SHORT REPORT

Cancer mortality among local authority pest control officers in England and Wales

H F Thomas, P D Winter, L J Donaldson

Abstract

Objective—To examine cancer mortality by tumour site among local authority pest control officers.

***Methods*—Prospective mortality study, and follow up to the end of 1994, of 1485 male pest control officers aged between 17 and 69 and employed in 296 local authorities in England and Wales for at least six months between January 1980 and April 1984. Observed numbers of deaths were compared with those expected on the basis of the rates for relevant calendar year, cause, sex, and age specific groups for England and Wales.**

***Results*—200 deaths occurred during the follow up period of which 65 were certified as due to malignant neoplasms. No tumour type showed significantly more deaths than expected. Total all cause, lung cancer, and respiratory disease mortality were significantly lower than expected.**

***Conclusions*—15 year follow up of a group of men handling a wide range of pesticides did not show any significant risk of cancer. This may be partially explained by the healthy worker effect and also the limited power of the study to detect significant increases in the less common tumours. Further long term follow up of this cohort will continue. Chemical control of pests that can cause human disease and can contaminate food and water has been, and will continue to be, a major public health measure. It is important to ensure that the health of those applying pesticides is not at excess risk. Negative results are important.**

(*Occup Environ Med* 1996;53:787–790)

Keywords: cancer; mortality; pesticides

MRC Epidemiology Unit (South Wales), Llandough Hospital, Penarth, South Glamorgan, Wales
H F Thomas

MRC Environmental Epidemiology Unit, University of Southampton, Southampton General Hospital, Southampton
P D Winter

Department of Applied Epidemiology, University of Newcastle upon Tyne, Newcastle
L J Donaldson

Correspondence to:
Dr H F Thomas, MRC Epidemiology Unit (South Wales), Llandough Hospital, Penarth, South Glamorgan CF64 2XW, Wales.

Accepted 12 July 1996

Over the past 40 years there has been public concern about the potential cancer risk of pesticides.[1–4] This group of chemicals includes rodenticides, insecticides, fungicides, nematicides, and acaricides. In 1982 a study of local authority pest control officers suggested that they may be at excess risk of contracting bladder tumours due to the use of α-naphthylthiourea (ANTU) which had a β-naphthylamine impurity.[5] This paper reports the mortality experience of a cohort of men mainly identified during that study.

There are extensive publications on the toxicology of pesticides but there is not sufficient human evidence to rate any, with the exception of arsenical compounds, as definite human carcinogens.[6–8] The lack of evidence, although generally reassuring for the public health, is also partly attributable to the difficulties of conducting epidemiological surveys of possible occupational and environmental cancer hazards. Surveillance of workers exposed to potential occupational hazards has been advocated by many leading researchers.[9–11]

Subjects and methods

In 1980 about 360 local authority environmental health departments in England and Wales were contacted during a study of bladder tumours among local authority pest control officers (previously called rat catchers or rodent operatives).[5] Over 290 departments provided details of some 1250 men (there were less than 60 women) who were currently employed as pest control officers. The departments were contacted again at the end of 1983 and asked to update details of pest control officers employed from 1980 until early 1984. A total cohort of 1491 men was established. The number of men employed in each department ranged from one to 32. Only 30 departments (10%) had employed more than 10 pest control officers during the study recruitment phase. Men employed for less than six months were excluded from the cohort. Details of the men were sent to the National Health Service Central Register at Southport and 1485 (99·6%) were "flagged" for notification of death and embarkation.

A random sample of 110 men was sent a postal questionnaire in 1984 to obtain details of their work and social habits; 74 replied.[12] The mean age on entering employment was 37 (range 18–55) years. Each man reported using around 10 different pesticides during and before the study period. The main pesticides used (by at least half of the respondents) were warfarin, γ-HCH, pyrethrins or pyrethroids, and chlordane (table 1). Only three (4%) men

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reported having used ANTU. Smoking habits and alcohol consumption were comparable with those reported by men in the 1982 general household survey.

The mortality analysis used a computer program (PYCL) adapted from that of Hill.[13] The numbers of observed deaths, coded by underlying cause with the international classification of diseases, 9th revision (ICD-9)[14] were compared with those expected on the basis of the rates for the relevant calendar year, cause, sex, and age specific groups for England and Wales. Confidence intervals (CIs) were calculated by the usual methods based on the Poisson distribution. Most men (1264; 85%) were, for the statistical analysis, assumed to have been first at risk from mid-1980—that is, when first recruited. This group actually included 715 men who were first employed in 1974 or earlier. The main cohort, defined in 1980, was supplemented by about 70 men joining in each of the next three years. The follow up was until December 1994, providing at least 15 years of follow up for 85% of the cohort.

Results

Two hundred deaths occurred during the follow up period of which 65 were due to neoplasms. Table 2 shows the numbers of cancer deaths by site and a comparison with those expected on the basis of national rates and the 95% CIs for the observed/expected (O/E) rates. The total all cause mortality (O/E 0·78; 95% CI 0·68–0·90) was significantly lower than expected. There were no significantly raised standardised mortality ratios (SMRs) for O/E rates for any non-malignant causes or among the 32 cancer types examined (ICD-9 codes 140–208).[14] Deaths were attributed to 14 types of malignancy with the most common being lung (19), stomach (seven), rectum (seven), and bladder (seven). On six certificates the cause was given as "carcinomatosis" without the primary site being identified. There was a significant deficit of deaths attributed to lung cancer (ICD 162) with 30 expected and 19 recorded (O/E 0·63, 95% CI 0·38–0·98).

Although none of the cancer sites showed a significantly raised SMR there were seven bladder cancer deaths compared with the 2·91 expected. Review of the available information on these men did not indicate that any of them had handled ANTU.

No death certificates mentioned aplastic anaemia. There were four deaths attributed to leukaemia with 1·87 expected; the confidence intervals ranged from 0·58 to 5·48. One primary hepatic tumour was identified.

Non-cancer related mortalities were unremarkable apart from respiratory disease (ICD 460·0–519·9), which showed a significant deficit (eight deaths, 19·8 expected, O/E 0·41, 95% CI 0·17–0·80). There were 11 deaths (9·47 expected) due to accidents (ICD 800—999·9). These included one fall from a height when working but no acute pesticide poisonings.

Table 1 Pesticides handled by a percentage of the random sample of 74 local authority pest control officers[12]

Chemical	Ever used	Current use	Past use
Warfarin	96	91	5
γ-HCH	88	76	12
Cooper's multispray*	69	47	22
Pyrethrins or pyrethroid	59	50	9
Chlordane	50	42	8
Brodifacoum	49	46	3
Carbaryl	49	35	14
Bendiocarb	47	43	4
Fenitrothion	45	34	11
Coumatetralyl	45	26	19
Zinc phosphide	40	24	16
Difenacoum	39	31	8
Dieldrin	38	16	22
Pirimiphos methyl	35	23	12
Malathion	35	26	9
Formaldehyde	33	28	5
Diazinon	32	23	9
Fluoracetmide	29	14	15
Chlorophacinone	27	22	5
Norbormide	22	8	14
Cyanide	20	15	5
α Chloralose	14	9	5

*Mixture of pyrethroids and diazinon.
Fifteen chemicals used by ⩽ 5% of respondents are not listed.

Table 2 Cancer deaths in the cohort of pest control officers

	Observed	Expected	O/E (95% CIs)
All causes	200	255·7	0·78* (0·68 to 0·90)
All neoplasms	65	82·62	0·79 (0·61 to 1·00)
All malignant neoplasms:	65	81·88	0·79 (0·61 to 1·01)
Oesophagus	1	3·64	0·27 (0·01 to 1·53)
Stomach	7	6·00	1·29 (0·47 to 2·40)
Large intestine	3	5·48	0·55 (0·11 to 1·60)
Rectum	7	3·60	1·95 (0·78 to 4·01)
Liver	1	0·90	1·12 (0·03 to 6·22)
Pancreas	3	3·55	0·84 (0·17 to 2·47)
Larynx	1	0·85	1·18 (0·03 to 6·56)
Lung	19	30·34	0·63* (0·38 to 0·98)
Connective tissue	1	0·31	3·18 (0·08 to 17·70)
Melanoma	1	0·66	1·52 (0·04 to 8·48)
Prostate	2	5·05	0·40 (0·05 to 1·43)
Bladder	7	2·91	2·40 (0·97 to 4·95)
Brain	2	2·30	0·87 (0·10 to 3·14)
Leukaemia	4	1·87	2·14 (0·58 to 5·48)

*P < 5%.

Discussion

This cancer mortality analysis, which provides at least 15 years of follow up from first exposure for most men, showed that deaths due to all cancers or individual types of malignancy were not significantly more than would be expected in a similar sample of men in the general population. Although this gives some reassurance that the pest control officers are not exposed to powerful carcinogens the findings of this first follow up must be interpreted cautiously.

It is known that in industrial cohort studies there are usually low mortalities in the early period of follow up (around the first 15 years) due to selection of healthy workers who have to be fit to enter work and also to survive in the industry.[15] These factors were found to be more notable for respiratory disease and lung cancer in a study of men exposed to vinyl chloride monomer which examined the healthy worker effect.[15] Our study also found fewer deaths than expected for these two conditions. The data that we have on smoking among pest control officers suggests that in 1984 the proportion of current smokers was similar (36% *v* 38%) to that in the general male population.[12]

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Although the study population was the largest cohort of pesticide applicators ever studied in the United Kingdom, and recruited from over 290 local authority departments, the study size is modest and the power of the study to identify less common tumours is limited. For all neoplasms and lung cancer the power of the study to detect a relative risk of 2·0 (at the 5% significance level) is over 99%.[16] However, for individual tumours from which six or less deaths are expected the power to detect such a risk (at the 5% significance level) is reduced to $\leqslant$ 65%.[16]

Cancer mortality studies have the limitation that they provide little information on cancers which are usually non-fatal, such as skin tumours, or for which treatment is now often succesful, such as some haematological malignancies. When the death certificate does not identify a primary site, such as for the six deaths coded as carcinomatosis, information on the specific sites may not be available. If such certificates reflect incomplete registration then useful information has been omitted which could potentially lead to an underestimate of the number of deaths from specific tumour types.

There is some evidence that follow up of mortality by only the Office of Populations, Censuses, and Surveys (OPCS) can underestimate the number of deaths.[17] This is more likely in very long term studies in which it has not been possible for the OPCS to identify and "flag" subjects; in this study only six men (0·4%) were not "flagged" by the OPCS. Five emigrations were notified. A few deaths or emigrations may still be missed by the OPCS among the "flagged" population, usually among those aged over 70 years[17]—a group of 345 (23·2%) men in this study. Overall, any biases due to incomplete ascertainment in this study are likely to be minimal.

A major review of studies of licenced pesticide users[18] shows that different studies have found an increased risk of some cancers including Hodgkin's disease,[19] lymphoma,[20] skin cancer,[20 21] lung cancer,[21-23] cancer of the testis,[9] brain cancer,[18] and bladder cancer.[21] Some studies have, in contrast, found significantly lower risks for lung cancer.[18 19] Many of these studies have less than 15 years of follow up and the cohorts studied handle various pesticides and deal with a wide range of pests. It is therefore not surprising that there are inconsistencies in their findings. A recent literature review identified 280 cases of aplastic anaemia linked to pesticide exposure.[24] No cases were identified in this study. Doll and Peto found that although hepatomas are the tumours reported most often in laboratory animal studies of pesticides there has not been a significant increase in the incidence of liver tumours in developed countries since the introduction of long lasting pesticides such as dieldrin and dicophane (DDT).[25] One primary hepatic tumour was identified in this study.

Most workers exposed to chemicals are exposed to a variety which may potentially interact. Individual exposure is generally difficult to assess and reflects environmental controls and the use of personal protection. The study of a random sample of these men suggested that most wore some protective clothing and over half usually wore overalls, gloves, and protective face masks.[12] Safety clearance to approve pesticides for use in the United Kingdom relied for almost 30 years on a voluntary registration scheme. This was superseded by a mandatory scheme in 1986.[26] This study provides qualified reassurance that no strong general carcinogens have been cleared for use as rodenticides or insecticides in the United Kingdom although the study lacks the power to detect even large increases in relative risk for less common tumours. The Ministry of Agriculture, Fisheries and Food advisory committee on pesticides in 1966 reviewed ANTU, and recommended its use should end and that stocks should be destroyed. The World Health Organisation advised against its use in 1973.[5] It remained in use in the United States until at least 1978, presumably because it was judged that the β-naphthylamine impurity had been removed.[5]

There are differences in the pesticides approved for use by individual countries within Europe.[27] Increasing harmonisation of regulations and the encouragement of free trade may require a review of approval procedures used by member states to agree a common approach.

Carcinogenic agents may have a long latency (perhaps more than 20 years) and if they are responsible for increasing the incidence of a rare tumour a large study population is required or, if numbers are limited, a very long period of follow up. It is intended to re-examine the mortality experience of this cohort in 1999 and 2004.

The Health and Safety Executive provided financial support to identify and flag the cohort. Approval to conduct the study was given by the Leicestershire Health Authority ethics commmittee. We are grateful to the staff of the environmental health departments who provided information and also to staff of the National Health Service central register at Southport. We thank Peter Sweetnam for statistical advice.

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