CORRESPONDENCE

Exposure to asphalt or bitumen fume and renal disease

EDITOR,—The correspondence from Dittmer and Armitage[1] provides further support for a causal association between exposure to various hydrocarbons and the development of renal disease. Since 1912, some case reports,[2–4] case-control studies,[5] and cross sectional[6] studies together with animal experiments[7 8] have provided compelling evidence for a causative role for hydrocarbon exposure in the development of both tubular and glomerular lesions.

We now report the case of a road worker exposed to asphalt and bitumen fumes who presented in 1990 at the age of 36 with nephrotic syndrome. He was then normotensive, had proteinuria with 24 hour urinary protein of 12.2 g, showed some clinical oedema, and his renal biopsy was consistent with a diagnosis of stage 2 membranous glomerulonephritis. Later that year he presented with an unexplained deterioration in renal function. This followed several weeks of abdominal pain, and he then had haematuria without pyuria, serum creatinine of 298 compared with his previously normal value of 85 µmol/l, and a marked deterioration in his renal biopsy with 20 glomerular profiles per section and tubulointerstitial scarring occupying 40% of the biopsy.

Before developing renal disease, the patient had been employed as a road worker for more than 10 years, during which time he was repeatedly exposed to intermittent but high concentrations of asphalt or bitumen fumes. Since the diagnosis of nephrotic syndrome due to membranous glomerulonephritis, the patient stopped being exposed to asphalt and bitumen fumes. Subsequent assessments of proteinuria have shown a reduction of up to 50% from 12 to 5 g/day. However, he currently is hypertensive with serum creatinine 170 µmol/l, urea 10.4 mmol/l, serum albumin 30 mmol/l, and 24 hour urinary excretion of protein 5.0 g.

Searches of the scientific literature in 1990 and subsequently have not found any specific references to exposure to asphalt or bitumen and renal disease. We therefore investigated the issue further by means of (*a*) detailed fume analyses, and (*b*) a study of the renal health of road workers exposed to asphalt or bitumen.

It was clear from the fume analyses that exposures in this industry include a wide range of aromatic and aliphatic hydrocarbons. Time weighted average exposures ranged from 0.4 to 8.9 mg/m^3 measured as total organic fume (not including inorganic particulates), but short term or peak fume exposures were as high as 300–900 mg/m^3. During all his years exposed to these fumes, the patient had never been provided with or worn respiratory protective equipment.

The study of renal health included 92 people regularly exposed to asphalt or bitumen fumes as road workers, 38 hard rock quarry workers not occupationally exposed to hydrocarbons, and 43 office workers also not exposed to hydrocarbons.

Each participant was given a questionnaire which included questions about occupational and recreational exposures and medical history including renal disease. Urine and blood samples were collected for urinary chemistry, blood biochemistry, and microscopic analyses. Any person with an abnormal finding on blood or urine analyses were retested and examined by a nephrologist to assess the presence or otherwise of renal disease.

The criteria which determined an abnormal test result were as follows: (*a*) persistently raised serum creatinine >120 µmol/l; (*b*) persistently raised serum urea >7.5 mmol/l; (*c*) persistent microscopic haematuria or pyuria; (*d*) 24 hour urinary protein >150 mg / day; or (*e*) corrected creatinine clearance <90 ml/min.

The presence of renal disease was determined as pre-existing or idiopathic according to the following criteria. Pre-existing renal disease: (*a*) family history or history of renal disease (*b*) abnormal renal ultrasound. Idiopathic renal disease: (*a*) no known cause for abnormalities; (*b*) abnormal creatinine, urea, and creatinine clearance; (*c*) abnormal proteinuria; or (*d*) abnormal urinalysis—haematuria or pyuria.

The findings of the study are summarised in tables 1–3.

We concluded from this study that: (*a*) workers regularly exposed to asphalt or bitumen fumes were far more likely to have evidence of early stage renal disease than those working in a quarry or office; (*b*) workers regularly exposed to asphalt or bitumen fumes were far more likely to have at least one abnormal renal function test than those working in a quarry or office; and (*c*) the renal dysfunction was non-specific, but the overall findings were consistent with previous findings—such as those from the similar study done by Yaqoob *et al*.[6]

We think that chronic glomerulonephritis and chronic tubulointerstitial nephritis are renal diseases which may result from exposure to hydrocarbons—such as those experienced from asphalt or bitumen fumes generated during road making.

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Table 1 Age and blood pressure

	No exposure		*Exposure to bitumen or asphalt*	*All*
	Office	*Quarry*		
People (n)	43	38	92	173
Age (mean)	39	32	35	36
BP (mean systolic)	133	131	133	133
BP (mean diastolic)	86	83	83	84

Table 2 Renal disease

	No exposure		*Exposure to bitumen or asphalt*	*All*
	Office	*Quarry*		
Number of people	43	38	92	173
Pre-existing renal disease (n (%) NS)	4 (9.3)	2 (5.3)	1 (1.1)	7 (4.0)
Idiopathic renal disease (%)**	0 (0)	0 (0)	12 (13.0)	12 (6.9)

**p<0.01.

Table 3 Renal function

	No exposure		*Exposure to bitumen or asphalt*	*All*
	Office	*Quarry*		
People (n)	43	38	92	173
Haematuria	4	2	2	8
Proteinuria	0	0	17	17
Raised creatinine	0	0	8	8
At least one abnormality (n (%))**	4 (9.3)	2 (5.3)	24 (26.1)	30 (17.3)

**p<0.01.

1 Dittmer ID, Armitage AJ. Prolonged exposure to an epoxy resin leading to interstitial nephritis. *Occup Environ Med* 1997;**54**:766.
2 Anderson K. Acute nephritis due to turpentine absorbed by the skin. *BMJ* 1912;**3**:881.
3 Beirne GV, Brennan JT. Glomerulonephritis associated with hydrocarbon solvents mediated by antiglomerular basement membrane antibody. *Arch Environ Health* 1972;**25**:365–9.
4 Cagnoli L, Casanova S, Pasquali S, *et al*. Relation between hydrocarbon exposure and the nephrotic syndrome. *BMJ* 1980;**280**:1068–9.
5 Bell GM, Gordon ACH, Lee P, *et al*. Proliferative glomerulonephritis and exposure to organic solvents. *Nephron* 1985;**40**:161–5.
6 Yaqoob M, Bell GM, Stevenson A, *et al*. Renal impairment with chronic hydrocarbon exposure. *Q J Med* 1993;**86**:165–74.
7 Halder CA, Warne TM, Hatoum NS. Renal toxicity of gasoline and related petroleum naphthas in male rats. In: Mehlman MA, Hemstreet CPIII, Thorpe JJ, *et al*, eds. *Renal effects of petroleum hydrocarbons*. Princeton, New Jersey: Princeton 1984:73–88.
8 Easley JR, Holland JM, Gipson IC, *et al*. Renal toxicity of middle distillates of shale oil and petroleum in mice. *Toxicol Appl Pharmacol* 1982;**65**:84–91.

Authors' reply—The report of Douglas and Carney of a further case of renal disease associated with hydrocarbon exposure, together with their cross sectional study of those with prolonged exposure to bitumen and ashphalt further strengthens the case for an association between renal disease and hydrocarbon exposure. Yaqoob *et al* have also convincingly shown, that in particular, proteinuria may be associated with hydrocarbon exposure.[1]

This highlights the need for a careful occupational and social history to be taken at the time of presentation. This case also highlights the need for performing a renal biopsy in adults presenting with unexplained proteinuria. If interstitial nephritis is found then a short course of steroids may result in a dramatic improvement in renal function as we noted in the case of our patient exposed to

epoxy resin fumes. If there was evidence of significant renal damage then it may also be wise to counsel the patient to avoid further contact with the substance. Indeed in our case, the patient found that his general health improved dramatically when direct contact with the resins stopped. The issues of general industrial health and possible compensation or litigation also need to be considered.

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1 Yaqoob M, Bell G M, Stevenson A, *et al*. Renal impairment with chronic hydrocarbon exposure. *Q J Med* 1993;**86**:165–74.

Cancer risk in the rubber industry: a review of recent epidemiological evidence

EDITOR,—Although the comprehensive review of the rubber industry reported by Kogevinas *et al*[1] only considers papers published after 1982, several of these studies relate to groups of workers from much earlier eras—for example, 1910 for the German study,[2] 1946 for the British Rubber Manufacturers Association (BRMA)[3 4] and Veys studies.[5 6] By considering the findings of these earlier rubber workers along with studies of more recent groups of workers we are getting a picture of 80 years of cancer experience in the industry, which is not the same as the situation that exists today.

It should also be borne in mind that the very large cohort studies—such as the 34 000 workers in the BRMA study[3 4]—have very much greater statistical power than those of the smaller studies, in which confounding factors and the role of chance are more difficult to evaluate. This does not seem to have been fully taken into account and indeed Kogevinas *et al* tell us that they have not "paid much attention to statistical significance". This is disappointing as is the omission of a full meta-analysis of the studies, which if it had been carried out, would have added considerable weight to their conclusions.

In general, the review would seem to endorse and reflect the evaluation by the International Agency for Research on Cancer (IARC) of the industry in the 1987 monograph, supplement 7[7] of a moderate increase in risk of cancer at several different organ sites which are not consistently found in similar studies carried out in other parts of the world. Evidence suggests that this merely reflects alternative methods of handling and processing rubber and different sources of supply of raw materials around the world. The review, however, seems to give less endorsement to the IARC evaluation in 1982, monograph 28,[8] that there was "sufficient evidence for a causal association" between work in the rubber industry and developing bladder cancer and leukaemia.

Although it is interesting to look at the worldwide experience of rubber workers, from the point of view of the United Kingdom industry, domestic findings are more relevant, given that they are based on the largest and most closely researched populations of rubber workers anywhere in the world. These studies include the Health and Safety Executive study (40 000 workers),[9] the BRMA study[3 4] (34 000 workers), and the Veys studies[5 6] (14 000 workers) followed up for 40 years. The findings from this domestic epidemiology enable us to conclude, with considerable confidence, that in the United Kingdom occupational leukaemia was never a factor and that the problems of bladder cancer were eliminated in the 1940s with the discontinued use of chemicals contaminated with β-naphthylamine. These studies also showed a small but nevertheless significant excess of stomach, lung, pharyngeal, and oesophageal cancers. With more detailed analysis, however, and consideration of confounding and socioeconomic factors, the occupational importance of these excesses seems to be less clear as time goes on. Geographical and urban confounding factors and a lack of a clear time-dose response also lessen the possibility of occupational causes.

Having expressed our confidence in the United Kingdom findings, I re-emphasise that they are largely based on results from an earlier generation of rubber workers and that their experience may not be the same as those currently employed in a modern day rubber factory.

So that we may investigate more recent experience, the BRMA initiated a further collaborative project with Birmingham University, to carry out a new study of its members' employees. The collection of data for this study was completed last year and it includes nearly 10 000 male and female workers with at least 12 months of employment and who were first employed between 1982 and 1991. This study involves 42 rubber factories engaged in manufacturing the full range of rubber goods. This cohort study will look at both cancer incidence and mortality and make full use of all available occupational hygiene and exposure data.

Examination of the health experience generated by the study to date will take place later this year to see if there is sufficient information for a full analysis to be carried out or whether it would be appropriate to delay this until more data are available.

Kogevinas *et al* have given an interesting and important overview of health hazards observed in rubber workers employed during the past 80 years and I agree with him that more relevant, modern, and comprehensive epidemiology is necessary if we are to obtain a true picture of the situation today.

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1 Kogevinas M, Sala M, Boffetta P, *et al*. Cancer risk in the rubber industry: a review of recent epidemiological evidence. *Occup Environ Med* 1998;**55**:1–12.
2 Weiland S, Mundt K, Keil U, *et al*. Cancer mortality among workers in the German rubber industry:1981–91. *Occup Environ Med* 1996;**53**:289–98.
3 Sorahan T, Parkes H, Veys C, *et al*. Cancer mortality in the British rubber industry: 1946–80. *Br J Ind Med* 1986;**43**:363–73.
4 Sorahan T, Parkes H, Veys C, *et al*. Cancer mortality in the British rubber industry: 1946–80. *Br J Ind Med* 1989;**46**:1–11.
5 Veys C. Bladder cancer as an occupational disease in the rubber industry: an in depth factory study to show the past extent of the risk and confirmation of its subsequent disappearance. *Progress in Plastics and Rubber Technology* 1992;**8**:1–14.
6 Veys C. Bladder cancer in rubber workers. *Progress in Plastics and Rubber Technology* 1995; **11**:86–7.
7 International Agency for Research on Cancer. *Monographs on the carcinogenic risk to humans. Overall evaluation of the carcinogenicity.An updating of IARC monographs.* Vols 1–42 (suppl 7). Lyon: IARC, 1987.
8 International Agency for Research on Cancer. *Monographs on the carcinogenic risk of chemicals to humans.* Vol 28. *The rubber industry*. Lyon: IARC, 1982.
9 Baxter PJ, Werner JB. *Mortality in the British rubber industries 1967–6*. London: Health and Safety Executive, 1980.

Authors' reply—We thank Straughan for his comments. We agree that some of the studies we reviewed provide a picture of 80 years of cancer experience in the industry, which is not the situation existing today (in industrialised countries). We tried to identify and report separately results for studies examining workers first employed after the 1960s. These studies did not clearly indicate the absence of an excess risk of cancer. Unfortunately the number of subjects and cancer deaths or cases in these studies is small and does not allow definite conclusions to be drawn yet.

Considerable heterogeneity exists between and within countries in exposure circumstances in the rubber industry. What we did in our review was to give an overall picture of risks in this industry. This overall picture does not apply to all countries, nor to all periods. However, an overall picture may highlight conditions that are not easily recognised at a local level. One example is the identification of an increased risk for laryngeal cancer, which had not been previously reported although it seemed consistent between centres. Another example refers to the statement by Straughan (and others), that the British studies do not indicate an excess risk for bladder cancer after the discontinuation of use of β-naphthylamine. What is usually meant is that there was no significant excess risk, which is correct. What can be distinguished, however, looking at the overall picture (see figure 1 of our review) is a small but consistent excess risk for bladder cancer even in studies conducted in relatively late periods. There is a lack of detailed exposure information in most studies but it is probable that β-naphthylamine was not used in these late periods. We agree with Straughan that it is difficult to exclude the possibility that the observed small excess risk is due to a late effect of early exposures.

The findings of the large BRMA study are, indeed, more stable (statistically) than those of studies in the Nordic or other countries, but they are not necessarily either more or less confounded than those of other studies. We understand Straughan's plea for a full meta-analysis in which large studies are not given the same weight as small studies. The variability of exposures over time, geography, and process argue against performing a meta-analysis which presumes homogeneity of exposure.

It is commendable that the BRMA has been and continues to be actively involved in examining risk of cancer among workers in the rubber industry. We do hope that the new study initiated by the BRMA will do justice to the concluding sentence of our paper: "The preventive measures taken in the rubber industry in recent years may decrease risks, but this has not been documented yet in epidemiological studies"

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Inhalation of ammonium nitrate fuel oil explosive (ANFO): and possible concomitant exposure

EDITOR,—Donoghue[1] reports on respiratory symptoms and rhonchi in a miner after exposure to ammonium nitrate fuel oil explosive (ANFO). As diesel fuel is the most commonly used fuel in ANFO the vapour he refers to might be components of diesel fuel. He excludes concomitant exposure to nitrogen dioxide because the inhalation occurred before any explosion took place. Although diesel powered machines are commonly used in underground work he does not discuss possible exposure to diesel exhaust. I have measured up to 15 ppm nitrogen dioxide during construction of a tunnel where the only known source was diesel exhaust. Exposure to such high concentration may contribute to respiratory symptoms and rhonchi. Therefore nitrogen dioxide should not be excluded as a concomitant causative factor. In a study of the contribution of gases from diesel exhaust and from the blasting cloud caused by the ANFO explosive during excavation of a tunnel, diesel exhaust contributed most to the total amounts of nitrogen dioxide in the tunnel.[2]

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1 Donoghue A.M. Inhalation of ammonium nitrate fuel oil explosive (ANFO). *Occup Environ Med* 1998;**55**:144.
2 Søstrand P, Lian K, Myran T. The contribution of inorganic gases from diesel exhaust and from the blasting cloud during excavation of a tunnel. The 3rd IOHA International Scientific Conference. *Occupational Hygiene Special Issue* 1997;**4**:1–13.

Occupational asthma due to amylase

EDITOR,—In a letter to you,[1] Hendrick points out my inadvertently overlooked report in *The Lancet* on allergy to α-amylase and papain,[2] and makes generous reference to my other work on enzymes.

For clarification, I should point out that the evidence, supported by the findings with amylase, that sensitisation and consequential symptoms may occur from proteases independently of proteolytic activity, must not obscure the fact that proteolytic mechanisms may cause other clinical and subclinical effects. As well as skin irritation, non-sensitised people may experience epistaxis or haemoptysis, whereas rhinorrhoea or asthma are more likely to be due to allergy. Propensity to cause proteolytic effects varies between different proteases, and susceptibility to such effects varies between people.[3] I think I have experienced such effects myself;[4] and possible long term consequences have been described.[5]

Although he concurs with the use of skin prick tests as an index of sensitisation, Kendrick expresses reservations about my evidence of causality in respect of chest symptoms from α-amylase. This is understandable if I relied solely on his condensation of an already condensed report. Although my report derived from a comprehensive ongoing investigation, I had hoped I had summarised sufficient information to make my point.

His reservations seem to derive from the fact that papain was also handled in the workplace, and that some of those sensitised to α-amylase were also sensitised to papain.

At this factory papain and α-amylase were handled in pure form, seldom, and at different times and places. The association between handling one or other material and the development of symptoms was clear cut, and because of the short period of handling, with intervals of at least a month between these periods, there was time for symptoms to regress between exposures.

I had already validated a skin prick test for papain sensitivity,[6] and was able to use the same test material for papain, and a similar one for α-amylase. Positive prick test findings seemed to confirm the specificity and likely mechanism of the typically asthmatic symptoms from each enzyme.

Had I had any reasonable doubt as to causality I would not have published the warning. Happily, subsequent reports, including the excellent one by Aitkin *et al*,[7] of which Hendrick is a coauthor, which included inhalation challenge tests with fungal α-amylase,[7] have endorsed my conclusions and added further knowledge.

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1 Hendrick DJ. Occupational asthma due to amylase. *Occup Environ Med* 1998;**55**:361.
2 Flindt MLH. Allergy to a-amylase and papain. *Lancet* 1979;i:1407–8.
3 Flindt MLH. Variables affecting the outcome of inhalation of enzyme dusts. *Ann Occup Hyg* 1982;**26**:647–55.
4 Flindt MLH. Biological miracles and misadventures: identification of sensitisation and asthma in enzyme detergent workers. *Am J Ind Med* 1996;**29**:99–110.
5 Musk AW, Gandevia B. Loss of pulmonary elastic recoil in workers formerly exposed to proteolytic enzyme (alcalase) in the detergent industry. *Br J Ind Med* 1976;**33**:158–65.
6 Flindt MLH. Respiratory hazards from papain. *Lancet* 1978;i:430–43.
7 Aiken TC, Ward R, Peel ET, Hendrick DJ. Occupational asthma due to porcine pancreatic amylase. *Occup Environ Med* 1997;**54**:762–4.

Health of children born to medical radiographers

EDITOR,—There are increasing concerns that parental workplace exposure to potentially hazardous agents could affect the health of future offspring. In considering this, Roman *et al*[1] have developed a questionnaire based method for collecting data on reproductive outcome and child health which has been applied to a study population comprising (predominantly female) members of the College of Radiographers. I have recently had cause to revisit their report of this work in more detail and have several comments.

The study relies on postal questionnaires for details of adverse pregnancy outcomes, major congenital abnormalities, and malignancies, but only reports of cancer were validated by reference to national registration schemes and medical records. Comparisons with national cancer registration rates for England and Wales and congenital malformation rates derived from data compiled by the Liverpool Congenital Malformation Registry showed little evidence to suggest an increased risk for cancer or for major congenital malformations. Within specific systems no excess relative risk was evident for Down's syndrome, but two significantly increased relative risks were found—for "other musculoskeletal" malformations and for "chromosomal anomalies other than Down's syndrome"—both dominated by adverse outcomes reported by female radiographers. My particular interest is with the group of six cases of chromosome anomalies other than Down's syndrome.

Four cases of Turner's syndrome were reported by female radiographers, two being diagnosed before birth and the pregnancies terminated. The Turner phenotype, recognised in live born infants, is characterised by monosomy X and has a birth incidence in females of 1/2000–1/5000.[2] However, the frequency of monosomy X at conception is much higher, occurring in 1%–2% of all clinically recognised pregnancies.[3] Over 99% abort spontaneously, 70% of these between 11 and 14 weeks gestation.[4] The incidence of Turner's syndrome will vary considerably with different stages of pregnancy and this has implications for the calculation of expected numbers of cases. When evaluating Turner's syndrome in relation to aetiological influences it is important to have accurate information on the timing and method of diagnosis, and comparisons must be made with appropriate registry data. In around 80% of cases the X chromosome present is maternally derived[3] and therefore, by contrast with most cases of Down's syndrome, an error in meiotic non-disjunction cannot be attributed to the mother. Of those diagnosed after birth, some are mosaics with a normal 46 XX cell line as well as a 45 X line or a cell line with one normal and one abnormal X chromosome, and it has been suggested that mosiacism increases the likelihood of survival during pregnancy.[3] Such mosiacs are assumed to have arisen post-zygotically. Turner's syndrome is, therefore, most likely to occur due to an error in non-disjunction arising either during spermatogenesis or after fertilisation, and the origin of the error can often be determined by undertaking cytogenetic and molecular studies. Consequently, it is unlikely that maternal preconceptional exposure is relevant to the occurrence of the cases of Turner's syndrome reported to Roman *et al*[1] although it is possible that events immediately after conception could be implicated in the origin of any with a mosaic karotype. It is unfortunate that Roman *et al*[1] provide no karotypic data nor information on whether the mothers were working as radiographers when they conceived as this would have assisted in the interpretation of this association.

Two further pregnancies with chromosomal abnormalities were described—a gross chromosomal anomaly reported by a female radiographer and a trisomy 17 reported by a male radiographer—both of which were terminated. Referral to results of chromosomal studies would have provided karotypes and the possibility of the gross chromosomal anomaly being the result of a familial rearrangement could then have been explored. Trisomy 17 is rather a surprise as this is considered incompatible with embryo development[5] and has not, to my knowledge, been detected by antenatal diagnosis. There must be a possibility that this information is incorrect.

Aetiological mechanisms must be considered when assessing the biological plausibility

of epidemiological associations, and reliance on personal memory will not provide the detailed data which allow chromosomal abnormalities to be grouped in a meaningful way when exploring possible environmental influences. The study of Roman *et al*[5] illustrates the need to confirm diagnoses reported in questionnaires whenever possible, and to obtain any additional information that may assist in the scientific evaluation of epidemiological results.

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1 Roman E, Doyle P, Ansell P, *et al*. Health of children born to radiographers. *Occup Environ Med* 1996;**53**:73–9.
2 Zinn AR. Growing interest in Turner's syndrome. *Nat Genet* 1997;**16**:3–4.
3 Hassold T, Pettay D, Robinson A, *et al*. Molecular studies of parental origin and mosaicism in 45 X conceptuses. *Human Genet* 1992;**89**: 647–52.
4 Kline J, Stein Z. Environmental causes of aneuploidy: why so elusive? In: Dellarco VL, Voytek P E, Hollaender A, eds. *Anueploidy: etiology and mechanisms*. New York: Plenum Press, 1985:149–64.
5 de Grouchy J, Turleau C. *Clinical atlas of human chromosomes. 2nd ed*. New York: John Wiley, 1984.

CORRECTION

Older workers in the construction industry: results of a routine health examination and a five year follow up by V ARNDT, D ROTHENBACHER, H BRENNER, E FRAISSE, B ZSCHENDERLEIN, U DANIEL, S SCHUBERTH, T M FLIEDNER (1996;**53**:686–91).

The section entitled "Statistical methods: cross sectional study" (page 687) should read:

In the cross sectional study, crude prevalence and age adjusted (Mantel-Haenszel) relative prevalences (95% confidence intervals (95% CIs)) were calculated by occupational group for the following outcomes: age adjusted hearing loss at 1-6 kHz greater than 30 decibels (dB) in either ear, pathological findings at lung auscultation (which were defined as rales, rhonchi, or crackling), forced expiratory volume in one second (FEV_1) less than 80% predicted, diastolic blood pressure (DBP) ⩾95 mm Hg, abnormal findings in the electrocardiogram (ECG), body mass index (BMI) >27.8 kg/m^2, γ-glutamyl-transpeptidase (GGT) >28 U/l (measured at 25°C), reduced mobility of the spine (fingertips to floor test, Schober sign), local pain at the spine or tenderness of the paravertebral muscles (assessed by palpation of the paravertebral muscles and percussion of the spine), abnormal findings in the limbs (limited or disturbed mobility, pain, swelling, deformation, or amputation) and skin abnormalities (eczema or inflammation, itching, or dyshidrosis).

Also corresponding headlines in tables 2 and 3 (page 688) should read age adjusted hearing loss >30 dB (not hearing loss at 2, 3, and 4 kHz >105 dB) and diastolic blood pressure ⩾95 mm Hg (not diastolic blood pressure >95 mm Hg).

The authors deeply regret these errors, which do not alter the conclusions of the paper. We kindly ask the reader to comply with these corrections when interpreting the data presented in tables 2 and 3.

BOOK REVIEWS

Book review editor: R L Maynard

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Inhaled Particles VIII. Edited by: N CHERRY, T OGDEN. (Pp 744; Price $249.00, Dfl 403.00) 1997. Oxford: Pergamon, Elsevier. ISBN: 0080427405.

As indicated by the title, this book is the eighth in a series of proceedings of symposia on inhaled particles. These international symposia, held in 1960, 1965, 1970,1975, 1980, 1985,1991, and 1996 at various venues in the United Kingdom, were all sponsored by the British Occupational Hygiene Society (BOHS). Since the 1975 meeting, the proceedings were also issued simultaneously as special issues of the BOHS journal *Annals of Occupational Hygiene*. These symposia and their proceedings have served as landmarks documenting the state of the art in knowledge and techniques concerning human exposures to airborne particles, their deposition and clearance within the respiratory tract, and their health effects.

The broadening depth and scope of the symposia to now include radioactive, outdoor and indoor particulate matter have put a great strain on the organisers and especially on the editors. Additional pressures resulted from the fact that the two previous proceedings did not appear until three years after the meetings. Thus, the publication of this volume only one year after the symposium is a significant accomplishment and a tribute to the dedication of its new editors. A price for this accomplishment was the restriction of the 127 papers to four to six pages each, and the elimination of external peer reviews. The reviews and editing were done by the editors alone. Also, for the first time, there were no abstracts included in the papers and the questions from the audience, and authors' responses, that were notable features of the earlier volumes, were not included.

The quality of the papers and editing remain at a high level, and this volume should be a valuable addition to the bookshelf of all scientists, health professionals, and public health authorities who are seriously interested in the health effects of airborne particles. It is unfortunate that the price is high enough to limit its readership.

MORTON LIPPMANN

Determination of Airborne Fibre Number Concentrations: A Recommended Method by Phase-Contrast Optical Microscopy (Membrane Filter Method). By: World Health Organisation. (Pp 54; Price Swiss Franks 19/ US $17.10) 1997. Geneva: World Health Organisation. ISBN: 92-4-154496-1.

Airborne fibre concentrations have been evaluated by the membrane filter method for over 30 years. The original method, which was developed for use in asbestos factories, has been adapted for use with many different types of fibre in both occupational and non-occupational situations. By the 1970s it was clear that there were substantial differences between laboratories when evaluating the same samples. Much of these differences could be attributed to variations in the detailed methodology and this started the quest to devise a "standardised" version of the membrane filter method. This book is the latest attempt to standardise the method on this occasion to harmonise methods used for analysis of asbestos—for example, the European Reference Method (ERM) used throughout the European Union, with the previously published World Health Organisation method for man made mineral fibres (rockwool and glasswool). The new method is applicable to all fibre types, both organic and inorganic.

The method specification is contained within 17 statements which are supplemented by more detailed descriptions. It contains details of everything required from the type of filters to be used to the characteristics of the fibres to be counted. The accuracy, precision, and lower limit of measurement are also provided.

This is a specialist book which will be of limited interest to those not directly involved with measurement of exposure to fibres. However, it is clearly the authors' intentions to influence the appropriate national authorities to incorporate this version of the membrane filter method into their legislation. If this method does replace the ERM then measured fibre concentrations would probably increase, for some industries by perhaps as much as 50%. The implications for epidemiological studies, risk assessment, and standards setting need to be carefully considered.

JOHN W CHERRIE

Principles of Health and Safety At Work. By: A ST JOHN HOLT. (Pp 268; Price £33.95) 1997. Wigston, Leicestershire LE181NN: Institution for Occupational Safety and Health. ISBN: 0 901 357 21 9.

This book is aimed at managers and others who wish to obtain an understanding of the principles of occupational safety and health in the United Kingdom. It is published by the Institution of Occupational Safety and Health (IOSH) and it is the recommend text for their safety appreciation course, *Managing Safely*.

The text is divided into four sections: safety technology, occupational health and hygiene, safety management techniques, and law. There are 68 short chapters which cover the essential factual information required by someone responsible for managing health and safety. Each chapter includes several self assessment questions and a bullet point sum-

mary of the key points which could be used for revision. The scope of the book is enormous and includes machinery guarding, chemical safety, fires, electrical hazards, manual handling, and accident investigation. There are over 100 pages out of about 270 devoted to summarising the main pieces of health and safety legislation. This edition of the book has been revised to include new legislation, especially that referring to construction health and safety.

The book is clearly designed to support taught courses covering the basics of health and safety. Used in this way I am sure that it would be a valuable resource, with the section dealing with the law being particularly helpful. However, it is probably too succinct to be used as a textbook in other situations. There are extensive commendations for further reading, although these are almost exclusively publications from the United Kingdom Health and Safety Executive or British Standards.

The index is particularly poor and seems to have been naively compiled with some computer software. Many entries are unhelpful—for example, 45 entries referring to employees—or irrelevant—for example, an entry for yawning in respect of someone who has received an electric shock.

Overall, this book is a useful resource for managers and others in the United Kingdom who plan to attend a basic course in health and safety. The sections on legislation give a good overview of the relevant acts and regulations, although they are probably insufficient to act as a reference for those trying to comply with the law.

JOHN W CHERRIE

Fibrous Materials in the Environment. By: MRC Institute for Environment and Health. (Pp 108; Price £30) 1997. Leicester: Institute for Environment and Health, University of Leicester. ISBN: 1 899110 17 8.

Although the harmfulness to health of asbestos was originally described at the end of the 19th century, it was only in the 1960s after the publication of the seminal paper on mesothelioma in South African crocidolite workers by Wagner et al that general attention was drawn to these problems. The possibility that hazards from asbestos might extend to the general population rather than simply to workers in the asbestos industry gradually gained currency through the 1970s until in the 1980s the educated and reading public were being informed by their newspapers that inhalation of as little as one fibre of the mineral could prove fatal. The debate on the risks associated with exposure to asbestos became extremely polarised in the United States, resulting in widespread and inappropriate action to remove the asbestos from buildings where it was found, thus putting at risk a large and relatively unprotected workforce of asbestos removal men.

As asbestos has proved to be an exceptionally useful material, industry naturally has required substitutes and various other fibrous minerals have been produced, increasingly since the 1930s. Of these the most common are used in insulation as rockwool and glasswool. As the properties that make asbestos dangerous, namely fibre size and resistance to degradation are also those that make it industrially useful, concern has been raised as to whether such materials might imply similar risks to health.

This book, one of a series of reviews produced by the Institute for Environment and Health, provides a useful summary of the current understanding of the risks associated with both asbestos and more importantly, and less well-known, man-made mineral fibres. It provides useful background information on the many types of fibre produced and used in industry and documents comprehensively the amount and types of fibre to be found in materials and in buildings in the United Kingdom. After summarising the difficulties of measuring tiny respirable fibres, it summarises the scientific literature on fibre concentrations to be found in the general and domestic environment and makes estimates of the exposures that members of the United Kingdom population might expect over a lifetime. In parenthesis the sort of figures provided show nicely how protagonists in the polarised fibre debate can use figures to strengthen their case. For example, our background exposures to fibres in the environment average between 0.000001 and 0.0001 fibres per ml, a figure that might not unreasonably be looked upon as reassuring. However, calculating up a total lifetime exposure over 70 years can give a figure as high as almost 30 million fibres in total which to the unsophisticated sounds rather a lot. Those, however, who know something of the lungs' anatomy and physiology can take comfort from the fact that we have some 300 million alveoli for these fibres to be shared out among even assuming that most of them are deposited (which they are not!).

The book summarises the known health effects of asbestos and the, as yet, incomplete but reassuring literature on the epidemiology of workers exposed to other fibres. It then discusses the experimental animal and in vitro evidence with respect to man-made fibres. There is useful discussion of fibre deposition, clearance, and solubility leading to conclusions which in my view are wholly sensible. For asbestos, the authors argue against a general policy of removal and for management in situ unless the material is releasing unacceptable amounts of dust. For man-made mineral fibres, they express caution about the production of fine diameter fibres but point out that almost all the material used commercially is not respirable and that there is no reason to suppose that current levels of exposure pose any risk to the public.

All in all this is a remarkably informative book containing much information on mineral fibres that is not readily available elsewhere. The debate about the harmfulness of fibres needs to shift back to the protection of exposed workers and away from theoretical risks to the general population.

ANTHONY SEATON

Tobacco Or Health: A Global Status Report. By: World Health Organisation. (Pp 495; Price Swiss Franks 155/ US $139.50) 1997. Geneva: WHO. ISBN: 92 4 156184 X.

"Every 10 seconds, another person dies as a result of tobacco use". This is the stark introductory sentence to this reference book compiled by the World Health Organisation as a source of standardised baseline information on tobacco production, trade, consumption, health effects, and control in WHO member states. The book is divided into two parts: the first, comprising 60 pages, attempts to summarise the global situation in the late 1980s and early 1990s. The second and larger part provides a series of "country profiles" for each of the member states, typically of one or two pages. These list the latest available information on demographic and general health indicators, tobacco production, trade and industry, tobacco consumption, and smoking prevalence by age and sex, and national tobacco control policies and programmes.

Designed as a reference text, this is not a book to be read from cover to cover. Its strength is the near comprehensive coverage of national statistics on tobacco production and use, which are usefully summarised in part one. These may suffice for readers with an epidemiological background, among whom the adverse health effects of smoking are taken for granted. For a more general readership, however, a notable weakn ess of this book is the paucity of information on health consequences of tobacco use. The relevant chapter in the first part runs to only five pages, including four tables, and is supported by only two references, one of which is yet to be published (although it could have been cited as a series of recent articles in the *Lancet*). The discussion of health effects is entirely focused on mortality, mainly from broad groups of causes, such as total mortality and cancer deaths. Remarkably, there is no mention of the disability and loss of productivity related to cardiovascular and respiratory diseases, nor of the consequences of environmental tobacco smoke. Where the health effects are assessed for individual countries in part two, figures are provided mainly for developed countries and relate principally to estimates of tobacco related deaths and trends in lung cancer mortality.

This volume provides a powerful reminder, if such is needed, that tobacco use is a global phenomenon, with one third of adults now smoking, and two thirds of these residing in developing countries. The premise underlying the report is that widespread tobacco consumption and public health are mutually incompatible, but readers seeking a comprehensive collation and consideration of the epidemiological evidence linking smoking and ill health will be disappointed. It is apparent that the WHO intends these data to be a baseline for a global programme of surveillance of smoking habits and tobacco control. Evidently much more work is needed to compile a similarly comprehensive account of the effects of tobacco use on health in many countries, particularly in the developing world. A valuable addition to future editions would be evidence from countries with well developed tobacco control policies of the extent to which lowering smoking prevalence reduces death and disability. This might encourage much needed policy initiatives in many other countries where tobacco control has yet to achieve prominence on the public health agenda.

DAVID P STRACHAN

Cancer in the Offspring of Radiation Workers: a Record Linkage Study. By: G J DRAPER *et al* (Pp 72; Price £10) 1997. Chilton, Didcot, Oxford: National Radiological Protection Board. ISBN: O 85951 412 9.

This monograph gives full details of a study conducted to test the Gardner hypothesis—namely, that childhood leukaemia and non-Hodgkin's lymphoma result from the father's

exposures to ionising irradiation before conception. This study has also been published as a paper in the *BMJ*,[1] but this volume goes into far greater details than is available elsewhere. This is very much a book for the concerned specialist reader who wants the technical background to the *BMJ* article.

This study is essentially a record linkage exercise. The exposed fathers (and mothers) were defined as having records with the National Registry for Radiation Workers (NRRW) held by the NRPB. This is a database of over 120 000 people and it was linked with the national register of childhood tumours, a database of over 50 000 children with all types of cancers. Two other data sources on childhood cancers were also included.

For the three sources of data on childhood tumours, controls were found in various ways to ascertain if these children had a father in the NRRW. The parental estimated doses were created from the NRRW. In all a total of 200 fathers and mothers were linked to children with cancer. Eighty two children with leukaemia or lymphoma were linked to fathers' records at the NRRW, as were 79 control fathers. The corresponding numbers for mothers were 15 and three.

The cases in the original Gardner paper were excluded and the results for fathers showed that case fathers had a 1.77 significant excess risk over control fathers for having a child with leukaemia or lymphoma. However, the risk was associated with the lowest dosages and there were no dose responses in any of the comparisons. In this sense the Gardner hypothesis is refuted!

Furthermore, the risk in mothers was also significantly and greater in magnitude than the fathers. However, the small numbers make this result unreliable and difficult to use to extrapolate risk.

The explanation of the association found in these NRRW members exposed to low doses is not known. It could be chance, it could also be due to misuse of film badges by those in high risk industries. This explanation is unlikely in that the cancers were distributed widely across industries in the United Kingdom and were not confined, by any means, to the nuclear reprocessing or related industries. It may be due to other exposures associated with the wider radiation industries where many other hazardous substances exist as well as ionising irradiation. Finally, it could be some other, more subtle aspect, of wearing a film badge. The authors speculate that this might be associated with the mobility of the parents, thereby linking these results with the Kinlen hypothesis which is based on ideas of infectivity associated with population mixing. They do not produce evidence to suggest that film badge wearers are more mobile than other professions but the differences in behaviour may be more complex.

Further light might be shed on this association when the nuclear industry family study (NIFS) is analysed shortly. This study will answer some criticisms of the present study. For example, it is known that there are differences in behaviour of people within the nuclear industry and those outside it. The NIFS uses internal comparisons and so such differences can be accounted for.

Despite the lack of any explanation of this observation attention is now bound to be focused on other preconceptional and periconceptual exposures in both sexes and their possible links with childhood malignancies.

R A CARTWRIGHT

1 Draper G, Little MP, Sorahan T, *et al*. Cancer in the offspring of radiation workers: a record linkage study. *BMJ* 1997;**315**:1181–8.

Cancer risk in the rubber industry: a review of recent epidemiological evidence.

J K Straughan

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