graphed on Kodak Graphic Arts film (No. 4127). For serial sections, a 3-mm length of maxilla and surrounding tissue was excised from a preserved specimen, embedded in Paraplast under vacuum, sectioned at 4 μm, and treated with a variety of stains.
Specimens examined: Scanning electron microscopy: *Xenopeltis unicolor*, USNM (National Museum of Natural History) 163969; *Liophidium rhodogaster*, USNM 149835; *Scaphiodontophis annulatus* (= *zeteki*), KU (University of Kansas Museum of Natural History) 62125; *Sibynophis chinensis*, USNM 35521; *Lycophidion capense*, USNM 162450; and *Mehelya poensis*, USNM 199297. Histology: *Scaphiodontophis venustissimus*, KU 125493.
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Personal observation, based on intestinal contents of the following specimens: *Liophidium rhodogaster*, USNM 149835; *L. torquatus*, AMNH (American Museum of Natural History) 24802, CAS (California Academy of Sciences) 123194, USNM 149249; *L. trilineatus*, AMNH 60672; and *L. vaillanti*, USNM 150599. As intestinal contents, the sample may be biased in favor of relatively durable remains, such as scales and osteoderms. Although a greater variety of prey thus is not precluded, the inclusion of hard-bodied lizards in the diet of *Liophidium* is nevertheless established.
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* Present address: Section of Ecology and Systematics, Langmuir Laboratory, Cornell University, Ithaca, N.Y. 14850

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Aerial Spraying of 2,4,5-T and Human Birth Malformations: An Epidemiological Investigation

Abstract. *An investigation of the rate of birth malformations in the Northland region of New Zealand provides no evidence to associate spraying of 2,4,5-trichlorophenoxyacetic acid with the occurrence of any malformation of the central nervous system, including spina bifida. A statistically significant association between spray and malformation is found in the case of talipes. Whether this association indicates a causal relation remains to be established.*

Considerable interest has focused on the possible effects of the herbicide 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and its contaminant 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) on human health and environment (*1*). In this investigation, hospital records were used to identify the rates of occurrence of all diagnosed malformations, including stillbirths and neonatal deaths, but excluding miscarriages at less than 28 weeks fetal age, in specific areas of Northland, New Zealand, for each month over the period 1960 to 1977. These rates were compared with the densities of monthly aerial 2,4,5-T spray application in the same area during the same period. Seven mutually exclusive areas were defined, each centered on one of the region's maternity hospitals: Dargaville, Rawene, Kaitaia, Whangarei, Kawakawa, Kaikohe, and Kaeo.

During the study period, 37,751 babies were born in Northland hospitals; 436 of these were stillborn, 264 died shortly after birth, and 510 had diagnosed malformations (*2*). It is estimated that in Northland, as in the rest of New Zealand, well over 99 percent of all births occur in hospitals.

Aerial spraying of 2,4,5-T is carried out in Northland by a number of companies, and from the records each made available, it was possible to identify the site of each spray job and the quantity and date of 2,4,5-T application (*3*). As no aerial spraying took place in Northland in the first half of the 1960's, calendar years 1959 to 1965 were chosen as representative of those without environmental 2,4,5-T, and 1972 to 1976 as representative years with 2,4,5-T spraying. Within that period there was considerable variation in the quantity of 2,4,5-T applied in each year, in each month, and in each of the seven areas.

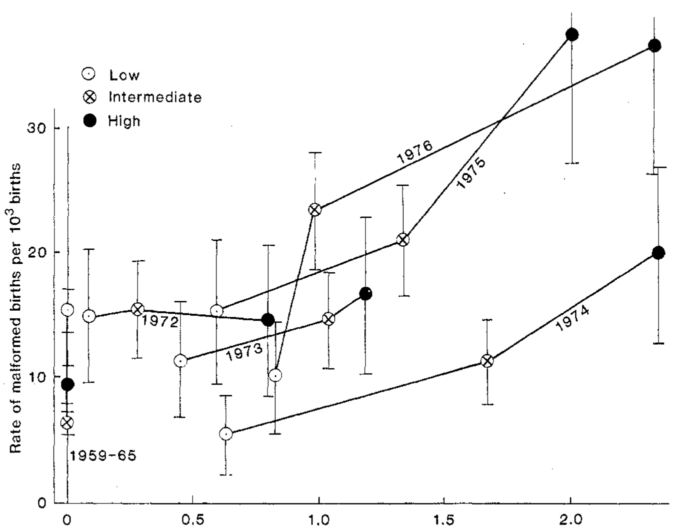

Fig. 1. Distribution of birth malformations during years of no spraying (1959 to 1965), which are treated as 1 year, and from 1972 through 1976. Areas of low, intermediate, and high averaged annual spray density correspond to the various hospital areas: low includes Dargaville, Rawene, and Kaitaia; intermediate is Whangarei; and high includes Kawakawa, Kaikohe, and Kaeo. Poisson error bars are shown: decay hypothesis, f = 1.00.

It was assumed that putative teratogen is removed from the environment at a fixed fractional rate f month by month and that its expected environmental concentration in area j in month k is given by

$$c(j,k) = s(j,k) + (1 - f) \times c(j,k - 1)$$

where k is 12 (calendar year minus 1959) plus calendar month, and $s(j,k)$ is the environmental concentration of chemical applied in area j in month k; both $c(j,k)$ and $s(j,k)$ are 0 in the early years. The quantity c is a measure of the probability of exposure to physiologically significant amounts of putative teratogen. Values of 1.00 and 0.25 are used for f. Estimates of fetal age at birth in the hospital records were used to estimate the month of conception k' of each baby born in area j. Such a baby was then assumed to have been exposed to an environmental concentration of putative teratogen proportional to $c(j,k'+1)$.

Two tests of the data were carried out. In the first, babies born between 1 January 1960 and 31 August 1966 were taken as a control group not exposed to 2,4,5-T and those born between 1 September 1972 and 31 August 1977 as a study group, and for each of the commoner malformations an incidence ratio and 90 percent confidence interval were calculated (Table 1). Defects of the heart, hypospadias and epispadias, talipes, and the grouping "all birth malformations" show an incidence ratio significantly greater than 1. In the second test, rates of "all birth malformations" were plotted against average annual exposure to putative teratogen with f equal to 1.00. Regions of high, low, and intermediate spray density are distinguished, as are the years of spraying and the nonspray period (Fig. 1). The incidence rate appears to be positively associated with exposure both across areas and across years.

Binary regression (*4*) was used to investigate further the correlation between individual malformations and exposure. Each birth is associated with a pair (Y_i, x_i) where Y_i is a binary random variable that takes the value 1 if the ith baby has a particular malformation, or one of a group of malformations, and 0 if it does not, and x_i is the exposure for the ith baby. The difference between the average exposure value for malformed babies T and the exposure value for all babies μ, suitably normalized, is the test statistic.

The correlation between any given malformation and exposure can be derived from the set of all births. It may also be considered in terms of the three variables: year (Y), month of the year (M), and hospital catchment area (A). For example, the influence of year, independent of month or of hospital area, can be examined by partitioning the births into subsets defined by month and hospital area [there are 84 (12 × 7) such subsets], calculating the test statistic for each subset, and combining these (*5*). This is referred to as a y-test. For any malformation seven tests of this kind are possible, with Y, M, A, YM, MA, AY, and YMA; only some results with Y, M, A, and YMA tests are described (*6*) (Table 2).

For this study it was decided that a necessary condition for evidence of a relation between spray and exposure would be tests with Y and A significant at the 5 percent level. In tests with M,

Table 1. Incidence ratios for the commoner malformations. Total number of births between 1 January 1960 and 31 August 1966 was 15,000; between 1 September 1972 and 31 August 1977 there were 9614 births. The change in total births is due to regional depopulation.

Malformation	Total number: 1960 to 1966	Total number: 1972 to 1977	Incidence ratio	Confidence bounds (90 percent): Upper	Confidence bounds (90 percent): Lower
All birth malformations*	148	164	1.73	2.08	1.44
Anencephaly	11	10	1.42	2.90	0.69
Spina bifida	18	13	1.13	2.05	0.62
Cleft lip	16	6	0.59	1.27	0.27
Isolated cleft palate	8	7	1.37	3.19	0.58
All heart malformations	8	20	3.90	7.38	2.06
Hypospadias, epispadias	5	18	5.62	11.73	2.69
Talipes	49	52	1.66	2.29	1.20

*Excludes stillbirths, neonatal death, or dislocated and dislocatable hip.

Table 2. Exposure to putative teratogen for individual malformations.

Malformation	Test	Exposure $f = 1.00$: T	μ*	P†	Exposure $f = 0.25$: T	μ*	P†
All birth malformations‡	Y	58	46	.033	231	155	.0000044
	M	103	122	.93	439	462	.89
	A	115	108	.20	439	397	.0089
	YMA	54	42	.037	231	157	.000013
Anencephaly	Y	43	43	.48	152	133	.33
	M	85	92	.54	319	354	.70
	A	85	101	.65	319	337	.58
	YMA	41	42	.47	152	157	.49
Spina bifida	Y	32	47	.76	125	154	.71
	M	74	99	.77	297	375	.93
	A	80	89	.63	297	332	.73
	YMA	31	42	.69	125	157	.72
Cleft lip	Y	5	27	.94	149	154	.51
	M	18	158	.96	547	616	.69
	A	22	33	.68	547	348	.0094
	YMA	5	42	.96	149	157	.52
Isolated cleft palate	Y	80	49	.14	271	143	.053
	M	170	109	.12	580	440	.066
	A	170	152	.34	580	522	.27
	YMA	80	42	.10	271	157	.079
All heart malformations	Y	75	41	.044	355	152	.00011
	M	98	150	.89	497	535	.74
	A	115	135	.78	497	492	.45
	YMA	70	42	.10	355	157	.00089
Hypospadias, epispadias	Y	104	50	.039	387	165	.00029
	M	127	126	.46	494	512	.59
	A	153	137	.30	494	497	.49
	YMA	100	42	.027	387	157	.00054
Talipes	Y	52	41	.15	207	153	.035
	M	98	125	.87	401	479	.98
	A	109	93	.14	401	328	.0058
	YMA	51	42	.21	207	157	.050

*The expected value of T if exposure values are assigned randomly to malformed babies, that is, if there is no correlation between malformation rate and exposure. Units of T and μ are kilograms per 10^2 km^2 per year. †The probability of a higher value of T if the exposure values are assigned randomly to the malformed babies. ‡Excludes stillbirths, neonatal death, and dislocated and dislocatable hip.

significant associations are likely to be disguised by imprecise knowledge of conception dates, termination periods (*7*), and the environmental characteristics of the putative teratogen. Care must be taken in interpreting any large number of statistical tests such as those of Table 2. Even if there is no correlation between malformation and exposure, it is to be expected that 5 percent of the tests will be significant at the 5 percent level. In Table 2, 25 percent of the tests are significant at the 5 percent level.

Within the terms of the chosen criterion, the results of Table 2 show (i) no identifiable association for anencephaly or spina bifida, (ii) a significant association for talipes when f equals 0.25 but not when f equals 1.00, and (iii) no identifiable association for cleft lip with or without cleft palate, for isolated cleft palate, for malformations of the heart as a group, or for malformations of the male genitalia.

To clarify the nature of the correlations in Table 2, all the tests were recalculated with the contribution of the Whangarei hospital area omitted. Whangarei City (population 35,000) is the only urban area in Northland (population 107,000), and the Whangarei area is responsible for about half the total births. The pattern of correlations obtained was almost unchanged except for some loss of significance. For hypospadias and epispadias the termination period may extend to the end of the first trimester of pregnancy, and for cleft lip, cleft palate, and talipes, to the end of the second trimester. Test statistics for these malformations were recalculated with the exposure values being the average environmental concentration of putative teratogen taken over the second to fourth months and second to seventh months of pregnancy, respectively, for the two groups of malformations. The recalculated statistics showed increases in significance over those in Table 2 for hypospadias and epispadias and for talipes, no change for cleft lip, and a decrease in the case of isolated cleft palate.

The incidence rate of talipes is significantly higher among New Zealand Maoris than among Pakehas (New Zealand Caucasians) (*2, 8*). Therefore, Maoris and non-Maoris (all those not specified as New Zealand Maori) were analyzed separately. These analyses were then combined by treating race as a partition of the data in the same way as Y, M, and A. The combined analyses, which allow for different malformation rates for Maoris and non-Maoris, produced results almost identical with those of Table 2, indicating that race is not primarily responsible for the correlations obtained in this case.

JENNIFER A. HANIFY
PETER METCALF
CHRISTOPHER L. NOBBS
KEITH J. WORSLEY
Northland Births Survey, Box 6256, Auckland 1, New Zealand

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6. The *YMA* test at the 5 percent level has 95 percent power against an alternative hypothesis that increases the expected exposure for malformed births by a factor of approximately $8.8/\sqrt{m}$ for $f = 1.00$, and $6.0/\sqrt{m}$ for $f = 0.25$, where m is the number of malformations. For example, the *YMA* test with $f = 0.25$ has 95 percent power when the expected exposure for spina bifida births is roughly twice the exposure for all births.
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8. R. N. Howie and L. I. Phillips, *N. Z. Med. J.* **71**, 65 (1970). It must be noted, however, that in this survey and in (*2*) the only evidence of race available was the race of the mother as recorded at the time of hospital entry; racial intermarriage is relatively common.
9. We thank Barr Bros. Ltd., James Aviation Ltd., Marine Helicopters Ltd., Thames Aerial Topdressing Ltd., and Wishart Helicopters Ltd. for making their records on spraying available for this study. We also thank C. Garlick, superintendent of Northland Base Hospital, Whangarei; the superintendents and staffs of the other hospitals used in the investigation; A. J. Scott, R. B. Elliott, and R. H. Briant of the University of Auckland; A. H. Smith of the Wellington Clinical School, University of Otago; P. Talagaspitiya and R. Talagaspitiya and D. Ware and S. Ware of Whangarei; and G. Barclay, A. Tasnadi, and D. Arrowsmith of Auckland. Communicate with the authors for a copy of the complete report at cost ($13.50 New Zealand including postage).

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Multiple Paternity in Belding's Ground Squirrel Litters

Abstract. *Sexually receptive female* Spermophilus beldingi *(Rodentia: Sciuridae) usually mate with several different males. The paternity of 27 litters born in 1977 and 1978 was ascertained by combining field observations of mating with laboratory paternity exclusion analyses. Most of the litters (78 percent) were multiply sired, usually by two or three males. This may be the highest frequency of multiple paternity ever directly demonstrated in a natural population.*

Multiple paternity in a litter or brood occurs in natural populations of mammals (*1*), snakes (*2*), salamanders (*3*), fish (*4, 5*), and arthropods (*6–9*); it has also been reported in laboratory populations of mammals (*10*), birds (*11*), fish (*12*), and insects (*8, 13*). In most field studies, multiple paternity is inferred from the presence, in a single brood, of at least three electrophoretically detectable paternal alleles at a given protein locus (*14*); that is, multiple mating is deduced on the basis of genetic information from females and their broods or from broods alone. This method underestimates the frequency of multiple paternity in a population, because multiply sired broods go undetected when mates share the same biochemical phenotypes at the loci considered. To circumvent this problem, probabilistic formulas have been proposed (*1, 15*) for estimating population values from the observed frequency of broods containing three paternal alleles.

In this report we quantify directly the occurrence of multiple paternity in a free-living population. Our analysis rests on field observations of mating in Belding's ground squirrels, *Spermophilus beldingi*, and, as in several recent investigations of genetic variation and vertebrate population structure (*16, 17*), on paternity exclusion studies in which allozymes in the blood are assayed by gel electrophoresis.

Belding's ground squirrels are diurnal rodents that inhabit subalpine meadows in western North America. We observed them in the central Sierra Nevada at the 3040-m summit of Tioga Pass, California (*18*). There ground squirrels are active from May through September; the rest of the year they hibernate. The population has been studied since 1969, and most of