

found in the men with exposure to pitch and tar (3 observed, 0.05 expected, $P < 0.001$) and an excess of tumours of the stomach was noted in the men whose exposure was uncertain (3 observed, 0.59 expected, $P < 0.05$). The first excess is to be anticipated on *a priori* grounds, but no explanation can be offered for the excess of stomach tumours in the absence of knowledge of exposure to possible carcinogens.

The finding of an excess number of second primary tumours of the larynx and bronchus in the oil exposed group is consistent with the notion that oil mist is acting as a carcinogen. To test this hypothesis, the occupations of all the male cases of carcinoma of the bronchus and larynx registered at the BRCR between 1967 and 1969 were categorized according to the Registrar General's classification of occupation. The proportion of men in each of the occupational orders was compared with the number of men in each order as a proportion of the total work force in the region during the same years, the assumption being that if tumours of these sites were related to oil exposure, then a significant excess should be observed in those orders which included men in oily jobs (order V and VII). An overall excess of bronchial tumours was noted in order V ($P < 0.001$) but not in order VII. Neither order showed an excess of laryngeal tumours. The excess of bronchial tumours in order V was due to an excess in 2 categories of workers, metal furnace men, and smiths and forgers.

The 2 occupations which between them contributed over half the cases of second primary tumours in the index series were the toolmakers and machine operators. In the regional analysis, machine operators were represented according to expectation, but the toolmakers showed a significant *deficit* of both bronchial and laryngeal tumours ($P < 0.001$ and < 0.05 respectively).

TABLE I

Site	Expected number	Observed number	<i>P</i>
All sites	8.24	28	< 0.001
Larynx	0.13	2	< 0.05
Bronchus	2.64	11	< 0.001
Skin	0.77	11	< 0.001
Remainder	4.70	4	n.s.

Expected and observed number of subsequent primary tumours in men with oil exposure.

Thus, if oil mist is carcinogenic, this effect appears to be exerted on only a sub-set of the exposed population composed, presumably, of persons having an enhanced susceptibility, the basis of which is unclear.

INDUSTRIAL BLADDER CANCER—THEN AND NOW. H. G. PARKES, British Rubber Manufacturers' Association, Birmingham.

The first cases of bladder cancer which were recognized as being of industrial origin made their appearance at the end of the last century. In the years that followed, the casual association with occupational exposure to aromatic amines was gradually understood and explored until it was possible, by 1950, not only to identify the principal carcinogens (e.g. β -naphthylamine benzidine and 4-aminodiphenyl) but also to make some quantitative evaluation of their relative potency. Thereafter, use of these chemical carcinogens was generally discontinued in favour of other and safer compounds. Industrial bladder tumour was recognized as a prescribed industrial disease in 1953 and regulatory control of the principal carcinogens was introduced with the Carcinogenic Substances Regulations in 1967.

Despite all this, it remains true to say that new cases of industrial bladder cancer are still making their appearance today—some 80 years after they were first reported. This observation does not appear to evoke the response of concern and profound shock which it would seem to merit.

Due to the long latency of these industrial cancers a very large population remains at risk today and only a small proportion (some 20,000 in Britain) have any form of routine surveillance or screening available to them. Many more deaths will yet take place among the victims of this industrial disease before the hazard can be said to have been eliminated.

To what extent are steps being taken to ensure that there is no repetition of this tragedy? Is it not possible that even today there are within industry chemicals not yet recognized for their carcinogenic potential which could be putting yet another industrial population at risk? The means of detecting such a threat and of preventing the long-term consequences are only now beginning to be understood and to be implemented. There

are at least 3 essential elements in any such prevention programme: (1) A system for routine toxicity and carcinogenicity checking of all industrial chemicals currently in use or likely to be introduced in the future; (2) provision for routine environmental monitoring of factory atmospheres for the presence of known or suspected pollutants and (3) careful and continuing epidemiological control of workers who may be at risk. Above all else, these elements will require an urgent revision and improvement of present systems of record keeping, maintenance and linkage, not only in the factory but also in the community.

NICKEL AND CADMIUM CARCINOGENESIS. G. KAZANTZIS, Department of Community Medicine, Middlesex Hospital, London.

Workers at a nickel refinery in Wales were first noted to have an unusually high mortality from cancer of the respiratory tract some 30 years after the plant had become operational. A proportional mortality study by Doll (*Br. J. Indust. Med.*, 1958, **15**, 217) showed a five-fold increase in deaths from lung cancer and a 150-fold increase from nasal cancer in these men. While at first the Mond nickel process involving exposure to nickel carbonyl gas was thought to have been responsible, a similarly high mortality experience was found among refinery workers in Ontario where the Mond process had not been used (Mastromatteo, *J. occup. Med.* 1967, **9**, 127). In both plants mortality experience fell to that expected from national mortality data in men first employed after changes had been made which involved drastic reduction in exposure to nickel.

Experimental work supports the epidemiological evidence for the carcinogenic activity of nickel. Malignant tumours have been produced in several animal species by nickel as the powdered metal and by a variety of nickel compounds introduced by different routes. These, with possible mechanisms of nickel carcinogenesis, have been reviewed by Sunderman (*Fd Cosmet. Toxicol.*, 1971, **9**, 105) who provided evidence for inhibition by nickel carbonyl of DNA dependent RNA synthesis.

Cadmium is a biologically active metal responsible for emphysema and renal tubular dysfunction following long-term exposure. A

survey of men who had been occupationally exposed to cadmium oxide dust for a minimum period of one year revealed an increased mortality from prostatic carcinoma (Kipling and Waterhouse, *Lancet*, 1967, **i**, 730). No further epidemiological evidence incriminating cadmium in human carcinogenesis has been produced. Traces of cadmium are present in cigarette smoke and smokers accumulate more cadmium in kidney, liver and lung than non-smokers. However, a causal role for cadmium in bronchogenic carcinoma has not been postulated.

A carcinogenic potential for cadmium has been demonstrated in several experimental animal studies. Finely divided cadmium metal injected into the thigh muscle of the rat gave rise to rhabdomyosarcoma (Heath and Daniel, *Br. J. Cancer*, 1964, **18**, 124). Cadmium sulphide and cadmium oxide injected subcutaneously and intramuscularly led to fibrosarcoma at the injection site with metastases in a high proportion of the dosed rats (Kazantzis and Hanbury, *Br. J. Cancer*, 1966, **20**, 190) and repeated injections of cadmium sulphate were followed by testicular atrophy and interstitial cell tumours of the testis (Haddow *et al.*, *Br. J. Cancer*, 1964, **18**, 667). No prostatic changes were observed following the repeated subcutaneous injection of cadmium sulphate or following its long-term administration in drinking water in concentrations below those required to demonstrate a toxic effect in the rat (Levy *et al.*, *Ann. occup. Hyg.*, 1973, **16**, 111).

Further epidemiological surveillance is required before the question of the carcinogenic potential of cadmium in man can be decided.

ASBESTOS CARCINOGENESIS. J. C. WAGNER, Medical Research Council, Penarth.

Carcinoma of the lung and diffuse mesotheliomata of the pleura and peritoneum have occurred in people exposed to asbestos dust. Various types of asbestos are used in industry and studies have been undertaken to establish whether these tumours are associated with exposure to specific types of fibre.

Carcinoma of the lung was first reported in cases of asbestosis in 1935. The incidence of these tumours has increased rapidly and by 1964, 60% of those workers in the United Kingdom diagnosed as having asbestosis to a