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Pulmonary asbestos and dust content in East Anglia

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ABSTRACT Measurements were made of the asbestos fibre and dust content of samples from 96 surgically excised lungs; 42 necropsies on patients with lung cancer, 11 necropsies on patients with non-pulmonary malignancies, and 59 necropsies on patients without any malignant disease. The patients' ages ranged from 45 to 74 years at the time of study. None of the patients had asbestosis. The distribution of fibres and dust content of the lungs showed a log-normal distribution. There was no significant difference in fibre counts or dust content between men and women, and between lung cancer and non-cancer patients. The only group with an association with a high asbestos fibre count was four necropsy cases of pleural mesothelioma. There was no significant relationship between asbestos fibre count and dust content of the lung. The present data suggest that asbestos fibre counts below 100 000 per gram of dried lung are not related to specific asbestos disease, although in the surgical cases who were closely questioned on their residential and occupational histories most of those with fibre counts above 30 000 per gram dried lung had had occasions of definite or very likely asbestos exposure.

The rare studies of the dust and asbestos fibre content of the normal English lungs have shown from 2·5 to 7·5 g of dust in the right lung[1] or 0·7 to 14·8 g per 100 g of dried lung,[2 3] and 85% of an urban population had asbestos fibre counts below 50 000 per g dried lung.[4]

In a study of seven Pittsburg residents, Gross and colleagues[5] found 0·6 to 2·7 g dust per 100 g dried lung and 300 to 3900 ferruginous bodies per g dried lung. Not all the ferruginous bodies however were asbestos bodies.

One hundred surgically resected lungs and 112 necropsy lungs drawn from Cambridgeshire and parts of adjacent counties were studied to try and assess the extent of exposure to asbestos.

Methods

The surgical specimens were selected as having some normal lung but otherwise were consecutive. Four of these 100 cases were 24 to 36 years old with 1600 to 4900 asbestos fibres and 12 to 106 mg dust per g dried lung. The other 96 were aged 45 to 74 years, as were all the 112 necropsy cases. These four youngest surgical cases have been excluded from the analyses of the rest of this paper because there were no matching necropsy cases.

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All but four of the 96 surgical cases had bronchogenic carcinoma. These four had emphysema, pulmonary embolism, a vascular hamartoma, and a pseudolymphoma. Fifty nine of the 112 necropsy cases had no malignant disease. The 53 patients with malignant disease comprised 42 with lung cancer, four with mesotheliomas, and seven with non-pulmonary cancers.

The technique was based on that of Whitwell *et al*[4] and all results are expressed per gram of dried normal lung. All the lungs were inflated with 10% formol saline prepared from usual reagent grade materials and tap water and stored in a large polypropylene container. After fixation two adjacent portions of the most dependent normal peripheral lung about 8 cubic centimetres each in size are taken and allowed to drain off their excess formol saline by standing two minutes on absorbent paper towelling before being put into plastic 30 ml conical based containers. The wet weight of each piece was recorded. One piece was dried for three days at 60°C and then reweighed. The other piece was digested in 10 ml 40% KOH at 60°C. This digestate was then diluted with 20 ml particle free fluid (Isoton Coulter Electronics) and centrifuged at 3600 rpm for 25 minutes. The supernatant fluid was removed and the black deposit was resuspended in a measured volume of Isoton (between 2 and 5 ml) to give a countable density of particles in the four Fuchs Rosenthal chambers which were used for

counting coated and uncoated straight or curved asbestos fibres using phase contrast microscopy to show up the uncoated fibres. Only asbestos fibres longer than 8 μm were counted. After the filling of the chambers the suspension was recentrifuged, the supernatant was removed, and the black dust deposit was dried at 60°C and then weighed. It was assumed that the removal of about 10 mm^3 of suspended dust was compensated by the increased weight from residual salts added in the formol saline, KOH and Isoton.

All the surgical patients who had lung samples estimated were interviewed before discharge from hospital when a detailed residential occupational and smoking history was taken. No such detailed information was available in the necropsy cases in whom usually only the occupation at the time of hospital admission was recorded.

Results

After an initial period of cross checking the counts, the surgical and necropsy material were analysed independently. Despite the standardisation of methods there were appreciable differences between the two groups. Four out of the 100 surgical cases (PGIS) had no detectable asbestos fibres; in contrast 35 of the 112 necropsy cases (PP) had no detectable fibres, 13 (31%) in those with lung cancer and 22 (37%) in those without lung cancer. The lowest level of detection achieved was 800 asbestos fibres per gram of dried lung and there were only two other counts of 1000 or lower.

The probability that there was an undercounting of uncoated asbestos fibres in the necropsy series is also suggested by the higher ratio of coated to non-coated fibres in the necropsy cases (1·01 mean ratio) than in the surgical cases (0·46 mean ratio). There was no significant difference between the ratio in necropsy cancer (0·83) and non-cancer cases (1·16). Throughout the rest of this paper fibre counts are the sum of coated and uncoated asbestos fibres.

Direct comparison between the two series was possible in six of the surgical patients who subsequently came to necropsy. The asbestos fibre counts and dust content of the surgical and necropsy specimens are shown in table 1. There is reasonable agreement between the two estimates for fibres and less so for dust content.

Table 1 *Comparison of fibre count and dust content in the same patient at surgical resection and necropsy*

Case number	Fibre count/g dry lung		Dust mg/g dry lung	
	Surgical	Necropsy	Surgical	Necropsy
14/98	1300	nil	50	13
61/109	2200	nil	159	11
38/94	5600	10900	27	29
78/7	20100	11900	83	201
55/19	21200	24800	78	4
64/18	23300	14200	146	44

Table 2 *Triplicate fibre counts on three adjacent pieces of one lung*

Sample A	30 700	24 600	28 600
Sample B	28 800	26 600	24 400
Sample C	31 600	29 400	31 600

Average count 28 500, SD 2800.

Table 3 *Duplicate samples*

Case number		Fibre count/g dry lung		Dust mg/g dry lung	
		Count 1	Count 2	Count 1	Count 2
Surgical	12	3300	6500	57	90
	46	1700	1400	125	186
	49	50000	88500	17	52
	69	3900	9000	145	79
	78	22100	22600	83	71
Necropsy	79	nil	1800	—	80
	103	nil	nil	46	46
	106	nil	1900	40	14
	111	nil	nil	41	13

In one lung three adjacent pieces were estimated in triplicate for fibre count. These are shown in table 2. Agreement was good between samples. Duplicate samples were assayed for asbestos fibre and dust content in five surgical and four necropsy cases and they (table 3) show the same level of agreement between samples as between surgical and necropsy specimens seen in table 1.

In five necropsy cases selected for having a reasonable number of asbestos fibres, fibres were counted in five different segments. It will be seen from table 4 that there is up to an eight-fold difference

Table 4 *Fibre counts/g dry lung in five sites*

Site	Necropsy case number				
	19	20	90	110	113
Upper lobe apex	46100	974600	21800	437900	22500
Lower anterior upper lobe	42900	2300700	22500	49500	36700
Lower lobe apex	13300	1725500	25100	269600	12300
Anterior basal	17500	2492400	24400	138700	8900
Posterior basal	10000	1093100	21900	210300	14800
Mean	26000	1717300	23100	221200	19000
SD	15300	613600	1400	130900	9900
Initial single sample	24800	1780700	22500	499800	43500

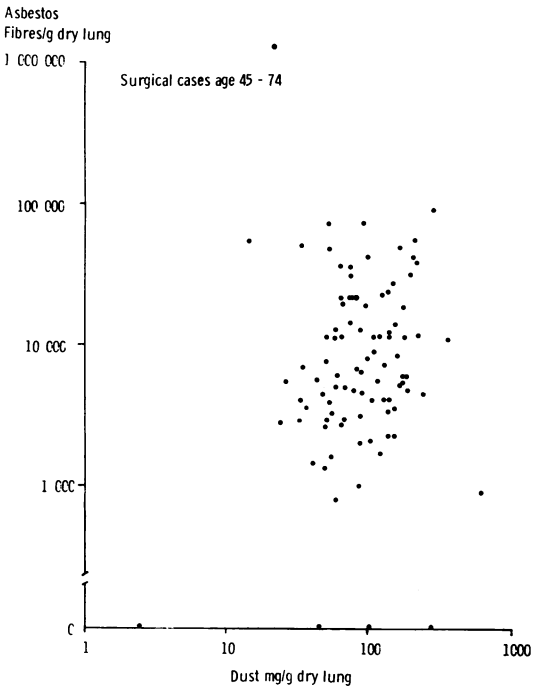


Fig 1 *Asbestos fibre counts plotted against dust content of surgical lungs.*

between the segment with the lowest and highest counts. In only one of the five cases was there uniformity of distribution of fibres between the segments. There was no part of the lung that was consistently rich or consistently poor in fibres. As a working rule in selecting material for KOH digestion throughout this study the lowest part of the lobe or lung with normal parenchyma was chosen. None of the lungs studied showed any interstitial fibrosis of asbestosis.

Although there were differences between the surgical and necropsy cases with fewer uncoated fibres and more cases with undetectable fibres in the necropsy series than in the surgical, it cannot be entirely explained by observer variability because in the cases in which direct comparison was possible the variation in counts and dust content were no greater than the variation recorded by each observer for the duplicate specimen analyses (table 3).

In the surgical as in the necropsy cases there was no increase in asbestos fibre or dust content in the lungs with increasing age. Figures 1 and 2 also show that there is no correlation between asbestos fibre and dust content of the lungs. These observations indicate that there is unlikely to be any general atmospheric pollution with asbestos in East Anglia.

There were 14 women in the surgical series, 13 of whom had lung cancer and one a pseudolymphoma. In the necropsy series 25 were women, of whom nine had primary lung cancer, one had a mammary carcinoma and 15 had no malignant disease. Figures 3 and 4 show that there was no significant difference in fibre counts or in dust count between males and females, even though in the surgical carcinoma and the necropsy non-carcinoma subsets the upper end of the male fibre counts were higher than for the females, and the male necropsy non-carcinoma subset had a low median asbestos fibre count.

There were no significant differences (figs 3 and 4) in the range of fibre counts or of dust content between the lung cancer and the non-cancer cases. However, the male necropsy non-cancer group had the highest proportion (50%) of undetectable fibre counts compared to 39% for the male necropsy cancer and 5% for the male surgical lung cancer cases.

Among the necropsy cases there were 11 patients with neoplasms other than carcinoma of the lung. Four of these 11 had pleural mesothelioma; the remainder were single cases of carcinoma of the pyriform fossa, oesophagus, pancreas, kidney,

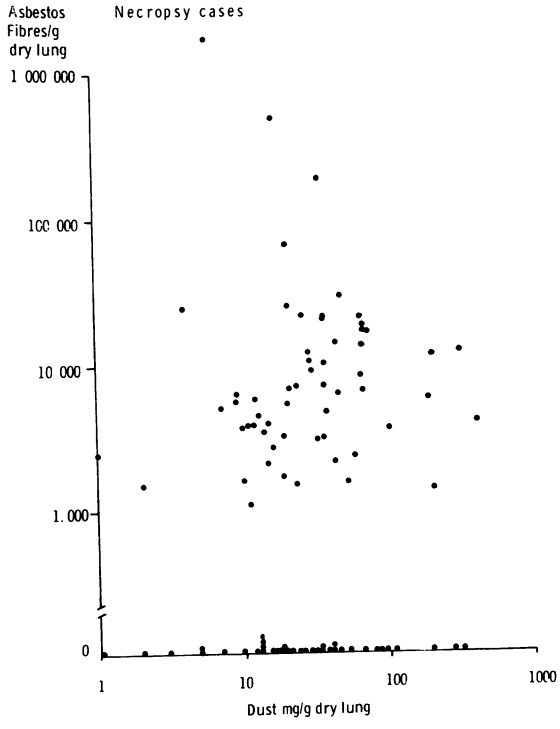


Fig 2 *Asbestos fibre counts plotted against dust content of necropsy lungs.*

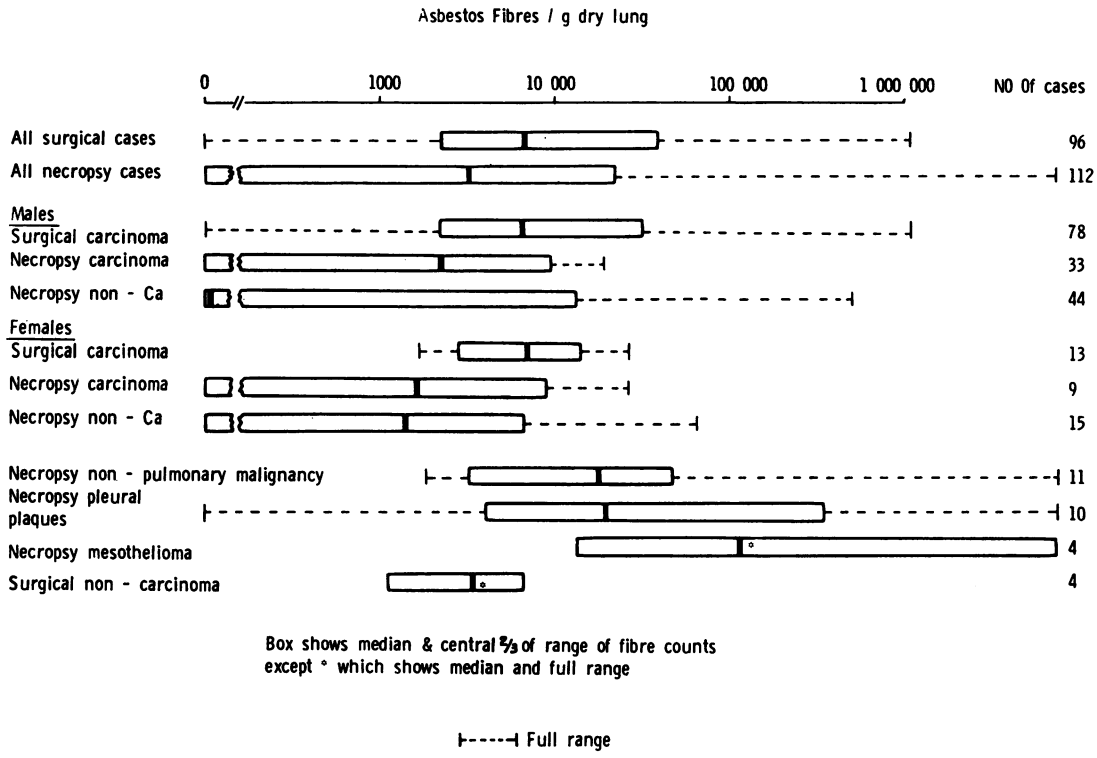


Fig 3 *Asbestos fibre counts by type of patient.*

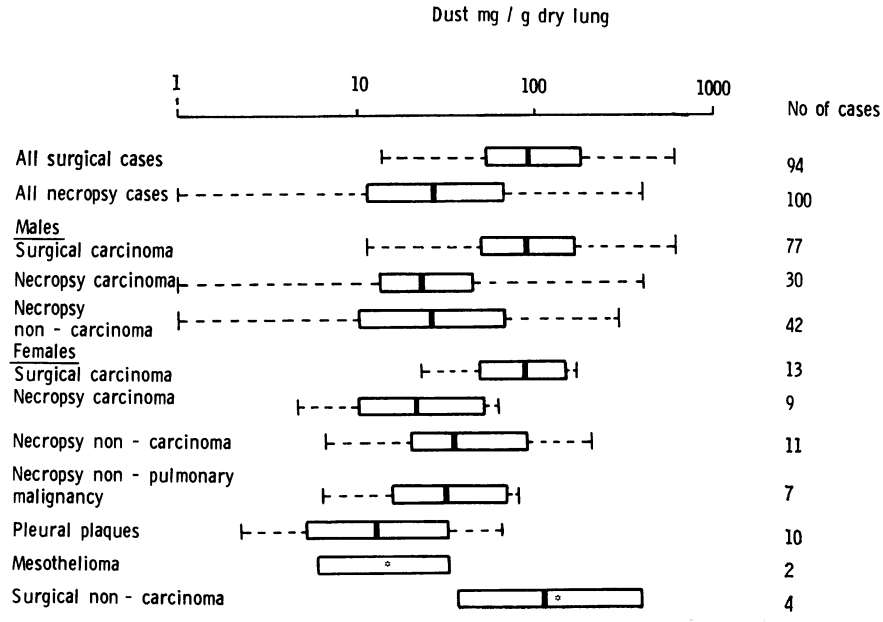


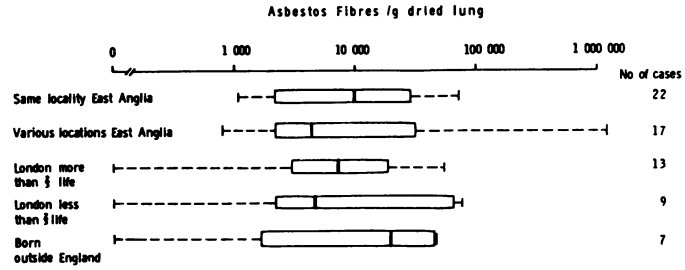
Fig 4 *Dust content by type of patient.*

Table 5 *Occupations of surgical cases with highest fibre counts*

Fibre count	*Occupations*
1 236 400	Boiler lining-brick layer, railway wagon repairs
85 100	Electrician mainly on boiler installation, gas works
72 700	Bakerlite and Diethyl plastics moulding, welding firm clerk
55 700	Railway shunter, metal lathe worker
55 600	Building demolition, carpenter, metal grinder
50 800	Boiler lagger, stoker, brick moulder
49 700	Building labourer, farm labourer
46 700	Building labourer, flour mill
41 800	Building labourer, farm labourer
40 700	Railway shunter, foundry mould maker, welder
38 200	Stone mason, foundry worker, asbestos roofing
36 400	Electronics research—soldering and glues
34 800	Grocer, car maintenance
31 200	Demolition, driver
30 900	Plastic moulding, firework maker, tyre retreads, welder
27 100	Vegetable sorter, bar maid

bladder and breast, and astrocytoma. The four mesotheliomas had asbestos fibre counts ranging from 14 400 to 1 780 700 per g dried lung with a median count of 116 200. The fibre count for this group is significantly higher than in any other group of cases. The dust content of the mesothelioma lungs is not different from any of the other groups (fig 4). The occupations of these four mesothelioma cases were given as police sergeant, retired bricklayer, retired machine operator, and maintenance engineer.

Nine male and one female necropsy patients had hyaline fibrotic pleural plaques. Four of these cases had lung cancer and three had mesothelioma. The asbestos fibre counts and dust content are shown in figs 3 and 4.



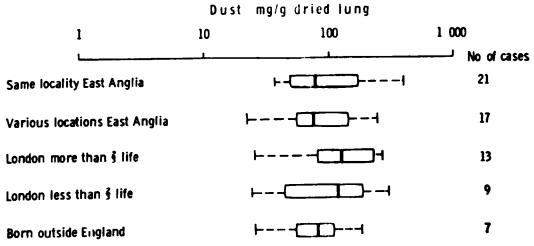


Fig 6 *Dust content by residence of patient.*

Detailed occupational histories were taken in the surgical group. The 16 cases with the highest asbestos fibre counts are shown in table 5. The most frequent occupation in this group was building labourer with demolition work forming a substantial part of the job. Builders with only construction experience had asbestos fibre counts in the central two-thirds of the total surgical group. The two cases who had worked with plastic moulding had fibres indistinguishable from asbestos but had not knowingly handled any asbestos additives to the plastics.

The highest dust content was in the lung of a clerk who was an active do-it-yourself builder and car maintainer.

In the surgical cases 22 had lived all their lives in the same town or village apart from war service. A further 17 had moved but remained in East Anglia. Nine had been born in London but moved to East Anglia early in life while 13 had spent most of their lives in London before moving to Cambridgeshire. Seven cases were born outside England. The remaining cases fell into no specific pattern. The asbestos fibre and dust content of the lungs in these residential groups are shown in figs 5 and 6 and there is no difference between them.

All but three of the surgical cases were smokers. The smokers averaged 57 pack-years (±SD 40) and there was no significant difference in the lung dust content between the light (25 or less pack-years) and the heavy smokers (more than 98 pack-years). Unfortunately the smoking and occupational histories were incomplete for the necropsy patients, so no meaningful analysis was possible.

Fig 5 *Asbestos fibre count by residence of patient.*

There were only two types of carcinoma present in reasonable numbers for analysis. Fifty-eight of the surgical cases and 15 of the necropsy cases were squamous cell carcinoma and adenocarcinoma was present in 20 of the surgical cases and 17 of the necropsy cases. The asbestos fibre counts and dust content of these cases were not statistically significantly different (figs 7 and 8). Seven per cent of the surgical squamous cell carcinomas were women compared with 14% in the necropsy series. Among the adenocarcinomas 20% of surgical and 31% of the necropsy cases were women.

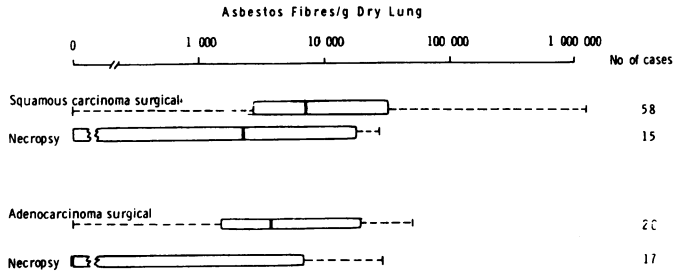


Fig 7 *Asbestos fibre count by type of carcinoma.*

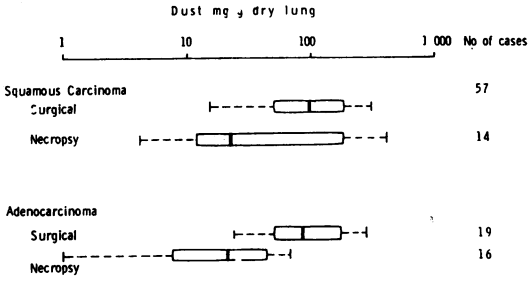


Fig 8 *Dust content by type of carcinoma.*

Discussion

This study shows that neither asbestos fibre counts or dust content show a normal distribution. The distribution is more satisfactorily examined on a log normal distribution. Tipton and Cook[6] and Sweet *et al*[7] regarded the median count as a more reliable estimate of the central tendency than the mean. Accordingly the figures show our data on a log scale with the median, the central 66%, and the total range for each of the items analysed.

The figures for the dust content of the lungs in the present study were comparable to those given for English coal[1 2] and haematite[3] miners without silicosis and for 100 Cincinnati hospital patients and coal miners.[7] These latter authors[7] found no significant effect of age or sex on the total dust content of the lungs, and we also observed no significant relationship of total dust content to sex or age in our East Anglian patients.

The effects of age and sex on the asbestos fibre count in the lung are not fully established and may depend in part on the method of assay. Most authors[8–10] found no effect of age, but Cauna *et al*[11] observed a decreasing incidence of asbestos bodies with age, while there are a few who suggest a peak age incidence in the 70s[12] or 40s.[13] Our own observation is that there is no significant effect of age on the asbestos fibre count. This would seem to suggest that there is no general background exposure but that the opportunities for occasional exposure may be increasing so that the younger age group have the same counts as the older age group.

The prevalence of asbestos bodies has been described as lower in women than in men[9–12] although Utidjian *et al*[14] found no difference. These differences may be in part related to the level of asbestos fibre count in the lung in that Rosen *et al*[9] reported an equal incidence in men and women at counts of fewer than 60 ferruginous bodies per 5 g of lung, and Whitwell *et al*[4] found twice the frequency of men than of women with asbestos fibre counts greater than 20 000 per gram. In the present series the male:female ratio in the cases above and below asbestos fibre counts of 20 000 shows an inconstant pattern. Among the surgical cases the ratio changes from 5:1 below 20 000 fibres per gram to 10:1 above this count. In the necropsy lung cancer group the ratios are 4:1 and 2:1, while in the necropsy non-malignant group the ratios are 2:1 and 4:1 respectively.

It is difficult to separate occupational from non-occupational exposure to asbestos. Even with detailed questioning the patient does not always remember exposure or never knew the nature of what he or she was working with. Whitwell *et al*[4] deduced that asbestos fibre counts above 20 000 per gram dried lung was suggestive of occupational exposure and that 50 000 or more was virtually certainly so. Warnock and Churg[15] reached a similar conclusion using 2200 ferruginous bodies per g of wet lung as the dividing level between occupational and non-occupational exposure. To assess the comparability of these two groups of authors'[4 15] counting methods we noted that the wet weight of our samples was on average 10·125 times the dry weight (SD ± 3·49 times) and that the ratio of coated to uncoated fibres were on average 1·01 and 0·46 depending on whether the series is necropsy or surgical material. Thus if our material is applicable to Whitwell *et al*[4] and Warnock and Churg[15] it suggests that the latter's level of 2200 ferruginous bodies per gram wet weight is comparable to Whitwell *et al*'s 50 000 asbestos fibres per gram dry weight. Our own cases suggest that industrial exposure results in asbestos

fibre counts over 30 000 per gram dry weight.

The similarity in these figures between different observers is remarkable in view of the differences between the two observers of this paper (table 1) and the ten-fold difference between four observers counting 30 μm sections reported by Oldham[16] and in view of our finding as much as a nine-fold difference in asbestos fibre counts between different areas of the same lung. It is also interesting that Ashcroft and Heppleston[17] found a mean ratio of coated to uncoated fibres of 0·43 in their necropsy series which is very similar to our surgical series.

There appears to be no area in East Anglia for high asbestos or dust exposure and it is of interest that the asbestos fibre counts and dust content are no better or worse for those that have lived all their life in East Anglia compared with those that have lived most of their lives in London.

The distribution of asbestos fibres in the lung is not properly understood. In three of 14 cases Rosen *et al*[9] found no asbestos bodies in one of the lobes examined and in two of these three it was the left lower lobe that had no bodies in it. Dick and Naylor[18] observed that if only one lobe had been examined in their cases then evidence of asbestos exposure would have been found in more cases if the upper lobe than the lower lobe had been sampled. The cases we analysed showed no consistent lobar or segmental preference but this may be that a selective distribution can only be seen at low levels of exposure and our five cases in table 4 were selected for high counts to improve the accuracy of counting.

Although our necropsy non-cancer group had more cases with no detectable asbestos fibres than in the necropsy or surgical cancer groups, the median and central two-thirds range of the asbestos fibre counts were not significantly different in any of the three groups. Within the exposure range observed in our cases we must argue that we have found no evidence that levels of asbestos fibres at a median count of 11 000 is related to the presence of lung cancer. This is in line with the observations of other authors.[4 9 10 12 13] Though Warnock and Churg[15] have argued otherwise, their evidence is questioned because of the different proportion of women in their control and test groups.

The median asbestos fibre count of 12 000 per gram dried weight in cases with hyaline fibrotic pleural plaques is not different from the median counts in our carcinoma and non-carcinoma cases. However, this is not the case with our four cases of mesothelioma who had a median count of 116 200 asbestos fibres per gram. Although this level of count is not as high as the median count of 750 000 asbestos fibres per gram for 100 mesotheliomas found by Whitwell and colleagues,[4] it is well above the minimal count for industrial exposure.

In the study on hyaline pleural plaques[19] one-third of cases had relatively few asbestos bodies in their lung and no evidence of occupational asbestos exposure. Using a less sensitive method 14% of patients with hyaline plaques had demonstrable asbestos bodies in their lungs and the plaques were present in 33% of randomised hospital necropsies.[20] Only one of our patients with pleural plaques had no asbestos fibres; this case and two others had no pulmonary or pleural malignancy. Our view is that plaques give a good indication of asbestos exposure but no indication of the severity of this exposure.

Whitwell *et al*[21] suggested that there might be an increased incidence of adenocarcinoma of the lung in asbestosis. Warnock and Churg[15] observed an increased proportion of adenocarcinomas in their cases with the higher number of ferruginous bodies. At the level of asbestos fibre counts in our cases we found no difference in median counts between the squamous and adenocarcinoma cases.

Our tentative conclusions are that in the general population of East Anglia non-occupational exposure to asbestos is not occurring as a continuing background experience but probably represents occasional short episode(s) of exposure, and these episodes result in fibre counts up to about 30 000 per g dried lung. Above this level exposure is likely to be occupationally related and above about 100 000 there is likely to be asbestos related lung disease. The non-asbestos-fibre dust was made up mainly of fine black carbon particles and as with the asbestos fibres there was no evidence of a continuing background experience nor could we relate it to cigarette consumption.

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