ORIGINAL ARTICLE

Occupational risk factors for prostate cancer and benign prostatic hyperplasia: a case–control study in Western Australia

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Objective: To assess the association of selected occupational exposures with risk of prostate cancer and with risk of benign prostatic hyperplasia (BPH).
Methods: This population-based case–control study recruited 606 men with a diagnosis of confirmed prostate cancer, 400 men who had undergone their first prostatectomy for BPH and 471 male controls randomly selected from the electoral roll between 1 August 2001 and 1 October 2002 in Western Australia. χ^2 tests and logistic regressions were used for univariate and multivariate analyses to investigate the association of the two outcomes with occupational exposure to pesticides, fertilisers, metals, wood dust, oils, diesel exhaust and polyaromatic hydrocarbons (PAHs).
Results: Exposure to toxic metals at a non-substantial level increased the risk of BPH (odds ratio (OR) 1.39, 95% confidence interval (CI) 1.1 to 1.84) and led to a non-significant excess risk of prostate cancer (OR 1.25, 95% CI 0.96 to 1.61). Non-significant excess risks were observed for prostate cancer after exposure to oils other than mineral oil (OR 1.54, 95% CI 0.95 to 2.51) and for BPH after exposure to PAHs (OR 1.20, 95% CI 0.91 to 1.58). A non-statistically significant protective effect for prostate cancer was seen after exposure to organophosphate pesticides (OR 0. 69, 95% CI 0.43 to 1.12). No other associations were found for either prostate cancer or BPH and no dose–response relationships were seen for the exposures investigated.
Conclusions: These results do not provide evidence that any of the occupational factors examined are risk factors for either prostate cancer or BPH.

Prostate cancer is one of the most common cancers among men in Western countries. In the US, prostate cancer is the second most common cancer after lung cancer—that is, one in five American men will develop prostate cancer during their lifetime.[1] In Australia, prostate cancer is the most frequently diagnosed cancer in men.[2] The aetiology of prostate cancer still remains unclear. Well-established risk factors are age, family history of prostate cancer, diet and ethnicity.[3] Level of physical activity has also been suggested to be a risk factor, but the evidence is not strong.[4–7]

Several potential occupational risk factors for prostate cancer have been investigated. Some studies have provided evidence of a positive association between developing prostate cancer and polyaromatic hydrocarbons (PAHs), diesel fuel and fumes.[8] Pesticides have been suggested to be a possible risk factor in some studies,[9–11] but have been found to be protective in other studies.[12] In addition, some studies have found that exposures to cadmium and fertilisers are markedly associated with prostate cancer.[13 14] Several studies have investigated the relationship between farming and prostate cancer. Some of them have reported excess risk of prostate cancer,[11 15] but others have found no association.[16]

Benign prostatic hyperplasia (BPH) is an extremely common condition among men aged 45–80 years. Nearly half of the Australian men aged >65 years report some urinary symptoms resulting from BPH.[17] Established risk factors that have consistently been associated with BPH include increasing age and there are geographical patterns, with BPH being more common in areas such as Africa and America. Also, there have been debates around the association of lifestyle factors such as physical activity, diet, smoking and alcohol consumption, which may be positively or negatively associated with BPH.[18–22]

Few data are available on occupational causes of BPH or indeed on any causative factors for BPH. In view of the lack of information on this topic, we elected to examine the same occupational factors for BPH as for prostate cancer.

This study used interview-derived data from a population-based case–control study to assess the association of prostate cancer and BPH with occupational exposure to metals, woods, oils, pesticides, herbicides, fertilisers, diesel fumes and PAHs.

MATERIALS AND METHODS

Data were obtained from a population-based case–control study, which was conducted at the University of Western Australia, Perth, Western Australia, Australia, over a 2-year period from January 2001.[23] The study procedure was approved by the ethics committee of The University of Western Australia and the Confidentiality of Health Information Committee, Department of Health, Western Australia.

Patients with prostate cancer

Patients with prostate cancer were identified from the Western Australian Cancer Registry. Those with cancer were considered eligible if they had a histopathologically confirmed prostate cancer in Western Australia between 1 January 2001 and 30 August 2002, were aged 40–75 years at the time of diagnosis, their usual care provider agreed to them being contacted and they were well enough to complete the questionnaire and the telephone interview.

Abbreviations: BPH, benign prostatic hyperplasia; PAH, polyaromatic hydrocarbon; TLV, threshold limit values

Patients with BPH

Patients with BPH were men aged 40–75 years at the time of surgery and had undergone their first prostatectomy for a diagnosis of BPH between 1 January 2001 and 31 December 2001 in either public or private hospitals in Western Australia. They were identified from hospital morbidity records through the Western Australian Data Linkage System. Patients were excluded if they had a history of prostate cancer or prostatectomy before January 2001, or if they had comorbidity severe enough to prevent completion of the questionnaire and interview. Patients with prostate cancer or with BPH were excluded if they had inadequate comprehension of English or were not on the Western Australia state electoral roll.

Controls

Eligible controls were men aged 45–75 years who were randomly selected from the Western Australia electoral roll between 1 August 2001 and 1 October 2002. Controls were frequency-matched to the predicted age distribution of patients with prostate cancer in 5-year age groups. Patients who had a previous diagnosis of prostate cancer or an operation for BPH, who could not complete the questionnaire or who had lack of English comprehension were excluded from the study.

Finally, 1066 patients with prostate cancer, 961 BPH and 1272 controls were identified as potentially eligible to participate in the study. Of these, on the basis of inclusion and exclusion criteria, 606 (57%) patients with prostate cancer, 402 (42%) with BPH and 471 (37%) controls participated and provided complete data.

Data collection

After participants gave informed consent, they were sent a questionnaire asking about demographic details, urinary symptoms, diet, smoking and occupational history. The lifetime history included each job held for $\geqslant$1 year with details on job title, employer, industry, start and finish years, number of hours worked per day and number of days worked per week.

Assessment of occupational exposure

For 14 specific occupations (carpenter, driver, electrician, plumber, forestry worker, farmer, labourer, machinist, mechanic, miner, fisherman, painter, railway worker and welder), further detailed sets of questions (known as job-specific modules) were obtained from the US National Cancer Institute[24] and modified to suit the Australian conditions of this study. If the patients had ever been employed in one of these occupations, they were asked the questions in the job-specific modules in a customised computer-assisted telephone interview. The modules asked about time spent in various tasks in that occupation.

An expert occupational hygienist (blind to case–control status) reviewed the occupational histories and the answers to the modules, and determined exposure to the following substances: toxic metals (mercury, cadmium, chromium, lead, manganese, uranium, tungsten, beryllium and nickel), inorganic arsenic and other metals (not arsenic or toxic metals); hard and soft wood, mineral oils, other oils (including synthetic, emulsified and vegetable oils), pesticides, synthetic and natural fertilisers, diesel fumes, and other PAHs (including petrol, other exhausts and other PAHs).

The hygienist first allocated likelihood of exposure to each substance as probable, possible or no exposure. She then allocated one of three levels of exposure using previous literature and her own professional knowledge and without regard to the probability of exposure. The reference levels were internationally recognised occupational safety guidelines (time-weighted average threshold limit values (TLV) set by the American College of Government Industrial Hygienists). Levels of exposure higher than the TLV were considered high, those $\leqslant$10% of the TLV were considered low and other exposures were considered medium. For the few people who reported wearing gloves, respiratory protection and overalls when mixing and applying pesticides, the level was dropped to one level lower. Frequency of exposure was allocated as the number of 8-h days per year and was calculated using the responses to the task questions. Frequency of exposure was categorised into four groups based on the standard year (40 h/week for 48 weeks): not exposed; exposed for $<$0.5 times the standard frequency; exposed for 0.5–1.5 times the standard frequency and exposed for $>$1.5 times the standard frequency. Those with missing frequency were allocated the lowest frequency group as many had only intermittent exposure.

Total dose of exposure was calculated by combining data from all jobs over a person's entire working life. Dose was classified as substantial if the patient was probably exposed to the substance at a medium or high level at a frequency of more than half a standard year for a combined total of $>$5 years, and as non-substantial if the dose was any other combination of exposures. For this dose variable, possible latency was taken into account by omitting exposures within the 5 years before the interview for calculation of the substantial exposure.

Statistical analysis

Contingency tables with χ^2 tests were generated to compare the two case groups (with prostate cancer or BPH) with the control group with regard to demographic and occupational variables. The data were analysed using unconditional logistic regression in the SPSS statistical software V.13 to estimate the association of the occupational exposures with prostate cancer and with BPH. All generated odds ratios (ORs) have been adjusted for age. The level of statistical significance was set at $p<0.05$.

As some of the controls may have had undiagnosed BPH, and some men may have had operations for lifestyle reasons although the BPH was very mild, we repeated the BPH analyses after excluding patients with mild urinary tract symptoms before their operation (n = 28) and controls with moderate and severe symptoms (n = 133).

RESULTS

The distribution of patients and controls according to the various characteristics has been described previously.[23] Patients with prostate cancer were slightly younger than the others and were more likely to report a family history of prostate cancer. We found no remarkable differences in the distribution of controls and in patients with either BPH or prostate cancer in terms of residential place (rural *v* urban) and country of birth (table 1). The distribution of total meat, fruit and vegetable consumption, a possible confounding factor, was similar in the three groups.

For prostate cancer, in the univariate analyses, we found no significant associations between any of the occupational exposures and case–control status (table 2). After adjusting for age, non-substantial exposure to toxic metals (OR 1.25, 95% confidence interval (CI) 0.96 to 1.61) and non-mineral lubricant oils (OR 1.54, 95% CI 0.95 to 2.51) seemed to be associated with increased risk of prostate cancer. However, the CIs included one and there was no dose–response relationship. A non-statistically significant protective effect was found for prostate cancer with organophosphate pesticides (OR 0.69, 95% CI 0.43 to 1.12) and other pesticides (OR 0.69, 95% CI 0.34 to 1.37). No subjects were exposed to pesticides at substantial levels. Also, non-significant protective associations were observed for those who were non-substantially exposed to natural fertilisers (OR 0.66, 95% CI 0.34 to 1.30). Further, we

Table 1 Characteristics of patients and controls

	Patients, n (%)		
	Prostate cancer	BPH	Controls, n (%)
Number	606	402	471
Age group, years			
⩽55	91 (15)	18 (4.5)	30 (6.4)
56–60	112 (18.5)	61 (15.2)	69 (14.6)
61–65	131 (21.6)	82 (20.4)	108 (22.9)
66–70	155 (25.6)	94 (23.4)	128 (27.2)
⩾71	117 (19.3)	147 (36.6)	136 (28.9)
Father with prostate cancer			
No	500 (87.3)	361 (93.5)	428 (93.7)
Yes	73 (12.7)	25 (6.5)	29 (6.3)
Brother with prostate cancer			
No	374 (88.6)	252 (90.3)	320 (93.6)
Yes	48 (11.4)	27 (9.7)	22 (6.4)
Residential status			
Urban area	449 (74.5)	287 (71.9)	330 (70.8)
Rural area	154 (25.5)	112 (28.2)	136 (29.2)
Country of birth			
Australia	337 (62.5)	226 (59.8)	289 (64.2)
UK	138 (22.9)	90 (23.8)	93 (20.7)
Others	88 (14.6)	62 (16.4)	68 (15.1)

BPH, benign prostatic hyperplasia.

found no significant associations between occupational exposure to other toxic metals, wood dusts, mineral oils, PAHs and prostate cancer; all ORs were close to null value (table 2). Adjustment for residential place (rural *v* urban) and family history did not change appreciably the results.

For BPH (table 2), a non-substantial exposure to toxic metals was associated with a 39% increased risk of developing BPH (OR 1.39, 95% CI 1.10 to 1.84). A non-statistically significant association was found between exposure to PAHs other than diesel exhaust and BPH (OR 1.20, 95% CI 0.91 to 1.58). Non-significant protective associations were seen with mineral and other oils. We found no significant associations between BPH and exposure to other metals, woods, pesticides, herbicides, fertilisers and diesel fumes. All ORs were adjusted for age, family history of prostate cancer and residential place, and the results remained essentially unchanged.

After excluding 28 (7%) patients with BPH with mild urinary tract symptoms and 133 (28%) controls with moderate and severe symptoms, there were no appreciable changes in ORs or CIs. Analyses for both BPH and prostate cancer by confidence, level, duration and frequency of each of the chemical exposures were carried out separately and these did not disclose any strong associations.

To ensure a more clearly unexposed comparison group, a model containing age, and any exposure to metals, pesticides, fertilisers, PAHs and oils was run for both BPH and prostate cancer. For both outcomes, the ORs for each of the exposures were virtually unchanged from the values in table 2.

DISCUSSION

Our study used state-of-the-art assessment methods for occupational exposure to examine several a priori hypotheses about occupational causes of prostate cancer and the same exposures for BPH. Several investigators have examined the risk of prostate cancer in relation to specific job titles or industry groups, and have suggested exposures of interest associated with that job title.[25–29] In this study, we examined the risk associated with exposure to specific agents experienced across several industries. We found no statistically significant relationships for prostate cancer, although there were non-statistically significant increased risks of prostate cancer with toxic metals and other oils, and protective associations with organophosphate pesticides and natural fertilisers. Similarly, there was a lack of consistent relationships with toxic metals and PAHs for BPH. Diesel exhaust, non-toxic metals and wood dust were not associated with prostate cancer or BPH in our study.

Study advantages

The major advantage of this study was the detailed, case-by-case exposure assessment so that exposures common to several industries were personally attributed to specific individuals. This cannot be done using registry data or from a job–exposure matrix. It was thus possible to examine the risk associated with specific exposures for individuals such as farmers who may have several different exposures. Other advantages are the relatively large numbers of cases and controls, and the fact that the study was population based, not clinic based. All hypotheses were specified a priori and were based on previous studies. For this reason we did not adjust for multiple comparisons.

Study limitations

The major disadvantage of this study was the relatively low response fraction, especially for controls. We were unable to examine the characteristics of the participants and non-participants in order to determine whether there was any selection bias.

The prevalence of occupational exposures was relatively low—that is, CIs were quite wide, especially for the subgroupings of chemicals. Some men may have had several exposures, but numbers of such patients were too small to examine this reliably.

Non-differential misclassification may have arisen from several sources, and would be likely to reduce the strength of any observed association.[30] Firstly, although the exposure assessment was carried out with great care, there may be some misclassification due to missing information on jobs or incomplete knowledge of all exposures in all jobs by the assessor. This is particularly so as prostate cancer is usually diagnosed late in life (45% diagnosed after age 70 years),[31] and may possibly have a long latent period. Occupational exposures from a long time ago may be more likely to be forgotten or misclassified.

Prostate cancer is also relatively common; an autopsy study on men aged >50 years dying of causes other than prostate cancer reported prevalence as high as 30%.[32] Therefore, if only a small proportion of the identified cancers were occupationally related, it might be difficult to show the association.

Men in this study had an average of eight jobs. Thus, it is likely that they experienced a range of exposures. Most of these exposures are not known to be associated with an increased risk of prostate cancer. We examined only those exposures where there was some previous evidence of an association from the scientific literature.

Metals

We found toxic metal exposure to be weakly associated with BPH, but not with prostate cancer. Of these, cadmium is hypothesised to be associated with prostate cancer, mainly on the basis of laboratory evidence.[33] However, a recent review concluded that the epidemiological evidence for cadmium as a risk factor for prostate cancer was not convincing, possibly because of poor assessment methods.[34] Whether cadmium concentration is increased in BPH tissue compared with normal prostate tissue is being debated.[35–37] In our study, all men who

Table 2 Association between prostate cancer and benign prostatic hyperplasia (BPH) and exposure to different chemicals over entire working time, adjusted for age

		Prostate cancer			BPH		
Chemicals and exposure	**Controls, n (%)**	**Cases, n (%)**	**p Value***	**Age adjusted OR (95% CI)**	**Cases, n (%)**	**p Value***	**Age adjusted OR (95% CI)**
Metals							
Toxic metals							
Not exposed	291 (62.4)	354 (58.7)	0.24	1.00 (reference)	219 (55.4)	0.06	1.00 (reference)
Non-substantial†	161 (34.5)	236 (39.1)		1.25 (0.96 to 1.61)	167 (42.3)		1.39 (1.10 to 1.84)
Substantial‡	14 (3)	13 (2.2)		0.79 (0.36 to 1.72)	9 (2.3)		0.81 (0.34 to 1.93)
Other metals							
Not exposed	255 (54.7)	312 (51.7)	0.58	1.00 (reference)	217 (54.9)	0.40	1.00 (reference)
Non-substantial	180 (38.6)	252 (41.8)		1.16 (0.89 to 1.49)	160 (40.5)		1.05 (0.79 to 1.39)
Substantial	31 (6.7)	39 (6.5)		1.10 (0.66 to 1.82)	18 (4.6)		0.66 (0.36 to 1.22)
Inorganic arsenic							
Not exposed	403 (86.5)	523 (86.9)	0.85	1.00 (reference)	339 (86)	0.85	1.00 (reference)
Non-substantial	63 (13.5)	79 (13.1)		1.10 (0.73 to 1.50)	55 (14)		1.00 (0.67 to 1.48)
Any metals							
Not exposed	187 (40.1)	230 (38.1)	0.68	1.00 (reference)	149 (37.7)	0.37	1.00 (reference)
Non-substantial	245 (52.6)	329 (54.6)		1.13 (0.86 to 1.48)	224 (56.7)		1.15 (0.86 to 1.53)
Substantial	34 (7.3)	44 (7.3)		1.12 (0.79 to 1.60)	22 (5.6)		0.79 (0.44 to 1.42)
Wood dust							
Not exposed	299 (64.2)	383 (63.3)	0.80	1.00 (reference)	249 (63.0)	0.94	1.00 (reference)
Non-substantial	156 (33.5)	205 (34)		1.06 (0.82 to 1.38)	136 (34.4)		1.05 (0.79 to 1.40)
Substantial	11 (2.4)	16 (2.7)		1.17 (0.53 to 2.58)	10 (2.5)		1.11 (0.46 to 2.67)
Pesticides							
Organophosphate							
Not exposed	428 (91.8)	567 (94)	0.16	1.00 (reference)	371 (93.9)	0.24	1.00 (reference)
Non-substantial	38 (8.2)	36 (6)		0.69 (0.43 to 1.12)	24 (6.1)		0.73 (0.43 to 1.24)
Organochlorines							
Not exposed	454 (97.4)	592 (98.2)	0.40	1.00 (reference)	387 (98)	0.59	1.00 (reference)
Non-substantial	12 (2.6)	11 (1.8)		0.76 (0.33 to 1.75)	8 (2)		0.77 (0.31 to 1.91)
Phenoxy herbicides							
Not exposed	435 (93.3)	563 (93.4)	0.99	1.00 (reference)	376 (95.2)	0.25	1.00 (reference)
Non-substantial	31 (6.7)	40 (6.6)		1.00 (0.61 to 1.63)	19 (4.8)		0.72 (0.40 to 1.29)
Other herbicides							
Not exposed	434 (93.1)	558 (92.5)	0.71	1.00 (reference)	374 (94.7)	0.35	1.00 (reference)
Non-substantial	32 (6.9)	45 (7.5)		1.03(0.62 to 1.66)	21 (5.3)		0.76 (0.43 to 1.34)
Other pesticides							
Not exposed	448 (96.1)	587 (97.3)	0.26	1.00 (reference)	382 (96.7)	0.65	1.00 (reference)
Non-substantial	18 (3.9)	16 (2.7)		0.69 (0.34 to 1.37)	13 (3.3)		0.81 (0.39 to 1.69)
Any pesticides							
Not exposed	414 (88.8)	535 (88.7)	0.95	1.00 (reference)	359 (90.9)	0.32	1.00 (reference)
Non-substantial	52 (11.2)	68 (11.3)		1.02 (0.69 to 1.50)	36 (9.1)		0.79 (0.50 to 1.23)
Fertilisers							
Synthetic							
Not exposed	358 (77)	471 (78.1)	0.66	1.00 (reference)	306 (77.7)	0.81	1.00 (reference)
Non-substantial	107 (23)	132 (21.9)		0.99 (0.74 to 1.32)	88 (22.3)		0.96 (0.69 to 1.32)
Natural							
Not exposed	446 (95.7)	587 (97.3)	0.14	1.00 (reference)	384 (97.2)	0.24	1.00 (reference)
Non-substantial	20 (4.3)	16 (2.7)		0.66 (0.34 to 1.30)	11 (2.8)		0.66 (0.31 to 1.40)
Any fertilisers							
Not exposed	351 (75.5)	467 (77.4)	0.45	1.00 (reference)	304 (77.2)	0.57	1.00 (reference)
Non-substantial	114 (24.5)	136 (22.6)		0.95 (0.71 to 1.26)	90 (22.8)		1.39 (0.70 to 2.76)
Oils							
Mineral oils							
Not exposed	229 (49.1)	297 (49.3)	0.58	1.00 (reference)	202 (51.1)	0.32	1.00 (reference)
Non-substantial	181 (38.8)	245 (40.6)		1.11 (0.86 to 1.45)	158 (40)		0.96 (0.72 to 1.29)
Substantial	56 (12)	61 (10.1)		0.89 (0.59 to 1.33)	35 (8.9)		0.68 (0.43 to 1.08)
Other oils							
Not exposed	420 (90.1)	532 (88.2)	0.21	1.00 (reference)	361 (91.4)	0.12	1.00 (reference)
Non-substantial	27 (5.8)	51 (8.5)		1.54 (0.95 to 2.51)	27 (6.8)		1.13 (0.65 to 1.98)
Substantial	19 (4.1)	20 (3.3)		0.87 (0.46 to 1.67)	7 (1.8)		0.42 (0.17 to 1.01)
Any oils							
Not exposed	225 (48.3)	285 (47.3)	0.44	1.00 (reference)	197 (49.9)	0.08	1.00 (reference)
Non-substantial	169 (36.3)	238 (39.5)		1.19 (0.91 to 1.56)	157 (39.7)		1.04 (0.77 to 1.39)
Substantial	72 (15.5)	80 (13.3)		0.93 (0.64 to 1.35)	41 (10.4)		0.62 (0.40 to 1.00)
Polyaromatic hydrocarbons (PAH)							
Diesel fumes							
Not exposed	217 (46.6)	294 (48.8)	0.70	1.00 (reference)	196 (49.6)	0.67	1.00 (reference)
Non-substantial	213 (45.7)	260 (43.1)		0.92 (0.71 to 1.19)	170 (43.0)		0.88 (0.66 to 1.16)
Substantial	36 (7.7)	49 (8.1)		1.07 (0.67 to 1.72)	29 (7.3)		0.89 (0.53 to 1.51)
PAH							
Not exposed	233 (50)	311 (51.6)	0.56	1.00 (reference)	182 (46.1)	0.13	1.00 (reference)
Non-substantial	210 (45.1)	270 (44.8)		1.01 (0.78 to 1.30)	201 (50.9)		1.20 (0.91 to 1.58)

Table 2 Continued

Chemicals and exposure	Controls, n (%)	Prostate cancer			BPH		
		Cases, n (%)	p Value*	Age adjusted OR (95% CI)	Cases, n (%)	p Value*	Age adjusted OR (95% CI)
Substantial	23 (4.9)	22 (3.6)		0.79 (0.43 to 1.46)	12 (3)		0.66 (0.32 to 1.37)
Any PAH							
Not exposed	183 (39.3)	230 (38.1)	0.74	1.00 (reference)	144 (36.5)	0.17	1.00 (reference)
Non-substantial	227 (48.7)	307 (50.9)		1.11 (0.86 to 1.45)	215 (54.4)		1.18 (0.88 to 1.57)
Substantial	56 (12)	66 (10.9)		1.02 (0.67 to 1. 53)	36 (9.1)		0.81 (0.50, 1.30)

BPH, benign prostatic hyperplasia; PAH, polyaromatic hydrocarbon.
*p Values from χ^2 tests
†Patients probably exposed to the substances at a medium or high level for more than half their working time for a total of >5 years.
‡Exposed to the substance, but not at a substantial level.

reported soldering were classified as exposed to toxic metals because of the lead exposure involved in this task. Silver solder can contain 20% cadmium, but this is really a brazing technique. Mechanics, machinists, welders, electricians and plumbers were asked whether they carried out welding, brazing and flame cutting tasks that could result in cadmium exposure. None of the workers identified working on cadmium-plated steel. Brazing was carried out in 21 jobs by eight men; none of them identified working on a metal alloy containing cadmium. The use of silver solder was reported in 10 jobs by 4 men (2 controls and 2 patients with prostate cancer). Owing to these small numbers, we were unable to examine occupational cadmium exposure separately.

Lubricating oils and greases

Lubricating oils and greases were found to be associated with prostate cancer in a large community-based case–control study in Montreal, Canada,[13] and in the auto industry.[29] In this second study, the oils included mineral oils, soluble fluids and synthetic fluids.[29] Like Boers *et al*,[12] we found association neither for oils overall nor specifically for mineral oils. We found that workers exposed at a non-substantial level to other types of oils had a 50% increased risk of prostate cancer; however, this increase was not statistically significant, and no dose–response pattern seen. A recent study suggested that soluble metalworking fluids were associated with prostate cancer, but only for exposure that occurred ⩾25 years before the cancer developed.[29]

Pesticides

We found non-significant protective associations for prostate cancer and BPH with organophosphate and organochlorine insecticides. Two recent cohort studies have found no association[38] and a non-significant protective association[39] with dichlorodiphenyltrichloroethane—an organochlorine insecticide. A prospective study on nearly 60 000 men in The Netherlands found a statistically significant protective association with all pesticide exposure.[12] The authors argued that their results might be biased because of the heterogeneity of pesticide exposure of farmers. However, our study avoided this problem by asking the men directly whether they had used pesticides and how often, and then using expert opinion based on evidence to determine which pesticides were used. They also said that perhaps diet was better among farmers and that this had confounded the results.[12] However, in our study, we found no difference between patients and controls with regard to overall intake of meat, vegetables and fruit (data not shown). An alternative explanation for the apparent protective effect of pesticides is that it is an effect of exposure to the sun mediated through higher vitamin D levels, which are thought to be possibly protective against prostate cancer.[40] Or perhaps rural residents are less exposed to other unidentified prostate carcinogens.

A recent meta-analysis of studies on pesticide applicators found an increased meta-rate ratio for prostate cancer, although there was considerable heterogeneity among the studies.[25] Most of our patients who were exposed to pesticides were farmers. Farmers experience a variety of exposures, including pesticides, fertilisers, mineral oils, wood and diesel exhaust, and the combination of exposures would be different for each farmer. In addition, the pesticides used in Australia and the conditions of application may well be different from those in the North American and European studies in the meta-analysis.

Diesel exhaust and other PAHs

Diesel exhaust was not found to be associated with prostate cancer in our study nor in a recent large prospective study.[12] A small case–control study in Germany found a strong relationship with diesel exhaust, but this study used controls with histological proof of no cancer or BPH, which may not be representative of the general male population.[8] The Montreal case–control study found prostate cancer to be associated with liquid fuel combustion products, as well as with PAHs from coal and diesel exhaust.[13] Although we found an association between BPH and non-substantial exposure to PAHs other than diesel exhaust, there was no dose–response relationship seen, so it may be just a chance finding.

Fertilisers

We found a non-statistically significant protective association for prostate cancer with natural fertilisers but not with

Main messages

- Little is known about the causes of prostate cancer generally and research on occupational causes has produced inconsistent results.
- Even less is known about whether occupation contributes to causing benign prostatic hyperplasia.
- We used state-of-the-art assessment for occupational exposure and found little evidence of a major effect of the examined occupational exposures on either disease.

Policy implication

There is little evidence from this study, or from the literature, that occupational exposures are a major cause of the burden of prostate cancer.

synthetic fertilisers. The association between BPH and natural fertilisers was also weakly protective, but based on small numbers. A previous study found increased risk of prostate cancer in workers in a nitrate fertiliser plant (a synthetic fertiliser), but there was no association with nitrate exposures within the plant.[41] Of the 16 men in our study who had substantial exposure to natural fertilisers, all but one worked on farms or plant nurseries not in fertiliser manufacture.

Wood dust

Wood dust has been inconsistently linked with prostate cancer,[13 42] and our study adds to the evidence against it being a risk factor for prostate cancer or BPH.

CONCLUSION

The association of prostate cancer and BPH with several occupational exposures, including metals, PAHs, oils, pesticides, fertilisers and wood were examined in this study. We found no evidence that any of these exposures were strong occupational risk factors for either prostate cancer or for BPH.

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