfield (1973) included infants both at the hospital as well as mothers referred antenatally from other parts of the province. The rates for anencephaly and spina bifida (Tables 3 and 4) in the Christchurch study by Legge (1982) were adjusted to allow for high-risk maternity patients who would not normally have been admitted to CWH. For the same reason, the rates originally reported by Howie and Phillips (1970) for these two defects (2.48/1000 and 1.12/1000 respectively) (Tables 3 and 4), were subsequently recalculated as 1.93/1000 and 0.93/1000 respectively (Tables 3 and 4) (Baker 1981).

WMH was the major maternity hospital in New Plymouth and therefore, potentially, subject to the same ascertainment biases as other hospital based studies. The Henderson study reported 12 cases of anencephaly and 16 of spina bifida, but in a subsequent search of ward notes, O'Connor identified 10 cases of anencephaly and 14 of spina bifida (Table 13).

Despite the major defects being easily recognised and recorded at, or shortly after birth, they are often subject to some degree of underreporting. Prevalence rates are related to the diligence of the ascertainment and the number of sources used in the ascertainment. Consistently, epidemiological studies of birth defects based on case ascertainment from multiple sources have the greatest validity. Of the studies noted in this paper, Foster (1984), Borman et al (1986, 1987), Borman and Cryer (1993), and Chapman (1983) ascertained cases from multiple sources.

Prevalence rates of birth defects will also be affected by the exclusion of stillbirths. The presence of anencephaly is incompatible with life and therefore few cases are live born. Therefore excluding stillbirths will have a major impact on the prevalence rate. Conversely, the majority of spina bifida cases are born live. In a number of studies, the assumption is made that all births (still and live) are included. Morris (1971), Chapman (1983) and Borman et al (1987) only included live births in their respective studies of Down syndrome, facial clefts and congenital heart disease. It is also possible that not all defects will be diagnosed and recorded on a medical record, but rather a broad 'defects' or an indistinct category will be included. For example, the Henderson data (Table 1) include some imprecise categories – 'liver defect', 'other joint defects', eye abnormality', 'ear abnormality', 'extra digits', 'web digits'.

In some situations, a fetus may be too macerated for a specific defect to be diagnosed, even at post-mortem.

Summary

- The overall rate of birth defects recorded at the WMH during 1965-71 was significantly higher that the rates reported from Northland (1966-77) and QMH in Dunedin (1967-70). It was not, however, significantly different from the rate found at NWH in Auckland during 1964-67.

- The rate of anencephaly at WMH was not significantly different from the rates reported in other hospital based studies.

- The rate of spina bifida at WMH was higher than the rate at NWH in Auckland, but not the rates in the other studies.

- The rate of neural tube defects at WMH in both the studies by Henderson and O'Connor was not significantly different from the rates reported in New Zealand studies.

- The rate of talipes at WMH was significantly higher than the rate reported from NWH.

- The rates of Down syndrome and facial clefts at WMH were not significantly different from the rates in other New Zealand studies.

- The rate of congenital heart defects at WMH was lower than at NWH.

- The rate of congenital dislocation of the hip at WMH was more than three times higher than the rate at NWH.

- The estimated rate of hypospadias at WMH was higher, but not significantly different from the rate at NWH. The WMH rate was significantly higher than the rate in Northland.

SECTION II

New Zealand Birth Defects Monitoring Programme (NZBDMP)

Background

In 1969 an ad hoc committee of the Medical Research Council (MRC) of New Zealand advocated for ‘immediate setting up of a system for monitoring “congenital defects”.

The Obstetric Regulations (1975) provided the mechanism to establish such a system. In accordance with these Regulations a ’Medical Notification of Birth or Stillbirth’ (H661) form (Appendix 2) was to be completed and signed as soon as practicable after a stillbirth (at least 28 weeks gestation), or the death of an infant in hospital, or before, but as close as practicable to, the discharge or transfer of an infant from hospital.

The NZBDMP, previously known as the Congenital Anomaly Monitoring Programme, commenced in 1977 to ’quickly provide public health administrators with information about temporal and spatial clusterings of congenital anomalies in the newborn so that appropriate action can be taken’ (Foster, 1979).

The H661 forms were sent to the District Medical Officer of Health of the Health District in which the mother resided. Births of infants in places other than hospitals were also notified. The forms were forwarded to the then Department of Health’s National Health Statistics Centre (NHSC), the predecessor of the New Zealand Health Information Service (NZHIS) for processing and tabulating. Monthly tabulations were sent to all Hospital Districts.

In September 1975, the NZ Paediatric Society and the Department of Health agreed to establish a Congenital Anomaly Register (CAR). Ascertainment was from multiple sources: the monitoring programme; the 'Medical Certificate of Causes of Fetal and Neonatal Death' form; the 'Medical Certificate of Causes of Death' form, post-mortem reports, discharge notification forms from public and private hospitals, and reports on the medical examination of infants at nine months of age. However, data was only collected on the 1978 birth cohort.

The Obstetric Regulations were expected to ensure a high case ascertainment and therefore provide a valid estimate of the prevalence of birth defects in New Zealand. It was subsequently found that the reporting of cases of birth defects by the H661 form varied by month, Area Health Board (comprising one or more health districts) and year. At times the national level was higher or as low as 70% of the total number of births in a health district (the number of births in each hospital was also being reported).

The H661 was discontinued in late 1991 as the mechanism for the monitoring of birth defects. Since 1992, the NZBDMP has ascertainment cases from births in, or those requiring treatment for a birth defect, in a public hospital.

The NZBDMP has been a member of the International Clearinghouse for Birth Defects Surveillance and Research (ICBDSR), formerly the International Clearinghouse for Birth Defects Monitoring Systems, since 1977. New Zealand data are included in the ICBDSR Annual Report.

Data

The earliest data available from NZBDMP are for the period 1980-89. Data from the existing H661 forms were transferred to a computer database and have been used in this study. These data are from the same source as reported in the regular NZBDMP released during 1980-89.

The data were available by district (either Health Districts or Area Health Boards as there were a number of boundary changes over the period – see Appendix 3) and for the 11 major birth defects which are monitored internationally (Table 17) by the ICBDSR and selected primarily because of the impact on infant health and their high diagnostic validity.

Table 17: Specific birth defects monitored by the NZBDMP, 1980-89

Anencephaly
Spina bifida
Hydrocephaly
Cleft palate
Cleft lip
Oesophageal atresia or stenosis
Anorectal atresia or stenosis
Hypospadias
Limb reduction deformity
Omphalocele
Down syndrome

Birth defects over time

As noted previously (Elwood et al 1992, Borman et al 1986, Borman and Cryer 1993), the rate of NTDs, anencephaly and spina bifida declined over 1980-89 (Table 18). The rate of hydrocephaly and omphalocele also declined, while there were slight increases in the rate of cleft palate and Down syndrome. There was little change in the rate of oesophageal atresia, rectal and anal atresia, hypospadias, and limb reduction defects.

Birth defects by area

During 1980-89, the New Plymouth district consistently had the highest reported rates of birth defects. Significantly high rates were also reported in the Whangarei/Northland, Auckland, South Auckland, and Palmerston North Health districts (Table 19). Conversely, there were significantly low rates reported from Rotorua, Wanganui, Hutt and Wellington, West Coast, Christchurch and Dunedin districts. Table 19 also shows fluctuations in annual rates at the district level, which may be attributable to reporting, diagnosis, prevalence, or incidence.

Table 18: Rates of major birth defects, per 1000 live birth and stillbirth notifications, reported to the NZBDMP, 1980-89

Year	Anencephaly	Spina bifida	Hydrocephaly	Cleft palate	Cleft lip	Oesophageal atresia	Rectal and anal atresia	Hypospadias	Limb reduction deformity	Omphalocele	Down syndrome
1980	0.67	1.08	0.77	0.59	1.03	0.18	0.15	1.24	0.26	0.39	0.80
1981	0.58	1.17	0.38	0.52	0.80	0.10	0.22	1.02	0.38	0.18	0.96
1982	0.58	0.91	0.35	0.69	0.85	0.19	0.27	1.04	0.37	0.27	0.62
1983	0.46	1.19	0.38	0.55	0.65	0.18	0.34	1.84	0.48	0.24	0.85
1984	0.28	0.98	0.37	0.70	1.00	0.15	0.15	1.37	0.33	0.28	1.07
1985	0.35	0.71	0.25	0.60	0.85	0.23	0.19	1.39	0.48	0.21	0.98
1986	0.34	0.70	0.44	0.76	1.00	0.23	0.28	1.34	0.30	0.21	0.87
1987	0.33	0.87	0.22	0.85	0.67	0.09	0.30	1.58	0.24	0.26	0.80
1988	0.18	0.46	0.28	0.76	0.74	0.23	0.19	0.99	0.23	0.12	0.85
1989	0.19	0.53	0.37	0.64	0.90	0.09	0.18	1.28	0.34	0.18	1.03
1980-89	*0.38*	*0.84*	*0.37*	*0.67*	*0.84*	*0.17*	*0.23*	*1.31*	*0.34*	*0.23*	*0.89*

Source: NZBDMP

Table 19: Rates of birth defects, per 1000 live birth and stillbirth notifications, reported to the NZBDMP, by District, 1980-89

District	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1980-89	95% CI	
Whangarei/Northland	28.46	25.59	27.36	23.76	24.03	17.78	25.94	18.55	18.05	49.03	25.89	23.69	28.25
Takapuna	31.45	22.75	14.81	19.27	14.18	26.84	16.62	25.12	17.40	19.86	21.50	19.58	23.56
Auckland	22.31	25.43	28.84	26.76	20.98	28.45	27.44	27.18	23.39	29.46	26.33	25.20	27.48
South Auckland	20.61	22.96	27.29	31.03	32.31	28.34	34.30	19.40	9.05	10.06	26.35	24.55	28.24
Hamilton	32.20	26.76	25.12	24.42	26.43	18.82	23.03	16.86	18.73	19.01	22.72	21.34	24.17
Rotorua	19.05	17.79	15.75	22.48	21.17	17.09	16.10	10.21	17.51	17.84	17.35	15.94	18.86
Gisborne	20.83	19.34	17.18	25.32	27.50	27.80	19.84	25.88	22.89	29.51	23.60	20.84	26.63
Napier	21.26	23.14	20.97	21.93	26.69	25.30	24.83	24.56	25.31	27.04	24.21	22.13	26.43
New Plymouth	29.55	43.02	33.41	36.73	39.14	32.26	35.51	32.69	38.75	29.49	35.08	32.30	38.03
Wanganui	15.24	14.81	21.43	24.54	28.15	16.89	20.41	23.41	10.63	15.60	19.08	16.80	21.58
Palmerston North	30.56	24.17	16.86	25.41	20.09	22.43	22.94	28.40	29.16	30.46	25.15	23.11	27.32
Hutt	20.92	15.24	17.00	16.19	15.07	18.88	17.88	16.72	17.81		17.29	15.67	19.04
Wellington	21.43	13.17	19.30	12.45	14.07	17.13	15.49	19.28	22.46		17.04	15.49	18.69
Wellington Region									10.83	22.31	19.96	17.06	23.22
West Coast									18.35	10.61	11.70	6.01	21.03
Nelson	23.08	28.08	18.70	19.03	24.43	20.41	16.11	16.68	17.41	20.03	20.92	18.67	23.37
Christchurch	15.16	17.61	16.77	15.29	13.85	19.06	12.95	17.28	14.38	15.01	15.68	14.55	16.87
Timaru	20.05	28.23	27.13	26.84	13.27	26.10	30.74	28.41	22.01	22.35	24.83	22.10	27.80
Dunedin	28.82	17.60	21.54	17.20	14.07	18.86	18.30	21.99	19.86	20.96	19.51	17.55	21.63
Invercargill	23.11	14.07	25.13	24.70	25.40	22.17	26.50	22.83	23.35	28.03	23.55	21.34	25.93
New Zealand	*23.34*	*21.94*	*22.36*	*22.89*	*21.90*	*22.83*	*22.58*	*21.60*	*20.53*	*24.16*	*22.39*	*21.96*	*22.83*

Source: NZBDMP

Throughout 1980-89, the regular reports from the NZBDMP specifically mentioned and provided further details about the high rates of birth defect notifications from the New Plymouth district (Appendix 4). During the same period, the NZBDMP investigated a number of so-called clusters or implemented enhanced monitoring of birth defects in the various areas of the country (Appendix 5). Although no possible aetiological factor was identified in the high rates notified from New Plymouth, there was a suggestion there might be a diagnostic bias in the data being reported.

The NZBDMP stated in the April-June 1986 report: 'In the past, districts reported as having rates more than 20 percent above the national average have frequently fallen into this category due to a high number of congenital dislocated hips or talipes. As procedures for checking infants and criteria for diagnosis for these two conditions may vary between hospitals district data were also presented excluding cases with these conditions. As a result the national rate in this quarter [April-June 1986] for all defects was 24/1000 completed notifications (310 cases), compared to 15/1000 (189 cases), excluding congenital dislocated hips and talipes.'

Subsequent quarterly reports from the NZBDMP presented tables of all birth defects reported and also the number excluding congenital dislocated hips and talipes.

Earlier, Hanify et al (1980) observed 'the period 1966 to 1977 had seen a dramatic rise in the diagnosis of dislocated and dislocatable hip (DDH) in Northland. Over the first half of the period the rate of diagnosis was 0.4 per thousand births, and over the second half 6.1 per thousand births. We tentatively associated this rise with the more general introduction of the Ortolani click technique of DDH diagnosis.' Accordingly, DDH was excluded from most of their subsequent analysis.

To investigate the possible impact of diagnostic bias in the data from New Plymouth, the NZBDMP subsequently tabulated data separately for births with 'congenital dislocation of hips and talipes'. In 1986-89, the reporting rate of all

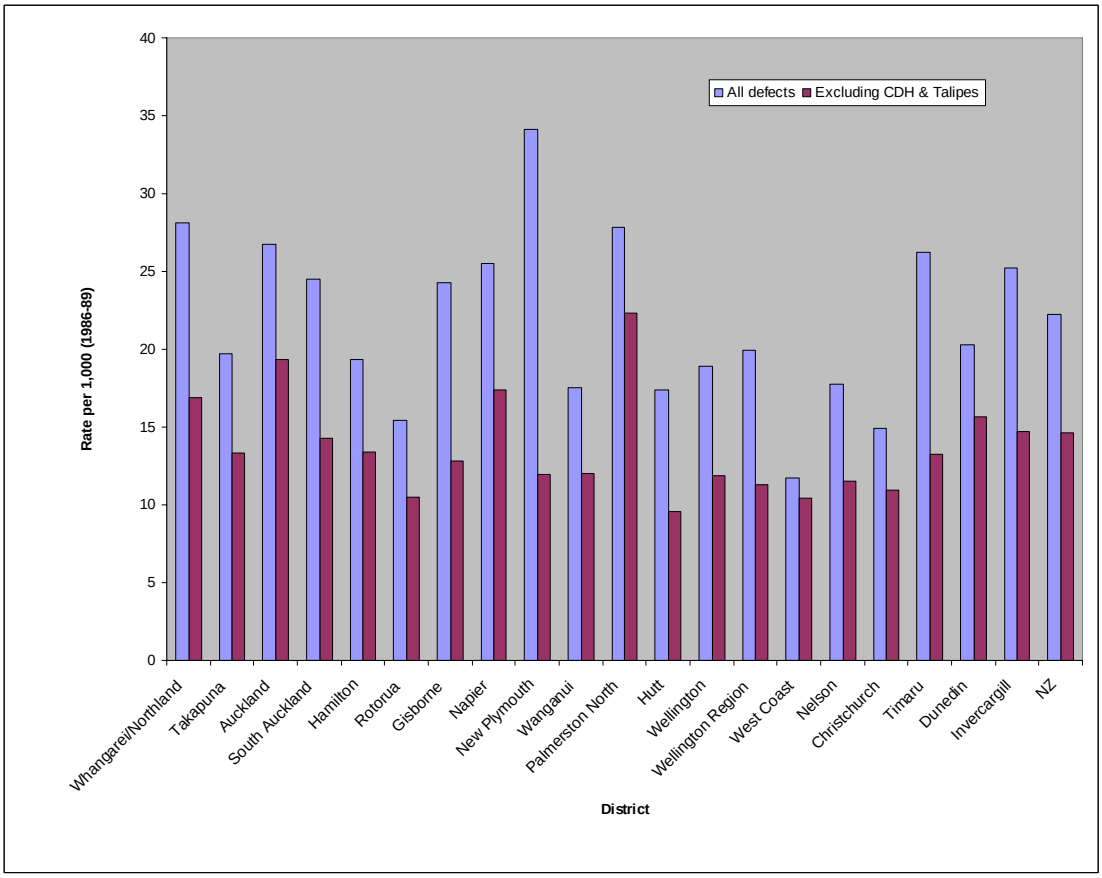
birth defects was significantly higher than the national average in Whangarei/Northland, Auckland, New Plymouth (the highest rate) and Palmerston North districts (Table 20). The rates in Hamilton, Rotorua, Wanganui, West Coast and Christchurch districts were significantly lower than the New Zealand rate. When cases of congenital dislocation of hips and talipes were excluded, the rates remained significantly high in Whangarei/Northland, Auckland, and Palmerston North districts. The rate in the New Plymouth district (11.92 per 1000 births) was not significantly different from the national average (14.67/1000). Rates in Rotorua, Hutt, West Coast and Christchurch district remained significantly low (Figure 1).

Table 20: Rates of birth defects, and excluding congenital dislocation of hip (CDH) and talipes, per 1000 live birth and stillbirth notifications, by District, April 1986 - December 1989

	All defects			Excluding congenital dislocation of hip & talipes		
District	Rate/1,000	95% CI		Rate/1,000	95% CI	
Whangarei/Northland	28.08	24.70	31.79	16.89	14.25	19.88
Takapuna	19.71	16.78	23.00	13.34	10.91	16.16
Auckland	26.75	25.24	28.33	19.37	18.05	20.75
South Auckland	24.47	21.18	28.12	14.30	11.70	17.31
Hamilton	19.37	17.49	21.39	13.42	11.83	15.16
Rotorua	15.47	13.53	17.62	10.50	8.88	12.34
Gisborne	24.28	19.87	29.39	12.85	9.67	16.77
Napier	25.48	22.31	28.96	17.42	14.77	20.43
New Plymouth	34.15	29.99	38.73	11.92	9.51	14.78
Wanganui	17.54	14.16	21.49	12.00	9.19	15.42
Palmerston North	27.86	24.61	31.42	22.30	19.34	25.59
Hutt	17.43	14.58	20.67	9.57	7.45	12.12
Wellington	18.94	16.31	21.89	11.89	9.77	14.34
Wellington Region	19.96	17.06	23.22	11.32	9.19	13.81
West Coast	11.70	6.01	21.03	10.40	5.14	19.27
Nelson	17.76	14.19	21.96	11.54	8.69	15.06
Christchurch	14.91	13.34	16.63	10.92	9.54	12.43
Timaru	26.23	21.71	31.42	13.25	10.07	17.13
Dunedin	20.32	17.41	23.58	15.68	13.08	18.66
Invercargill	25.23	21.71	29.16	14.73	12.02	17.88
New Zealand	*22.22*	*21.57*	*22.88*	*14.67*	*14.14*	*15.23*

Source: NZBDMP

Figure 1: Rate of birth defects, per 1,000 notifications, by Districts, April 1986-December 1989



Source: NZBDMP

Winder (2004a, 2004b) reported that in the Taranaki region, an orthopaedic surgeon Victor Hadlow, has been various described as a 'Man on a 'hip' mission and 'Taranaki's Victor of Bones'. Since 1964, Hadlow had 'dedicated himself to ensuring every baby born in Taranaki Base Hospital's maternity unit has been, and will continue to, be tested for displaced hips' (Winder 2004c).

Hadlow remarked that 'Taranaki has always had the highest number of bone specialists per head of population in New Zealand' (Winder 2004a). A previous study by Hadlow (1988) found that the rate of congenital dislocation of the hip was more than three times the rate at NWH (Table 9).

Summary

- The rate of birth defects in New Plymouth was consistently high during 1980-89. However, significantly high rates were also reported in the Whangarei/Northland, Auckland, South Auckland, and Palmerston North Health districts.
- In 1986-89, the reporting rate of all birth defects was highest in New Plymouth, but also significantly higher than the national average in Whangarei/Northland, Auckland, and Palmerston North districts.
- The rate in New Plymouth in 1986-89 was not significantly different from the national average if cases of congenital dislocation of hips and talipes were excluded.
- There was no evidence from these data, of an increase in the rate of birth defects in the New Plymouth district following the accident at the IWD plant in April 1986.

CONCLUSION

The data in this study show no evidence of increased prevalence rates of birth defects in the New Plymouth population that could be attributable to dioxin. However, current data limitations mean the possibility of a small increased risk cannot be excluded. These results are consistent with findings from other international population studies.

REFERENCES

Baker PA. 1981. Cost benefit analysis of screening spina bifida. New Zealand Medical Journal 93:386-9.

Beals RK. 1978. Club foot in the Maori: a genetic study of 50 kindreds. New Zealand Medical Journal 88:144-6.

Borman B, Chapman CJ, Howard JK, et al. 1987. Using a national register for the epidemiological study of congenital heart defects. New Zealand Medical Journal 100:404-6.

Borman B, Cryer C. 1990. Fallacies of the international and national comparisons of disease occurrence in the epidemiology of neural tube defects. Teratology 42:405-12.

Borman B, Cryer C. 1993. The prevalence of anencephalus and spina bifida in New Zealand. Journal of Paediatrics and Child Health 29:282-8.

Borman B, Howard JK, Chapman CJ. 1986. Secular trends in the prevalence of anencephalus in New Zealand. New Zealand Medical Journal 99:183-5.

Borman B, Smith AH, Howard JK 1986. Risk factors in the prevalence of anencephalus and spina bifida in New Zealand. Teratology 33:221-30.

Brinkman GL, Matthews REF, Earl WB. 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. Wellington: Ministerial Committee of Inquiry.

Brinkman GL, Matthews REF, Earl WB. 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. Wellington: Ministerial Committee of Inquiry.

Buckfield P. 1973. Major congenital faults in newborn infants: a pilot study in New Zealand. New Zealand Medical Journal 78:195-204.

Carnachan H. 2002. Toxic Waste. Investigate. April 2002:28-37

Cartlidge I. 1983. Club foot in the Polynesian: an epidemiological survey. New Zealand Medical Journal 96:515-7.

Cartlidge I. 1984. Observations on the epidemiology of club foot in Polynesian and Caucasian populations. Journal of Medical Genetics 21:290-2.

Chapman CJ. 1983. Ethnic differences in the incidence of cleft lip and/or cleft palate in Auckland, 1960-1976. New Zealand Medical Journal 96:327-9.

Chapman CJ, Stott S, Port RV, et al. 2000. Genetics of club foot in Maori and Pacific People. Journal of Medical Genetics 37:680-3.

Department of Health. 1997a. 2,4,5-T, spina bifida, and after. New Zealand Medical Journal 86:99-100.

Department of Health. 1997b. 2,4,5-T and Human Birth Defects. Wellington: Department of Health.

Department of Health. 1980a. A Review to Determine Whether the Health of Employees has been Adversely Affected by their Association with the Manufacture of 2,4,5-T. Wellington: Department of Health.

Department of Health. 1980b. Report to the Minister of Health of an investigation into allegations of an association between human congenital defects and 2,4,5-T spraying in and around Te Kuiti. New Zealand Medical Journal 91:314–5.

Elwood JM, Little J, Elwood JH. 1992. Epidemiology and Control of Neural Tube Defects. Oxford: Oxford University Press.

Foster FH.1979. Congenital anomaly monitoring system in New Zealand. New Zealand Medical Journal 90:509-10.

Foster FH.1982. Congenital anomaly monitoring in New Zealand, 1979-81. New Zealand Medical Journal 95:780-1.

Foster FH. 1984. The New Zealand Congenital Anomaly Register. New Zealand Medical Journal 97:150-3.

Gibbs A. 2009. Personal communication.

Hadlow V. 1988. Neonatal screening for congenital dislocation of the hip: a prospective 21-year survey. Journal Bone Joint Surgery (British) 70-B:740-3.

Hanify JA, Metcalf P, Nobbs CL et al. 1980. Congenital malformations in the newborn in Northland: 1966-1977. New Zealand Medical Journal 92:245-8.

Hanify JA, Metcalf P, Nobbs CL et al. 1981a. Aerial spraying of 2,4,5-T and human birth malformations: final report of an epidemiological study carried out in the Northland region of New Zealand. Auckland: Northland Birth Defects Survey.

Hanify JA, Metcalf P, Nobbs CL et al. 1981b. Aerial spraying of 2,4,5-T and human birth malformations: an epidemiological investigation. Science 212:349-51.

Howie RN. 1978. Personal communication. Cited in Baker 1981.

Howie RN, Phillips LI. 1970. Congenital malformations in the newborn: a survey at the National Women's Hospital, 1964-67. New Zealand Medical Journal 71:65-71.

Institute of Medicine. 2009. Veterans and Agent Orange: Update 2008. Washington, DC: The National Academies Press www.nap.edu/catalog/12662.html

Legge M. 1982. Incidence of central nervous system abnormalities in new born infants born at the Christchurch Women's Hospital between 1970 and 1975. New Zealand Medical Journal 95:417-8.

Morris R. 1971. Down's syndrome in New Zealand. New Zealand Medical Journal 73:195-8.

Ngo AD, Taylor R, Roberts CL, et al. 2006. Association between Agent Orange and birth defects: systematic review and meta-analysis. International Journal of Epidemiology 35:1220-30.

O'Connor PM. 2001. Rates of reported illness in Paritutu and Moturoa. New Plymouth: Taranaki District Health Board.

O'Connor P. 2002. Neural Tube Defects At Westown Maternity Hospital, 1965-72: A Report to the Taranaki District Health Board. New Plymouth: Taranaki District Health Board.

Pesatori AC, Consonni D, Bachetti S, et al. 2003. Short- and long-term morbidity and mortality in the population exposed to dioxin after the "Seveso accident". Industrial Health 41:127-38.

Rowe RD, Freedom RM, Mehrizi A, et al. 1981. The Neonate with Congenital Heart Disease. 2nd ed. WB Saunders: Philadelphia.

Sare WM, Forbes PI. 1972. Possible dysmorphogenic effects of an agricultural chemical: 2,4,5-T. New Zealand Medical Journal 75:37-8.

Schecter A, Constable JD. 2006. Commentary: Agent Orange and birth defects in Vietnam. International Journal of Epidemiology 35:1230-2.

Smith AH, Matheson DP, Fisher DO, et al. 1981. Preliminary report of reproductive outcomes among pesticide applicators using 2,4,5-T. New Zealand Medical Journal 93:177-9.

Smith AH, Fisher DO, Pearce NE. 1982. Congenital defects and miscarriages among New Zealand 2,4,5-T sprayers. Archives of Environmental Health 37:197-200.

Winder V. 2004a. Science and Medicine - Man on a 'hip' mission. Puke Ariki (http://www.pukeariki.com/en/stories/scienceAndMedicine/hadlow.htm)

Winder V. 2004b. Taranaki's Victor of Bones. Puke Ariki (http://www.pukeariki.com/en/stories/scienceAndMedicine/hadlow_bones.htm)

Winder V. 2004c. The Important of Hip Checks. Puke Ariki (http://www.pukeariki.com/en/stories/scienceAndMedicine/hadlow_hipchk.htm)

Appendix 1

Accident at the IWD plant in April 1986

In April 1986 there was an accident at the IWD plant which resulted in the uncontrolled release to the atmosphere of TCDD from a reactor producing trichlorophenol, the main intermediate in the 2,4,5-T production process.

Data from the NZBMP was used to investigate the impact of this incident on the rates of birth defects in New Plymouth. Usually teratogenic effects of chemicals (eg, thalidomide, valproic acid) are manifest in specific birth defects, categories or syndromes of defects. Data from the NZBMDP for the 1980s are only available for all birth defects and not for specific birth defects. Therefore, any effect of the accident on specific birth defects or categories of birth defects will be masked by considering the total birth defect category.

The exposed period was taken to be October 1986-March 1987 (the data in the NZBDMP is only available quarterly), which included women potentially exposed in their first trimester and giving birth nine months later. The control period was 1980-September 1986. Table 21 shows that the rate of birth defects in New Plymouth in the ‘exposed’ period was 31.64 (95%CI 21.51-45.09) lower than the rate during the control period (35.26/1000 notifications, 95%CI 32.41-38.30).

Table 21: Rates of birth defects, per 1000 live birth and stillbirth notifications, by District

	Control period 1980-September 1986				Exposed period October 1986-March 1987			
	Cases	Rate/1000	95%CI		Cases	Rate/1000	95%CI	
Whangarei/Northland	514	26.39	24.11	28.84	19	17.12	10.74	26.11
Takapuna	476	22.11	20.12	24.24	8	8.18	4.04	15.15
Auckland	2123	26.41	25.26	27.60	106	24.80	20.31	29.99
South Auckland	792	26.41	24.55	28.37	52	25.39	19.11	33.13
Hamilton	1023	22.97	21.54	24.47	47	18.33	13.60	24.23
Rotorua	568	17.85	16.37	19.42	16	8.76	5.28	13.82
Gisborne	258	23.63	20.79	26.74	14	23.14	13.48	37.54
Napier	491	24.02	21.89	26.30	31	27.63	19.14	38.75
New Plymouth	593	35.26	32.41	38.30	28	31.64	21.51	45.09
Wanganui	250	19.16	16.83	21.73	11	17.43	9.50	29.84
Palmerston North	555	24.90	22.82	27.12	35	29.91	21.17	41.19
Hutt	395	16.69	15.05	18.46	38	27.76	19.92	37.77
Wellington	439	17.03	15.44	18.74	30	17.09	11.77	24.10
Wellington Region	171	19.96	17.06	23.22	0			
West Coast	9	11.70	6.01	21.03	0			
Nelson	318	21.13	18.83	23.64	8	14.95	7.39	27.69
Christchurch	717	15.83	14.66	17.06	40	13.40	9.69	18.10
Timaru	299	25.03	22.23	28.10	13	20.90	11.94	34.45
Dunedin	353	19.33	17.32	21.50	24	22.77	15.02	33.30
Invercargill	416	23.77	21.49	26.22	18	19.46	12.06	29.99
New Zealand	10760	22.51	22.06	22.95	538	20.33	18.61	22.17

Source: NZBDMP.

A second, more restricted, 'exposed period' was designated as January-March 1987, the quarter nine months after the April 1986 accident. Cases and births for the quarter October –December 1986 were included in the 'control' period. Table 22 shows that the rate of birth defects in New Plymouth in the 'exposed' period was 34.09 (95%CI 20.21-54.50) little different from that in the control period (35.10/1000, 95%CI 32.29-38.09).

Table 22: Rates of birth defects, per 1000 live birth and stillbirth notifications, by District.

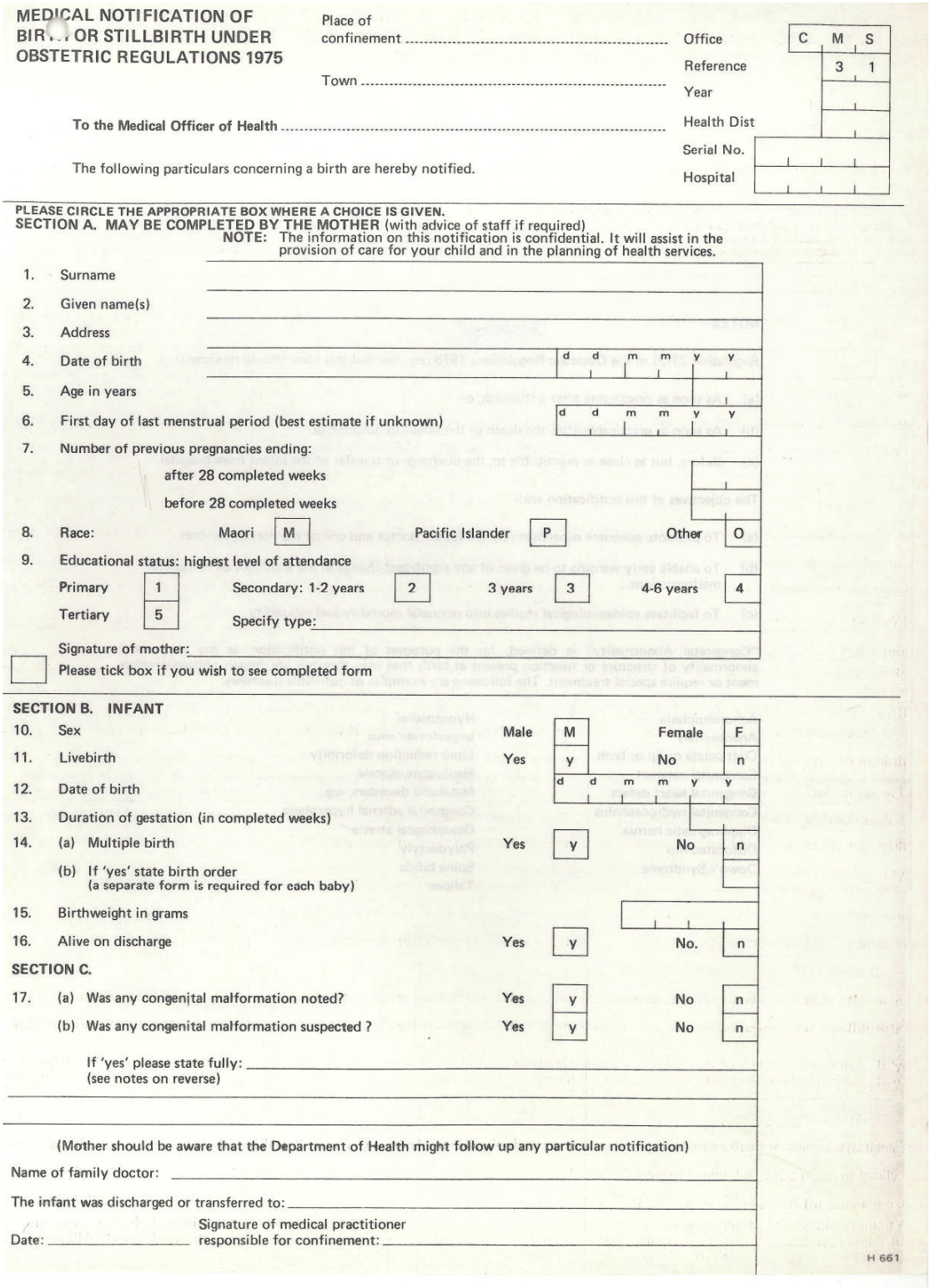
	Control period 1980-December 1986				Exposed period January-March 1987			
	Cases	Rate/1000	95%CI		Cases	Rate/1000	95%CI	
Whangarei/Northland	523	26.09	23.84	28.48	10	18.69	9.91	32.73
Takapuna	480	21.73	19.79	23.82	4	9.48	3.62	21.53
Auckland	2180	26.41	25.27	27.58	49	23.18	17.30	30.48
South Auckland	826	26.57	24.74	28.51	18	18.89	11.70	29.11
Hamilton	1048	22.85	21.44	24.32	22	17.97	11.64	26.69
Rotorua	577	17.61	16.17	19.15	7	7.87	3.72	15.10
Gisborne	263	23.42	20.64	26.48	9	30.30	15.55	54.45
Napier	510	24.25	22.14	26.51	12	22.56	12.60	37.85
New Plymouth	606	35.10	32.29	38.09	15	34.09	20.21	54.50
Wanganui	255	19.06	16.76	21.59	6	20.20	9.04	40.49
Palmerston North	571	24.97	22.91	27.16	19	32.26	20.24	49.21
Hutt	415	17.04	15.41	18.80	18	26.32	16.30	40.56
Wellington	454	17.02	15.46	18.70	15	17.42	10.33	27.85
Wellington Region	171	19.96	17.06	23.22	0			
West Coast	9	11.70	6.01	21.03	0			
Nelson	321	20.90	18.64	23.37	5	22.12	9.24	46.82
Christchurch	742	15.84	14.69	17.06	15	10.40	6.16	16.62
Timaru	305	24.84	22.08	27.85	7	24.39	11.52	46.82
Dunedin	360	19.21	17.24	21.34	17	29.41	17.98	45.84
Invercargill	428	23.77	21.53	26.19	6	14.08	6.30	28.23
New Zealand	11044	22.46	22.02	22.90	254	19.85	17.45	22.49

Source: NZBDMP.

There is no evidence from these data, of an increase in the rate of all birth defects in the New Plymouth district following the accident at the IWD plant in April 1986. The data were not available to detect an increase of a specific defect or group of defects.

Appendix 2

Medical Notification of Birth or Stillbirth (H661) form



MEDICAL NOTIFICATION OF BIRTH OR STILLBIRTH UNDER OBSTETRIC REGULATIONS 1975

Place of confinement .....
Town .....
Office C M S
Reference 3 1
Year
Health Dist
Serial No.
Hospital

To the Medical Officer of Health .....

The following particulars concerning a birth are hereby notified.

PLEASE CIRCLE THE APPROPRIATE BOX WHERE A CHOICE IS GIVEN.

SECTION A. MAY BE COMPLETED BY THE MOTHER (with advice of staff if required)

NOTE: The information on this notification is confidential. It will assist in the provision of care for your child and in the planning of health services.

1. Surname
2. Given name(s)
3. Address
4. Date of birth (d d m m y y)
5. Age in years
6. First day of last menstrual period (best estimate if unknown) (d d m m y y)
7. Number of previous pregnancies ending:
   after 28 completed weeks
   before 28 completed weeks
8. Race: Maori M Pacific Islander P Other O
9. Educational status: highest level of attendance
   Primary 1 Secondary: 1-2 years 2 3 years 3 4-6 years 4
   Tertiary 5 Specify type:

Signature of mother:

☐ Please tick box if you wish to see completed form

SECTION B. INFANT

10. Sex — Male M Female F
11. Livebirth — Yes y No n
12. Date of birth (d d m m y y)
13. Duration of gestation (in completed weeks)
14. (a) Multiple birth — Yes y No n
    (b) If 'yes' state birth order (a separate form is required for each baby)
15. Birthweight in grams
16. Alive on discharge — Yes y No. n

SECTION C.

17. (a) Was any congenital malformation noted? Yes y No n
    (b) Was any congenital malformation suspected ? Yes y No n

If 'yes' please state fully: (see notes on reverse)

(Mother should be aware that the Department of Health might follow up any particular notification)

Name of family doctor:

The infant was discharged or transferred to:

Date: Signature of medical practitioner responsible for confinement:

H 661

Appendix 3

Boundary changes, 1985-88

In the course of the period of study for the NZBDMP data, there were changes to the boundaries of the various reporting organisations. The impact on the comparison of the rates between the various local areas through the period is unknown.

During October-December 1985 Area Health Boards (AHBs) were introduced to replace some Health/Hospital Board Districts. There were some boundary changes. The Wanganui AHB did not include Taumarunui Borough or County which were included in the Hamilton Health District. The Nelson AHB boundaries were markedly different from those of the Nelson/Greymouth Health District. The areas covered by the Greymouth Health District plus Marlborough County and Picton and Blenheim Boroughs were now included in the Christchurch District Health Office.

In 1987, there were boundary changes in the Auckland and South Auckland Health Districts. On 1 November 1987, the Taranaki AHB took effect on, bringing together the former Taranaki Hospital Board and the New Plymouth Health District Office.

In April-June 1988, two new AHBs were established. The Otago AHB, combined the Otago, Vincent, Maniototo and Waitaki Hospital Board areas, but Marlborough AHB covered the same area as the previous Marlborough Hospital Board.

In July-September 1988, the Wellington and Hutt Health Districts were amalgamated into the Wellington Region Health District

Appendix 4

Comments from the NZBDMP with relevance to New Plymouth, 1980-90

March 1980: ‘New Plymouth and Gisborne had rates over twice as high as the national figure…’ ‘Examination of the notified anomalies from New Plymouth and Gisborne did not disclose geographical clustering nor unusual clusterings of diagnosis.’

March 1980: ‘The February statement contained a list of hip conditions reported from New Plymouth Health District. After further consideration of representations which have been made to NHSC it has been decided to exclude clicky hips from the regional figures unless further information confirms dislocation. It appears that orthopaedic examination of all infants occur in some areas and this could well affect the rates for those areas.’

September 1980: it was noted ‘For New Plymouth, the district with the highest rates (35 per 1000 completed notifications over the three months) 54 percent of the cases were suspected or noted congenital dislocation of the hip.’

March 1981: ‘The two districts with the highest rates were New Plymouth, 45 per 1000, and Palmerston North with 39 per 1000 completed notifications. No temporal or spatial clusterings observed in either of these districts for the period Jan-Mar 1981’.

April 1981: ‘The highest rate was for the New Plymouth district with 47 followed by Nelson with 37 and Auckland with 36 per 1000 completed notifications’.

May 1981: ‘New Plymouth district’s rate of 48 per 1000 completed notifications was over twice as high as the national rate (22 per 1000). Seventeen (77 percent) of the 32 cases reported from New Plymouth over the past three months involved diagnoses of congenital dislocation of the hip’.

June 1981: ‘The two districts which have the highest rates were New Plymouth 39 and Nelson 35. In New Plymouth district 9 of the 17 cases or 53 percent, reported for the three months March-June were diagnosed as suspected or noted congenital dislocation of the hips.’

July 1981: ‘New Plymouth district again had the highest rate of infants reported with anomalies for the three months May to June. The rate of 48 per 1000 completed notifications was more than twice as high as the national rate. Nine (41 percent) of the 22 cases reported were diagnosed as dislocated or dislocatable hips.’

August 1981: ‘New Plymouth district continue to have the highest district rate, a trend evident since March 1981. As noted in previous statements this district has reported a high percentage of suspect or noted congenital dislocated (or dislocatable) hips. Of the 54 anomalies notified from New Plymouth since January 1981 34 or 63 percent involved this diagnosis.’

September 1981: ‘Variations in screening neonates for such conditions as dislocatable hips probably result in higher anomaly rates being reported from some districts and are reflected in the three monthly rates’. ‘Fifty seven percent (12) of the anomaly cases reported from New Plymouth district for July to September involved dislocatable(ed) hips.’

October 1981: ‘Five of the eight New Plymouth cases were diagnosed as congenital dislocatable(ed) hips.’

November 1981: High rate in the Timaru district with half of the cases being congenital dislocated (or dislocatable) hips.

January 1982: '…some districts report very few cases of common anomalies (e.g. talipes, dislocatable hips).'

February 1982 'The three districts with the highest rates, New Plymouth 35, Palmerston North 38, and Nelson 35, showed no evidence of clustering. The differences in screening practices for some anomalies (mainly orthopaedic) are reflected in the consistently higher rates recorded for some districts, and should be remembered when inter-district rates are compared.'

May 1982: 'The two highest [rates] were South Auckland (35) and New Plymouth (40). No clusters were identified among the cases from these districts. Although a feature of the New Plymouth cases was three infants with dissimilar anomalies (and of varying gestational stages) reported from a relatively small community. Close surveillance for future cases will be maintained.'

June 1982: 'New Plymouth, the district with the highest rate (40 per 1000) reported 55 percent of the cases as either dislocatable hips or talipes'. Similarly in Hamilton these conditions accounted for 51 percent of the total reported cases. The percentages of talipes and dislocatable hips reported from the other districts with high 3-monthly rates were in the range 25 to 40 percent.'

June 1982: It was reported that in 1981 in New Plymouth the observed/expected ratio of all monitored anomalies (ie, anecephaly, spina bifida, hydrocephaly, cleft palate, cleft lip, oesophageal atresia, anorectal atresia, hypospadias, limb reduction deformity, omphalocele, Down syndrome) was 1.2 compared to national average 0.9. It is not known if this difference was statistically significant. There were also twice as many observed cases than expected of anorectal atresia and hypospadias. However, the report noted: 'Because of the small numbers of cases involved the data in this table should be interpreted with caution.'

July 1982: ‘The three districts with the highest 3–monthly aggregated rates … were New Plymouth 42, Hamilton and Wanganui 31. No specific clusters were identified in these districts.’

September 1982: ‘Whangarei health district had a high rate of 37, one third of the cases were congenital dislocated hips.’

October 1982: ‘The three districts with the highest rates were Whangarei 39, Invercargill 38 and New Plymouth 35. Apart from a small cluster of cleft lip/palate cases which are being investigated in the Whangarei district no other clusters were noted.’

January 1983: ‘Timaru (59) Auckland (34) Napier (31) and New Plymouth (30) had rates above the national average for November-January [1982].

February 1983: ‘New Plymouth notified 6 cases of CDH (37 percent) and 2 cases of cleft lip and 1 of cleft palate.’ … it would appear that the 3 cases of cleft lip and 1 of cleft palate which occurred in New Plymouth in January-February are significantly greater than could be expected’. ‘Three cases of cleft palate notified from Hamilton are also significantly greater than the expected number.

March 1983: ‘Six of the New Plymouth cases (32 percent) were suspected of noted dislocated hips, and 4 (26 percent) were cleft lip/palate cases’. The latter were investigated.

April 1983: ‘Districts with rates more than 20 percent above the national average were New Plymouth 38, South Auckland 37, Timaru 32, Gisborne 31 and Palmerston North 29.’

May 1983: ‘‘Districts with rates more than 20 percent above the national average were New Plymouth 39, Auckland and South Auckland 31, Timaru 30, and Wanganui 28. Forty seven percent of the New Plymouth cases were suspected or noted dislocation of hips, followed by talipes at 16 percent.’

June 1983: ‘Districts with rates more than 20 percent above the national average were New Plymouth 39, Invercargill 31, Palmerston North 29, Whangarei, Auckland and Gisborne 28 respectively. Nine of the New Plymouth cases (53 percent) were dislocated hips, followed by 3 (18 percent) cardiac cases.’

July 1983: ‘Fifty seven percent, 8 of the 14 New Plymouth cases, were dislocated hips. Each of the remaining 6 cases had a different anomaly.’

August 1983: ‘Districts with rates more than 20 percent above the national average for June – August were Invercargill 36, South Auckland 31, Takapuna 30, Timaru 29, Gisborne 28.’

September 1983: ‘Districts with rates more than 20 percent above this level [the national average for July – September was 23] were South Auckland 39, Hamilton and Wanganui 31, Invercargill 30 and New Plymouth and Timaru 29.’

October 1983: ‘Districts with rates more than 20 percent above the national average for August – October were Wanganui 41, Hamilton 36, South Auckland 32, New Plymouth 31 and Invercargill 28. Talipes and congenital dislocation of hips accounted for 47 percent of Hamilton’s and 56 percent of New Plymouth’s anomaly notifications.’

November 1983: ‘Districts with rates more than 20 percent above the national average of 23, for the three months September – November were New Plymouth 40, Hamilton 33, Wanganui 32 and South Auckland 30. The most frequently occurring anomalies, talipes and congential hip dislocation accounted for 57 percent of New Plymouth cases, 40 percent of Hamilton’s and 38 percent of South Auckland cases.’

December 1983: ‘Five districts had rates of 20 percent or more above the national average for October – December. They were New Plymouth 35,

Whangarei 30, Auckland, South Auckland and Nelson 28. As usual, the main anomalies reported in districts with high rates were talipes or congenital dislocation of the hip either on their own or combined.'

January 1984: 'The 4 districts with rates 20 percent or more above the national average for November to January were Whangarei 39, New Plymouth 36, Rotorua 29 and Palmerston North 27. Congenital dislocated hip and talipes were the main anomalies reported from New Plymouth, accounting for 59 percent of cases. There were 3 cases (18 percent) of hypospadias or epispadias also reported.'

February 1984: 'Districts with rates 20 percent or more above the national average for December 1983 to February 1984 were Invercargill 35, Rotorua 34, New Plymouth 31, Gisborne 29, Whangarei and Napier 25.'

March 1984: 'Districts with rates 20 percent or more above the national average for January to March were New Plymouth 35, South Auckland and Invercargill 29, Rotorua 28 and Gisborne and Napier 27. Fifty-three percent, 8 of the 15 cases notified from New Plymouth were either talipes or congenital dislocated hips. Between January and March 3 infants, 2 with hypospadias and 1 with epispadias, were born to women domiciled in the New Plymouth district. As this is statistically significantly higher than the expected number from the district surveillance will be continued.'

April 1984: 'Districts with rates 20 percent or more above the national average for February to April were New Plymouth 40, South Auckland 34, Gisborne 32, Napier 28 and Hamilton and Rotorua 26. Nine (53 percent) of the New Plymouth cases notified were dislocated hips.'

May 1984: 'Districts with rates 20 percent or more above the national average for March to May were New Plymouth 38, Wanganui 37, Hamilton 32 and South Auckland 29. Seven (47 percent) of the New Plymouth cases involved dislocated hips, and 3 (20 percent) cardiac anomalies.'

May 1984: ‘In February [1984] an investigation was launched into a small cluster of neural tube defects reported from the Rotorua district. Reports on 6 of the 7 cases have been analysed, 1 case was lost to followup. There was no apparent common link among the cases. Some of the case histories were complex, and included a family history of neural tube defects and exposure to medication and possible exposure to herbicide sprays.’

June 1984: ‘Districts with rates 20 percent or more above the national average for the 3 months April to June were New Plymouth 35, Hamilton 34, Whangarei, Gisborne and Wanganui 30 and Nelson 29. The most commonly reported anomaly in the New Plymouth district was congenital dislocated hips with 8 cases (50 percent) followed by 2 cases of talipes (13 percent).’

July 1984: ‘Six districts had rates 20 percent or more above the national average for May to July. The highest of these was New Plymouth – 39, followed by Nelson – 37, South Auckland – 35, Wanganui – 33, Hamilton – 31 and Napier – 30. Nine, 50 percent, of New Plymouth cases notified were congenital dislocated hips.’

August 1984: ‘The 3 districts with rates 20 percent or more above the national average for June to August were New Plymouth 45, South Auckland 39 and Nelson 32. New Plymouth reported 11 cases of congenital dislocated hips and 3 cases of talipes. Together these accounted for 64 percent of the anomalies notified from the district for June to August.’

September 1984: ‘Four districts had rates 20 percent or more above the national average for July to September. They were New Plymouth 45, South Auckland 39, both Hamilton and Napier 27. Nine (43 percent) of the New Plymouth notifications involved congenital dislocated hips and 4 cases (19 percent) talipes.’

October-November 1984: ‘In general, districts with very high rates such as New Plymouth tend to notify a high proportion of the commonest anomalies,

ie, congenital dislocated hips and talipes, counting in some instances for up to 75 percent of the anomalies reported.**'**

October-November 1984: 'In November 2 districts (New Plymouth and Invercargill) both notified 1 infant with oesophageal atresia and another with imperforate anus.'

January-March 1985: 'Districts with anomaly rates 20 percent or more above the national average for the 3 months January to March were Gisborne 42, Whangarei and Napier 35, South Auckland 34, and Nelson and New Plymouth 27. New Plymouth reported 14 cases 11 of which (79 percent) involved dislocated hips.'

April-June 1985: 'Districts with anomaly rates 20 percent or more above the national average for the 3 months April – June were South Auckland and New Plymouth 32, Wanganui and Palmerston North 30, Hutt and Timaru 29 and Gisborne 27. Seven cases (44 percent) of the 16 reported from New Plymouth involved congenital dislocated hips.' New Plymouth also notified 2 cases of hypospadias, 1 of cleft palate and 1 of Down syndrome.

July-September 1985: 'Districts with anomaly rates 20 percent or more above the national average for September quarter were New Plymouth 46, South Auckland 36, Auckland 32 and Timaru 31. Over half of the New Plymouth cases (63 percent) involved congenital dislocated hips, and a further 4 cases (17 percent) were talipes.' One case of Down syndrome was also notified.

October-December 1985: 'Districts with anomaly rates 20 percent or more above the national average for the 4th quarter were Timaru 42, Auckland 36, South Auckland and New Plymouth 35 and Gisborne 34.' Congenital dislocated hips and talipes accounted for 75 percent of the reported cases in New Plymouth.

January-March 1986: **:** ‘Districts with anomaly rates 20 percent or more above the national average for the 1st quarter were South Auckland 46, New Plymouth 45 and Timaru 41. New Plymouth reported 9 cases (45 percent) of congenital dislocated hips and 4 cases (20 percent) of talipes were notified.’ One case of anorectal atresia or stenosis was also reported from New Plymouth.

April-June 1986: ‘In the past, districts reported as having rates more than 20 percent above the national average have frequently fallen into this category due to a high number of congenital dislocated hips or talipes. As procedures for checking infants and criteria for diagnosis for these two conditions may vary between hospitals Table 1 now includes columns showing the numbers and rates by districts of infants with anomalies, excluding cases of congenital dislocated hips or talipes. As a result the national rate in this quarter for all defects was 24/1000 completed notifications (310 cases), compared to 15/1000 (189 cases), excluding congenital dislocated hips and talipes. Districts with anomaly rates 20 percent or more above the national average with the inclusion of congenital dislocated hips and talipes were New Plymouth 53, Invercargill and Timaru 37, South Auckland 34, Dunedin 31 and Napier 29. Excluding reported cases of congenital dislocated hips and talipes, districts 20 percent above the national average were South Auckland 23, Wanganui 21, Takapuna and Timaru 20, and New Plymouth 19.’

July-September 1986: ‘Excluding cases of talipes and congenital dislocation of hips, districts with anomaly rates 20 percent or more above the national average were Timaru 27, Palmerston North 24, Auckland 23, Napier and Northland 22, and Invercargill 20.’

October-December 1986: : ‘Excluding cases of talipes and congenital dislocation of hips, districts with anomaly rates 20 percent or more above the national average for the 3 months October–December were Napier 25, Auckland and Palmerston North 21, and South Auckland 20.’

January-March 1987: ‘Districts with anomaly rates 20 percent or more above the national average for the 3 months January-March were Palmerston North (79 percent), Dunedin (71 percent), Nelson (29 percent), Hutt (29 percent), Auckland (21 percent) and Napier (7 percent).’

Two cases of spina bifida were notified from New Plymouth and 1 case of hypospadias.

April-June 1987: **‘**Districts with anomaly rates 20 percent or more above the national average for the 3 months April-June were Palmerston North (71 percent), Napier (64 percent), Gisborne and Timaru (43 percent), Dunedin (29 percent), Takapuna, Invercargill and Nelson (21 percent).’

July-September 1987: ‘Districts with anomaly rates 20 percent or more above the national average for the 3 months July-September were Auckland (67 percent), Palmerston North (33 percent) and Timaru (53 percent).’

One case of spina bifida and 1 case of cleft palate reported from New Plymouth.

October-December 1987: ‘Districts with anomaly rates 20 percent or more above the national average for the 3 months October-December were Palmerston North (81 percent), Auckland (50 percent) and Dunedin (20 percent).’

January-March 1988: ‘Districts with anomaly rates 20 percent or more above the national average for the 3 months January-March were Takapuna (55 percent), Rotorua and Palmerston North (27 percent each) and Hamilton (18 percent).’

April-June 1988: ‘Districts with anomaly rates 20 percent or more above the national average for the 3 months April-June were Palmerston North (135 percent), Auckland (50 percent), Gisborne and Napier (43 percent), Invercargill and Wanganui (14 percent).’

One ‘minor’ case of exomphalos was reported from Taranaki. The Taranaki Area Health Board took effect from 1 November 1987, bringing together the Taranaki Hospital Board and the new Plymouth Health District Office.

July-September 1988: ‘Districts with anomaly rates 20 percent or more above the national average for the 3 months July-September were Napier and Invercargill (50 percent), Auckland (36 percent) and Otago (21 percent).’

October-December 1988: ‘Districts with anomaly rates 20 percent or more above the national average for the 3 months October-December were Palmerston North (79 percent), Takapuna (21 percent) and Timaru (21 percent).’

One case of cleft lip was notified from Taranaki.

January-March 1989: ‘Excluding cases of congenital dislocation of the hip and talipes, districts with anomaly rates 20 percent or more above the national average were Northland (72 percent higher), Otago (61 percent higher), and Auckland (44 percent higher).’

Two cases of cleft lip and two cases of cleft palate were reported from Taranaki.

April-June 1989: ‘Excluding cases of congenital dislocation of the hip and talipes, districts with anomaly rates 20 percent or more above the national average above were Marlborough (141 percent higher), Palmerston North (117 percent higher), Northland (70 percent higher), Southland (58 percent higher), Napier (47 percent higher) and Taranaki (23 percent higher).’

One case of cleft palate, one case of omphalocele, and one case of Down syndrome were reported from Taranaki.

July-September 1989: ‘Excluding cases of congenital dislocation of the hip and talipes, districts with anomaly rates 20 percent or more above the national average were Marlborough (141 percent higher), Palmerston North (117 percent higher), Northland (70 percent higher), Southland (58 percent higher), Napier (47 percent higher) and Taranaki (23 percent higher).’

One case of cleft lip and one case of limb reduction defects were reported from Taranaki.

October-December 1989: ‘Excluding cases of congenital dislocation of the hip and talipes, districts with anomaly rates 20 percent or more above the national average were Tairawhiti (127 percent higher), Northland (93 percent higher), West Coast (53 percent higher), Auckland, Palmerston North and Nelson (40 percent higher), and Hamilton (20 percent higher). No clusters of specific anomalies were noted in the districts with high rates.’

In Taranaki, one case of limb reduction defects was reported.

Appendix 5

Clusters investigated or further surveillance conducted by the NZBDMP, 1980-90

On a number of occasions during the decade 1980-89, the prevalence of birth defects was more closely monitored (because of an unusual occurrence or the number of observed cases exceeded the number expected) or there was an investigation (often in collaboration with a local Medical Officer of Health) of possible clusters in time or space.

1980: cleft palate, cleft lip (South Auckland to Rotorua, Hamilton and Napier), hydrocephaly (Wellington), spina bifida (Christchurch), congenital heart defects, and hypospadias (especially, in Takapuna, Auckland, and South Auckland Health Districts).
1981: hydrocephaly (Christchurch), limb reduction defects (South Auckland), anencephaly.
1982: non-specific defects in a small community in New Plymouth, oesophageal stenosis, limb reduction defects (time), neural tube defects, cleft lip/and or cleft palate (Rotorua), cleft lip/cleft palate (Whangarei), anorectal atresia or stenosis (Wanganui).
1983: cleft lip and palate (New Plymouth, Hamilton), tracheo-oesophageal atresia or stenosis (Takapuna, Auckland, South Auckland), Down syndrome (Palmerston North), spina bifida (Takapuna, South Auckland, Hamilton), hypospadias (Auckland, Napier), limb reduction deformities (time), neural tube defects (Rotorua), hydrocephaly.
1984: hypospadias and epispadias (New Plymouth), congenital heart defects (Napier), spina bifida (Wanganui), cleft lip and palate (Napier), hypospadias (Hamilton, Nelson), multiple defects (including exomphalos and umbilical hernia), anencephaly
1989: congenital cataracts (Wellington)