#### Summary

A method is described which uses non-radioactive sodium chromate enriched with chromium-50 ( $^{50}$ Cr) for the measurement of the red cell survival time. The chromium content of serial blood samples was measured by activation analysis and its rate of fall was used to calculate the  $T_{\frac{1}{2}}^{50}$ Cr. There was good agreement between the  $T_{\frac{1}{2}}^{50}$ Cr and the  $T_{\frac{1}{2}}^{51}$ Cr, measured simultaneously. The method would allow simultaneous comparison of the survival times of two different populations of red cells, using two erythrocyte labels which behaved identically. Other advantages and some disadvantages of the technique are discussed.

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## Nasal Cancer in Woodworkers in the Furniture Industry\*

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The purpose of this paper is to confirm and amplify preliminary reports which suggested that in the Buckinghamshire and Oxfordshire furniture industry there was an excessive risk of adenocarcinoma of the nasal cavity and sinuses among woodworkers (Macbeth, 1965; Acheson, Hadfield, and Macbeth, 1967).

#### Material and Method

#### Means of Ascertainment

The material consists principally of two groups of cases of carcinoma of the nasal cavity and accessory sinuses occurring in persons resident in Oxford County Borough, the Administrative County of Oxfordshire, and those parts of Buckinghamshire and Berkshire within the Oxford Hospital Region (Fig. 1). Group 1 consists of patients in whom the diagnosis was made during the decade 1956-65. As the ascertainment is thought to be complete, and the 1961 Census was taken at the midpoint of the decade, these figures have been used in the computation of incidence rates. Group 2 consists of all other patients ascertained in the same area who were diagnosed either before 1 January 1956 or after 31 December 1965. The means of ascertainment used were different in the two groups. In addition to these two groups of cases brief mention is made of eight woodworkers with nasal adenocarcinoma ascertained from other parts of Southern England.

The source of the cases in group 1 was the Oxford Cancer Register. This register was established in 1951, and it is thought that case-finding has been almost complete since 1956. Thus it is noteworthy that none of the additional searches described in the succeeding paragraph uncovered any cases for this decade not previously known to the register.

In group 2 the cases occurring since 1965 were obtained either direct from the hospitals concerned or from the register,

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and are thought to be complete for cases diagnosed up to the end of 1967. Attempts to uncover earlier cases were made along the following lines: (1) a search was made of the death registers for High Wycombe and the surrounding countryside, and for Banbury and district back to 1900; (2) a circular letter was sent to the records officers of all London hospitals requesting information about patients treated for nasal cancer who gave an address in the area under study; and (3) a search of the diagnostic index of the Radcliffe Infirmary was made back to its beginning in 1939.

In addition a circular letter was sent to each general practitioner practising in the area, and a systematic search was made of all claims for death benefit (which include a note of the cause of death) received since 1960 by the National Union of



Fig. 1.—Map of area showing numbers of cases of adenocarcinoma ascertained in persons ever employed in the furniture industry according to place of exposure.

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Furniture Trade Operatives and the National Union of Wood Machinists. The records of the Oxford Record Linkage Study were checked from its inception in 1962.

#### Area Studied

The population of the area in April 1961 was 1,006,885. The area consists of the southern part of the Oxford Hospital Region, which includes the cities of Oxford and Reading, each of which had at that time a population of about 100,000; the towns of Banbury, High Wycombe, and Aylesbury; the upper part of the Thames valley; and the line of chalk hills known to the north as the Chilterns and south of the Thames as the Berkshire Downs (Fig. 1).

As the boundary of the area unfortunately cuts across the county boundaries of Buckinghamshire and Berkshire, and occupational tables for the urban and rural districts are not available in the 1961 Census, the exact number of woodworkers in the area is not known except within the town of High Wycombe itself. Figures for the whole counties of Oxfordshire and Berkshire and for High Wycombe and the remainder of Buckinghamshire are shown in Table I.

Table I.—Distribution of Woodworkers in Oxfordshire, Berkshire, and Buckinghamshire According to the 1961 Census. (The Occupational Analysis of this Census was Carried Out on a 10% Sample)

		Bucki	nghams	hire		ı	Tot	otal	
	General Register Office Code	High Wycombe	Remainder	Total	Oxfordshire	Berkshire	No.	%	
Carpenters and ioiners	080	160	2,500	2,660	1,610	2,790	7,060	50.6	
Cabinet and chair makers	081	890	690	1,580	240	240	2,060	14.8	
Sawyers and wood- working machinists		700	1,250	1,950	300	480	2,730	19-6	
Coopers Pattern makers Woodworkers not	083 084	30	130	160	170	110	440	3.2	
elsewhere classified	085	270	660	930	250	470	1,650	11.8	
Total		2,050	5,230	7,280	2,570	4,090	13,940	100-0	

It can be seen that of the 13,940 woodworkers in the three counties 50.6% are carpenters and joiners. In this area these men are employed principally in the building trade and, in general, work with soft woods.

The 2,060 cabinet and chair makers work in the furniture industry. Three-quarters of these are employed in the Buckinghamshire furniture industry, which is concentrated in the High Wycombe area. Traditionally this industry derives from the manufacture of chairs made from the beeches which thrive in the chalky soil of the Chilterns. Chair making was already a thriving cottage industry in this district in the second half of the eighteenth century. However, over the last century the manufacture of all types of furniture has become commonplace in the High Wycombe area, and a wide range of imported hardwoods are used. In the period before the second world war the industry still consisted of a large number of small firms, most of which had fewer than 50 employees. Thus according to Kelly's Directory for 1939, there were in that year 150 firms of chair manufacturers in the High Wycombe area employing in aggregate about 5,000 hands. At that time about one and a half million chairs were made each year. Working conditions in these small firms were relatively poor by contemporary standards, and the use of exhaust ventilation was

A furniture-making industry on a much smaller scale has existed in Oxford and Banbury for at least 50 years. This has a different tradition from the Buckinghamshire industry and has been concerned with general, office, and ecclesiastical furniture, not chair making, the principal materials being oak and mahogany. Little beech is used.

It is safe to assume that the majority of sawyers and wood machinists and the unclassified woodworkers in the High Wycombe area have been employed in the furniture industry. In the remainder of the area many of these men are concerned with preparing timber for the building and joinery trades.

It is important to note that the Buckinghamshire furniture industry employs a substantial number of men who are *not* woodworkers. In addition to clerks and labourers these include upholsterers and French-polishers. In High Wycombe in 1961 these are thought to have comprised some 3,000 men in all, but exact figures are lacking.

#### Occupational Histories

An occupational history was taken by one of us (E. D. A.) in respect of all male patients with nasal carcinoma who were living in 1967 or for whom a close relative could be traced and interviewed. These 18 occupational histories are given as an Appendix, and abstracts of those in which there was an association with the wood trade are shown in Table VIII (Cases 001-011, 024, and 031-032). In addition it was possible to reconstruct part or all of the occupational histories of a number of other woodworkers with help from the High Wycombe Furniture Manufacturers' Association and the trade unions. These are shown in Table VIII.

#### Verification of the Histological Material

An attempt was made to obtain and to review all the histological material. With the co-operation of the pathologists in various hospitals in the area material was obtained for 40 (48.2%) of the 83 cases in group 1 and for 18 (27.7%) of the 65 cases in group 2. As the cases in group 2 dated back as far as 1939 it is not surprising that the amount of material found was relatively small. All material that was found was examined by one of us (R. H. C.) without knowledge of the clinical history or occupation of the patient.

Table II shows that there is a close correlation between the two classifications, the effect of R. H. C.'s classification being slightly to increase the numbers of adenocarcinomas at the expense of the transitional and squamous groups. It was concluded that in view of the close correlation it was permissible to use in the analysis the remainder of the material for which histological material could not be obtained for review. However, where both classifications were available R. H. C.'s was preferred. The findings in this paper can be demonstrated with either classification.

TABLE II.—Reclassification of 58 Cases of Carcinoma of the Nasal Cavity and Sinuses

Previous	R. H. C.'s Classification									
Classification of Carcinoma	Adeno- carcinoma	Squamous	Transi- tional	Ana- plastic	Other	Total				
Adenocarcinoma Squamous Transitional Anaplastic Type unspecified	15 1 2 1	23*	2	- 3 - 9	     	15 27 4 11				
Total	19	23	2	12	2	58				

<sup>\*</sup> Including three transitional squamous. † Malignant salivary gland myoepithelioma. ‡ Salivary adenocarcinoma.

## Results

## Sex and Histological Type

The relation of sex and the histological type of carcinoma for the two groups is shown in Table III. When both sexes are considered together, squamous carcinomas are seen to be commonest in both groups, accounting for about half the classified tumours. The next most frequent tumour is adenocarcinoma, which accounts for about one-quarter. Anaplastic

tumours and transitional cell tumours were rare. The relatively large proportion of carcinomas in which the histological type is unknown in group 2 is due to the fact that many of these cases date from a period for which hospital and laboratory records have been destroyed.

TABLE III.—Histological Type of Tumour by Sex

Types of Carcinoma		Males	Females	Total	Sex Ratio
		Group 1			
Squamous carcinoma Adenocarcinomas Transitional cell carcinomas Anaplastic Unclassified	19 23 2 8 4	17 0 3 5 2	36 23 5 13 6	1·1 ∞ 0·7 1·6 2·0	
Tetal		56	27	83	2·1
		Group 2			
Adenocarcinomas		17 10 2 2 11	7 3 2 5 6	24 13 4 7 17	2·4 3·3 1·0 0·4 1·8
Total		42	23	65	1.8

<sup>\* 1</sup> male saliwary myoepithelioma; 1 female basal cell carcinoma ethmoid; 1 female saliwary adenocarcinoma.

The overall male/female sex ratio for the material is about 2:1. When the sex ratios for the various histological types are compared it is clear that in group 1 the sex ratio for adenocarcinoma is strikingly and significantly different from that of the other types of tumour. Thus where adenocarcinomas are compared with all others:  $\chi^2=15.77$ ; 1 D.F.; P<0.001. For group 2 the trend is in the same direction but is not significant.

The crude annual average incidence rates per million for each sex and for both sexes together were as follows: squamous carcinomas—males 3.8, females 3.4, both sexes 3.6; adenocarcinomas—males 4.6, females 0.0, both sexes 2.3; transitional cell tumours—males 0.4, females 0.6, both sexes 0.5; anaplastic tumours—males 1.6, females 1.0, both sexes 1.3; other and unclassified carcinomas—males 0.8, females 0.4, both sexes 0.6; all types of carcinoma together—males 11.2, females 5.4, both sexes together 8.2.

#### Age at Diagnosis and Histological Type

The distribution by age at diagnosis of the patients with each histological type of tumour is shown for both groups in Table IV. Adenocarcinoma tends to occur in younger people than do the other classified tumours. This difference is particularly marked in group 1, where only 1 out of 23 adenocarcinomas occurred in a patient over 65, while almost exactly half the other classified tumours occurred in people over 65 ( $\chi^2=14.52$ ; 1 D.F.; P<0.001). In group 2 cases the age difference is in the same direction but is not significant.

TABLE IV.—Histological Type of Tumour by Age, Both Sexes Together

		- 1 JPC 01 2	uniour by r	ige, bein se	res Together
Age at Adeno- Diagnosis carcinomas		Squamous Carcinomas	Other Classified Carcinomas	Unclassified Cases	Total
		G	roup 1		
-14 15-44 45-54 55-64 65-74 75+	0 3 9 10 1 0	0 3 6 10 9 8	0 1 6 1 4 6	0 0 2 2 1 1	0 7 23 23 15 15
Total	23	36	18	6	83
		G	roup 2		
-14 15-44 45-54 55-64 65-74 75 + Unksown	0 0 2 4 3 2 2	0 1 2 3 10 7	0 0 0 8 3 0	1 1 3 3 3 4 2	1 2 7 18 19 13
Total	13	24	11	17	65

## Geographical Distribution by Histological Type and Sex

This analysis may be applied only to group 1, as the ascertainment of group 2 is incomplete and probably biased from a geographical viewpoint.

The material is set out in Table V for absolute numbers and for incidence rates per million per annum. For males the striking features are the high incidence rates for adenocarcinoma in Buckinghamshire and in particular in High Wycombe. In contrast, the other types of tumour are evenly distributed. In females there is a slight suggestion of an increase in the incidence of anaplastic and transitional tumours in High Wycombe, but as this is based on only three cases (two transitional and one anaplastic) little can be made of it.

TABLE V.—Geographical Distribution of Various Types of Tumour by Sex (Group 1 Only)

		λ	lales		Females				
	Squ.	Adeno.	Anapl., Trans., and Un- classified	All	Squ.	Adeno.	Anapl., Trans., and Un- classified	All	
Absolute Numbers									
Oxfordshire Berkshire Buckinghamshire High Wycombe Remainder Whole area	5 7 7 0 7 19	19 15 4 23	5 3 6 1 5 14	14 10 32 16 16 56	4 9 4 0 4 17	0 0 0 0	2 2 6 3 3 10	6 11 10 3 7 27	
Crud	le Aver	age Ann	ual Inciden	ce Rate	es per N	fillion			
Oxfordshire Berkshire Buckinghamshire High Wycombe Remainder Whole area	3·2 3·9 4·3 — 5·0 3·8	2·6 0·0 11·6 60·1 2·9 4·6	3·2 1·7 3·6 4·0 3·6 2·8	9·0 5·5 19·5 64·1 11·5 11·2	2·6 4·9 2·4 — 2·8 3·3		1·3 1·1 3·6 12·0 2·1 2·0	3·9 6·0 6·0 12·0 4·9 5·3	

## Occupation at Diagnosis and Histological Type

When all histological types of tumour are considered together there were 42 men who were "economically active" in group 1 at the onset of their illness and whose distribution in terms of occupation is therefore comparable with the findings of the 1961 Census. Of these 42 men 16 were woodworkers of all kinds (37.2%), as opposed to 3.3% of the population of the three counties; four were construction workers; and four came from the professional and technical groups. The rest of the men were distributed among the other occupational orders in very small numbers.

Accurate incidence rates for woodworkers and non-woodworkers cannot be calculated because the precise numbers in the occupational groups in the area covered by the study are not known. However, if a proportionate reduction is made to allow for those parts of Buckinghamshire and Berkshire outside the area, the crude average annual rates for woodworkers and non-woodworkers (all histological types) for the decade were about 150 and 9 per million per annum respectively for economically active males—that is, excluding children, unemployed, and retired persons. The rate for non-woodworkers is inflated by persons who worked previously in the furniture industry but had left the industry at the time of onset of symptoms (see Table VIII).

The next point of interest is the distribution of the risk within the woodworking trades themselves. Of these men 14 were cabinet or chair makers or wood machinists in the furniture industry at time of onset of their illness, while one was a crate maker (Case 024) and one was a brewer's cooper (Case 023). No cases were reported in group 1 among the carpenters and joiners, though these make up half of the total population of woodworkers (Table I). In the area under study the risk of nasal cancer is thus associated not with woodwork in general but with the woodworking crafts within the furniture industry.

Table VI shows for groups 1 and 2 the relation between occupation at onset of illness and histological type of tumour.

In group 1 14 of the 15 woodworkers in the furniture industry had an adenocarcinoma. The relation between working in the furniture industry and this type of tumour is highly significant. If unclassified cases are excluded and woodworkers in the furniture industry are compared with all others, adenocarcinoma being compared with all other tumours: in group 1  $\chi^2 = 18.68$ , 1 D.F., P<0.001, and in group 2 the trend is in the same direction but is not significant. It becomes clear that in this area the association between adenocarcinoma and the furniture industry has been an extremely strong one.

TABLE VI.—Relation of Histological Type of Tumour in Males to Occupation and Industry at Time of Diagnosis

Type of Carcinoma	Wood- workers in Furniture Industry	Others in Furniture Industry	Other Wood- workers	Others	Un- known	Total
		Group	1			
Adenocarcinoma Squamous Other classified	14 0	1* 0	0 2	8† 15	0 2	23 19
carcinomas Unclassified cases	0	0	0 0	8 3	1 1	10 4
All types	15	1	2	34	4	56
		Group	2			
Adenocarcinoma Squamous Other classified	3	2‡ 0	2§ 0	2   11	1 5	10 17
carcinomas Unclassified cases	0	0 1	0	3 8	1 1	4 11
All types	5	3	2	24	8	42

<sup>\*</sup>Yard labourer in furniture factory. † Of these, 5 were ex-workers in furniture industry—2 cabinet makers, 2 wood machinists, and 1 French-polisher. ‡ 1 French-polisher; 1 furniture factory yard labourer. § 1 timber merchant's representative; 1 joiner. || Including 1 ex-clerk in furniture industry.

Table VIII represents an attempt to determine the relative incidence of the tumours in the different trades within the furniture industry. Only High Wycombe cases have been considered because within that town it can be assumed that almost all the wood machinists and unclassified woodworkers are in the furniture industry. In the remainder of the area some substantial but unknown proportion of men in these trades work in the building industry. As the numbers are small and no allowance can be made for differences in age structure of the various trades the results must be interpreted with caution. However, it seems clear that the risk is greater among woodworkers in the furniture industry than among the other workers—who include French-polishers, labourers, upholsterers, packers, clerks, etc. The risk in machinists is similar to that among makers of chairs, cabinets, etc. No realistic estimate of the risk in workers never employed in the furniture industry can be obtained in High Wycombe. Thus of the 15 cases of adenocarcinoma diagnosed in the town between 1956 and 1965 all are known to have worked in the furniture industry at some time (Table VII).

TABLE VII.—Average Annual Incidence of Adenocarcinoma in High Wycombe (Group 1, Males) According to Occupation at Time of Diagnosis (1956-65)

Trade or Occupational Group	Patients with Adeno- carcinoma Classified at Diagnosis	Male Adult Population at 1961 Census	Average Annual Incidence per 1,000
Cabinet makers Chair makers Veneer maker Wood machinists	${2 \atop 3 \atop 1} 6 \atop 6$	1,025 835	0·6 0·7
All woodworkers in furniture industry Other workers in furniture industry	12 1	1,860 c. 3,000†	0·7 0·03
All workers in furniture industry	13	c. 5,000†	0.3
Other workers in High Wycombe, including carpenters and joiners	2*	c. 11,500	3
All occupations	15	16,500	0.1

<sup>\*</sup> These men were plasterers, but both were ex-cabinet makers in the furniture

industry.

† Unfortunately figures which are directly comparable with those for woodworkers are not available from the 1961 Census.

It is worth noting that two of the three women who developed carcinoma of the antrum in High Wycombe in the decade 1956-65 (one anaplastic and one transitional cell tumour) had worked in the furniture industry in early life, one as a Frenchpolisher and one as a sandpaperer (Cases 022 and 025). One other French-polisher (a male) in High Wycombe presented with an adenocarcinoma in 1966 and is counted in group 2.

In all the cases of adenocarcinoma in furniture workers where it was possible to examine the original clinical notes it was found that the growth had originated from the area of the turbinates and the ethmoid sinus. The term "antrum" sometimes used loosely, and we have not been able to confirm that any of these tumours started in the maxillary antrum.

#### Risk to Woodworkers in Furniture Industry in Area

The risk of adenocarcinoma to woodworkers in the furniture industry within the area is not limited to High Wycombe. In group 1 one patient (Case 005) was a wood machinist at a factory making general household furniture in Banbury at diagnosis; and another (Case 002) was a cabinet maker in Oxford. Case 004 had worked as a wood machinist from 1936 to 1945 at the same factory as Case 005. Case 003 had also worked in the same factory from 1924 to 1929 as a Frenchpolisher.

Among group 2 patients Case 003 had worked as a clerk in a firm of ecclesiastical furniture makers in Oxford. His office was a small hut within the machine shop placed so near to the machines that work had to stop when the telephone rang. Cases 036 and 038 were a wood turner and a timber yard labourer respectively from the rural Buckinghamshire furniture industry outside High Wycombe.

Though the number of cases occurring outside High Wycombe is too small to permit the computation of incidence rates, the evidence that the risk extends to woodworkers in the furniture industry in these areas seems strong. In particular the occurrence of three cases among persons who had worked in one factory in Banbury which never employed more than 200 workers, and the occurrence of two cases in the tiny Oxford industry, suggest that the risk is of the same order of magnitude in these areas as in Buckinghamshire.

## Additional Information from Occupational Histories

Summaries of the occupational data compiled from various sources are given as Table VIII. Abstracts of those obtained at first hand are given in the Appendix. The year of entry to the industry is known for certain in 16 cases of adenocarcinoma in groups 1 and 2. In the remainder, where the date is shown in parentheses it has been assumed that the patient entered the industry on leaving school at 14 years of age. Fig. 2 shows the interval in years between entry to the industry and diagnosis in cases of adenocarcinoma in persons who at any time worked in the furniture industry. The mean latent periods for these cases, for the 16 cases with a definite date, for the remaining eight, and for all cases are 38.8, 41.9, and 39.8 years respectively. Of these, the first is presumably the best estimate.

The occupational histories taken disclosed five patients (Cases 001, 003, 004, 017, and 018) who had left the industry before the development of symptoms and in whom the dates of entry and departure were certain. In an additional patient (Case 033), though it is certain he left the industry, the exact dates are not known. These cases are important for two reasons (Fig. 2 (b)). In the first place they show that tumours may develop in persons who have been in the industry for periods as short as five (Case 018), seven (Case 017), and nine (Case 003) years, and, on the other hand, that tumours may develop as long as 34 years after the patient has left the industry. These cases are also important because they help to define the

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earliest date at which the carcinogen was present in the industrial environment. Thus, as Case 018 left the industry in 1926 and Case 017 in 1930 we can be certain the hazard existed in the 'twenties. On the other hand, from Case 010, who did not enter the industry until 1935, it is probably safe to assume that the hazard continued to be present up to the beginning of the second world war at least.

In view of these findings it is reasonable to assume that the cases of nasal adenocarcinoma reported here cannot be due to a substance introduced to the industry since the war. On this ground alone it is possible to exclude a number of exotic hardwoods, and synthetic glues, stains, and varnishes. In the occupational histories attention was therefore focused on the substances to which the patient had been exposed before the war.

TABLE VIII.—Summaries of Occupational Data Obtained in 25 Patients Associated at Some Time with the Wood Trade in Group 1; 11 Patients in Group 2; and 8 Other Patients Outside the Area

			,		11 Patients in	этоир	2; and 8 Oine	T Pan	ienis Ouis	ue ine Area		
Case No.	Sex	Year of Birth	Year of Entry to Industry	Nature of Industry	Occupation in Wood Industry	Year Left Indus- try	Town(s) in which Employed in Wood Industry	Year of Diag- nosis	Histology	Principal Types of Timber to which Exposed	Site of Tumour	Notes
						ford, Bi	ckinghamshire, and	Berksh	ire Cases 195	6-65		
001	M	1914	1928	Chair makers	Band sawyer*	1950	High Wycombe	1958	Adeno.	Unknown	Ethmoid	Vacuum chimney- sweep
002	M	1902	1917	School and educational	Cabinet maker	-	Oxford	1962	Adeno.	Oak, mahogany, Douglas fir, chestnut, Cyprus	Ethmoid	_
003	М	1910	1924	furniture General	French-polisher*	1933	Banbury†	1960	Adeno.	pine Oak, mahogany	Antrum	Since 1933 worked
004	М	1909	1925	furniture General furniture	Wood machinist*	1936	Banbury†	1964	Adeno.	Oak, mahogany	Ethmoid	in motor industry Since 1936 furnace- man in alumin-
005	м	1906	1922	General furniture	Wood machinist		Banbury†	1960	Adeno.	Oak, mahogany	Ethmoid	ium mill
006	М	1899	1920	General furniture	dimension saw Cabinet maker	_	Ipswich 1920-3; High Wy- combe 1923-63‡	1964	Adeno.	Oak, mahogany, wal- nut, beech	Mid- turbinate	Occasional snuff taker
007	M	1905	1918	General furniture	Cabinet maker	_	High Wycombe	1964	Adeno.	Mahogany, oak, beech	Ethmoid	
800	M	1902	1920	General furniture	Chair/table maker; veneer matcher	_	High Wycombe‡	1958	Adeno.	Beech, oak, mahogany	Ethmoid	
009	M	1912	1926	Chair makers	Chair maker	_	High Wycombe	1962	Adeno.	Mahogany, beech, oak, agba	Ethmoid	Occasional snuff
010	М	1921	1935	General furniture	Wood machinist	_	High Wycombe	1963	Adeno.	Almost entirely beech, a little oak, and African cherry	Ethmoid	
011	М	1900	1920	Chair makers	Wood machinist, chiefly fretsaw		High Wycombe	1964	Adeno.	Beech, oak, mahogany, African walnut	Mid- turbinate	
012	M	1909	1930	Chair makers	Chair maker		High Wycombe	1957	Adeno.	Beech, oak, mahogany	Mid- turbinate	
013 014	M	1908 1896	(1922) (1910)	Chair, furniture factory	Wood machinist Yard labourer and stoker	3	High Wycombe High Wycombe‡	1965 1956	Adeno. Adeno.	1919-44 stoked furnace with chips and dust of beech, oak, cherry,	Antrum Ethmoid	
015	М	1902	(1916)	Furniture factory	Wood machinist (spindle)	_	High Wycombe	1959	Adeno.	and chestnut Contact with beech	Olfactory	
01 <del>6</del> 017	M M	1894 1908	(1908) 1923	General	Wood machinist Cabinet maker*	1930	High Wycombe High Wycombe	1956 1964	Adeno.	and oak, 1927-60 Oak, mahogany, beech,	Ethmoid	Pagama a plasterer
018	М	1906	1921	furniture Furniture	Cabinet maker*	1926	High Wycombe	1959	Adeno.	Indian lamel, walnut	Antrum Ethmoid	Became a plasterer, date unknown After 1926 plasterer
019 020	M M	1910 1915	(1924) (1929)	factory	Veneer maker Wood machinist	=	High Wycombe High Wycombe	1963 1962	Adeno.		Ethmoid Turbinates	in building trade
021 022 023 024	M F M M	1890 1879 1891 1915	(1904) (1893) (1905) 1954	Army depot	and sander Cabinet maker Chair polisher Brewer's cooper Crate maker	<del>-</del>	High Wycombe High Wycombe Reading Bicester	1958 1960 1956 1964	Trans. Trans. Squ. Squ.	Deal; no hard woods	Nasal cav. Ethmoid Antrum Nasal cav.	Previously labourer
025	F	1909	1923	Furniture makers	Sandpaperer	1931	High Wycombe	1959	Anaplastic		Antrum	in Army depot Same firm as Cases 006, 008, 014
					Oxford, Bu	ckingha	mshire, and Berkshir	re Cases	before 1956	and since 1965	,	I
030	М	1890	(1904)	Furniture manufacture	Benchman	_	High Wycombe	1967	Squ.		Nasal cav.	
031	M	1902	1915	Furniture manufacture	Wood machinist	_	High Wycombe	1966	Adeno.	Mahogany, oak, walnut, beech	Ethmoid	
032	M	1912	1926	Furniture manufacture	French-polisher	_	High Wycombe Stokenchurch	1966	Adeno.	Elm, beech, mahogany and African woods	Nasal cav.	V. occasional snuff taker
033	M	1898	(1912)	Ecclesiastical furniture makers	Clerk	c: 1925	Oxford	1954	Adeno.		Turbinates	From c. 1925 worked as store- man in motor
034	M	1887 1880	(1901)		Timber§ mer- chant's rep.	1	Henley	1954	Adeno.		Ethmoid	industry
035 036 037	M M M	1000	(1094)	Furniture Furniture	Joiner    Wood turner Cabinet maker		Newbury Bledlow Ridge	1954 1948	Adeno. Adeno.		Max. antrum Antrum	
038	M	1901	(1915)	Furniture	Timber yard labourer		High Wycombe Chinnor	1948 1949	Adeno. Adeno.		Max. antrum Max. antrum	
039 040	M M	1873 1901	(1887) (1915)	Furniture Chair manufacture	Chair polisher Chair maker		High Wycombe High Wycombe	1947¶ 1939¶	Unsp. Unsp.		Nasal cav. Ethmoid	
					Cases with known Co.	ntact wi	th Wood Trade Out	side Ox	ford, Buckins	ghamshire, and Berkshire		
051	M	1909	(1915)	manufacture	Cabinet maker	-	?	1960	Adeno.			
052 053	M M	1906 1918	1920 1932	Furniture manufacture Furniture manufacture	Wood machinist (spindle) Cabinet maker	_	Newmarket and Ipswich Edmonton and	1964 1964	Adeno. Adeno.	Oak, walnut, beech, ash, elm, mahogany Oak, mahogany, walnut,	Ethmoid Antrum	
054	м	1918	(1932)	manuracture	Cabinet maker		Tottenham		A -1	ash, beech, and ply- wood		
055 056	M M	1905 1900	(1919) 1919	Furniture manufacture	Cabinet maker Cabinet maker	1940	North London Clapton Loughton, Essex	1964 1961	Adeno. Adeno. Adeno.		Nasal cav. Nasal cav.	
057	M	1905	(1919)	Furniture manufacture	Cabinet maker		East London	1964	Adeno.		Ethmoid	
058	M	1908	(1922)	Chair makers	Chair maker		Walthamstow	1965	Adeno.		Ethmoid	

Not in furniture industry at diagnosis; not counted as such in Table VI. † Same factory. ‡ Same factory. § Association with furniture trade uncertain. ¶ Worked in furniture factory for unknown period from 1901. ¶ Death.

In the 16 patients for whom information is available 15 stated they had been exposed to oak, 11 to beech, and 11 to mahogany. Exposure to polishes, varnishes, etc., was minimal except in Cases 003 and 032. Chair makers, cabinet makers, and veneer makers used glues, but not wood machinists. Inquiries made concerning the use of insecticides and seasoning agents before machining the wood were negative.

A further important result of the occupational inquiries was to confirm that the Banbury and Oxford patients had never worked in the Buckinghamshire furniture industry, and vice versa.

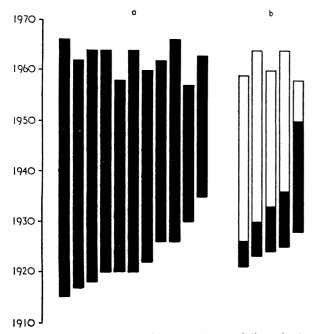


Fig 2.—(a) Year of entry to industry and year of diagnosis of adenocarcinoma in 11 cases where dates are definitely known. (b) Five additional cases where the patient left the industry before diagnosis. The period of exposure is shown in black.

More than one case occurred in two factories. As has already been reported three cases (Table VIII) were traced to a medium-sized factory in Banbury which specialized in oak dining-room suites and piano stools. Three other cases of adenocarcinoma and one of anaplastic tumour (Case 025) occurred in a factory of similar size in High Wycombe making reproduction type furniture and chairs, using beech, oak, and mahogany.

## Secular Trend

An attempt will now be made to determine how the risk of nasal cancer in the furniture industry has behaved in time during the period of study. As the Oxford Cancer Register was established only in 1951, and before 1948 some cases of cancer of the head and neck in High Wycombe tended to be referred to a variety of hospitals in London rather than to Oxford, morbidity data concerning cases occurring before 1950 may well be incomplete. Fortunately, however, it has been possible to examine the death registers for High Wycombe back to 1900. Since that date the population of the town has doubled.

In Table IX the death certificates where nasal cancer was mentioned, and the occupation given was in the furniture industry, are shown in column 2 according to year of death. In column 3 the same cases are shown according to year of diagnosis with the addition of all living cases known to have occurred in persons who ever worked in the industry up to the end of 1967.

In all, 12 deaths have been found. The first occurred in 1939 in a chair maker aged 38 (Case 040). In this case, and in the second which occurred in a chair polisher in 1947

(Case 039), it has proved impossible to determine the histological type of tumour. The first death known with certainty to have been due to an adenocarcinoma in High Wycombe occurred in a cabinet maker who died in 1953 (diagnosed in 1948, Case 037). Another case, not shown in Table IX because the man lived in Buckinghamshire outside High Wycombe, was diagnosed in a wood turner in the same year (Case 036). If the trend in column 2 is taken at its face value the suggestion is that the risk of death from nasal cancer in the industry began at about the time of the second world war and is still on the increase; thus in the decade 1950–9 there were only three deaths, while in the eight-year period 1960–7 there have been

TABLE IX.—All Known Cases of Carcinoma of the Nasal Cavity and Sinuses in Male High Wycombe Furniture Workers 1900-67

	Deaths According to Year of Death	All Known Cases, Including Deaths, According to Year of Diagnosis
1900-9	0	3
1910-9	0	?
1920-9	0	}
1930-9	1*	1*
1940-9	1*	2 (1*)
1950-9	3	6
1960-4	2 7 ~	8 7,2
1965-	5† <b>)</b> '	4† 5 12
Total to end of 1967	12	21

<sup>\*</sup> Two cases where histological type of tumour is unknown. † Including Case 030.

seven, five of which have been reported since 1965. Though the numbers are extremely small, and measurement of trends from death certificate material is open to many errors, it seems unlikely that nasal cancer—which forces itself on the attention of the clinician, and until recently was usually fatal—was prevalent in the High Wycombe furniture industry before the war.

The evidence supports a similar conclusion for Banbury, for a search of the death registers over a similar period revealed the death certificates of Cases 004 and 005, but no other cases of nasal cancer in woodworkers as far back as 1900.

The principal interest of the data in Table IX, column 3, which are classified by year of diagnosis, is that they contain a hint that the outbreak may have reached its peak. Thus while eight cases were diagnosed during 1960–4, only four cases have been diagnosed since the beginning of 1965 and one of these, which was a squamous growth (Case 030), may be irrelevant. A further shred of support is lent to this notion by the fact that outside High Wycombe no further cases have been reported in the Oxford Region since 1965 in the industry. However, careful observation will be necessary for a number of years before there can be any degree of certainty on this point. Whether or not the rate of incidence has reached its peak, the shape of the trends shown in Table IX suggests strongly that further cases will occur in the near future.

#### Discussion

## Woodworkers in Furniture Industry in High Wycombe

The material described in this paper shows beyond any reasonable doubt that certain classes of workers in the Buckinghamshire furniture industry are especially at risk in respect of adenocarcinoma of the nasal cavity and ethmoid sinuses. The trades principally affected are wood machinists, cabinet makers, and chair makers, in whom for High Wycombe the average incidence for the decade 1956-65 was  $0.7\pm0.2$  per 1,000 per annum.

In order to estimate for comparison the risk of adenocarcinoma of the nasal cavity and sinuses in the general male population, information was sought from three other cancer registries in southern and central England which obtain a record of the histological type of most of the tumours registered. The regions and periods covered were as follows: Birmingham Region 1960-6; South Metropolitan Cancer Region (which includes Wessex) 1961-5; South-Western Region 1961-5. During the stated years a total of 18 new cases of adenocarcinoma of this site were registered in a population of 5,284,000 men aged 15-64. This gives for this sex and age group an average annual incidence rate of this tumour of 0.6 per million, about one thousand times less than the rate in High Wycombe woodworkers.

In view of the apparent extreme rarity of this tumour among the general male population, it is reasonable to question the completeness of ascertainment. This might be due either to failure to register some substantial proportion of these tumours in the regions concerned or to failure to report the histology of the tumours. With regard to the first point, the average annual incidence for all types of nasal cancer together1 in males of all ages in the three regions was as follows: Birmingham 7.4; South Metropolitan 7.7; South-Western 12.3. All figures are expressed per million per annum. These figures are not grossly dissimilar from the Oxford incidence rates of 11.2 per million (all types) or 7.6 per million excluding adenocarcinomas (of whom all but three patients had been woodworkers). With regard to the second possibility, the proportions of cases in which the histological type of the tumour was unknown were: Birmingham 10.3%; South Metropolitan 17.4%; South-Western 15.9%. It seems unlikely, therefore, that there is any gross failure to register and classify adenocarcinomas in the regions studied. In order to make a conservative estimate of the increase in risk one might assume that half the cases were ascertained in the other regions. In these circumstances the relative increase in incidence in the High Wycombe woodworkers would be about 500-fold.

#### Woodworkers in Furniture Industry outside High Wycombe

There is strong evidence that the risk extends to woodworkers in the furniture industry in Oxfordshire. According to the 1961 Census (Table I) there were 240 cabinet makers and chair makers and 300 wood machinists in Oxfordshire at that time. In this diminutive population there occurred during the decade three cases (Cases 002, 004, and 005) in persons still in the industry at time of onset, giving an average annual incidence of about 0.6 per 1,000, very close to that found in High Wycombe. Furthermore, there are two other Oxfordshire cases (Nos. 003 and 033) which cannot be used in the computation of incidence. There is also no doubt that the risk extends to the Buckinghamshire furniture industry outside High Wycombe (Cases 032, 036, and 038).

The next important point to consider is whether the risk extends to woodworkers in the furniture industry outside the southern portion of the Oxford Region. The inquiries in relation to this question are not yet complete. Table VIII (Cases 051-058) shows the particulars of eight cases ascertained from cancer registers and hospitals in the London area and East Anglia. In all of these the diagnosis has been made since 1960. According to the 1961 Census there were 12,500 cabinet and chair makers and 19,290 wood machinists in the London, Eastern, and South-Eastern Regions, which correspond approximately with the area of ascertainment. Thus, making the extreme assumption that all the wood machinists worked in the furniture industry, the population at risk is approximately 30,000. Given an average incidence rate in males aged 15-64 of 1 per million per annum, one nasal adenocarcinoma would be expected in about 30 years. The actual occurrence of eight cases in the period 1960-6 gives a provisional annual incidence of about 4 per 100,000 per annum, some 40 times the expected figure. For cabinet makers and chair makers, who account for seven of the cases, the provisional incidence is 8 per 100,000 per annum. These figures almost certainly underestimate the

actual risk, because case ascertainment in London is difficult because two of the registers rarely record either occupation or histological type. At this point, therefore, it can safely be concluded that the risk extends to woodworkers in the furniture industry in other parts of England, but it is not yet clear whether its magnitude is as great as in the High Wycombe area and Oxfordshire.

It is worth noting that the two factories in the original survey area where there were multiple cases (Table VIII) are concerned with the manufacture of general household furniture, not with chair-making, and are 50 miles (80 km.) apart. These factories are more typical of British furniture-making at large than of the local Buckinghamshire tradition. It seems likely, therefore, that a similar industrial environment existed in furniture factories in other parts of Britain.

### Other Workers in Furniture Industry

As in other industrial tumours the risk of adenocarcinoma of the nasal cavity and ethmoid extends, though perhaps to a less extent, to other persons in the industry, including French-polishers (Cases 003 and 032), clerical staff (Cases 033 and 034), and labourers (Cases 014 and 038). One of the difficulties here is that the numbers of persons at risk in these groups are unknown. For example, French-polishers were grouped with painters and decorators at the 1961 Census.

#### Carpenters and Joiners

In the southern part of the Oxford Hospital Region there is no evidence of an increased risk of nasal cancer in carpenters and joiners. This should not be regarded as final evidence that these trades are never at risk, not only because the numbers involved are relatively small but also because the work done by these tradesmen and their relation to the furniture industry may vary in different parts of the country. In this connexion it is of interest that Case 035, though described in hospital records as a joiner, is known to have worked in a furniture factory in Newbury for an unknown period beginning in 1901.

#### Nature of the Hazard

Though the precise nature of the hazard is unknown the evidence accumulated so far narrows the field of possibilities. The site of the lesions makes it likely that the substance (or substances) involved is inhaled. To follow the next step in the argument it must be recalled that, except in very small factories, French-polishing and varnishing of chairs and other furniture, and upholstering, take place in shops separated from the work areas where machining, making up, and sanding of woodwork are done. This is because the presence of more than a certain amount of wood dust in the air may spoil the finish of the product. This arrangement means that the French-polisher and upholsterer have relatively little exposure to wood dust, and on the other hand that the machinist and cabinet maker have relatively little contact with lacquers, sprays, and polishes. When this is borne in mind the fact that the disease is so far unknown in upholsterers, and relatively rare in French-polishers, becomes significant. Thus it is likely that the agent is contained in wood or substances used in the preparation of wood rather than in polish, varnish, or upholstery.

Further characterization of the component or type of wood dust concerned is complicated by the fact that it cannot safely be assumed still to be present in the dust of contemporary furniture factories. The environment which it is necessary to reconstruct is that of the furniture industry in the period 1920–39.

By means of detailed occupational histories taken from the patients or their relatives, and interviews with managers and

<sup>&</sup>lt;sup>1</sup> Excluding sarcomas and lymphomas.

other workers of long service, it has been possible to build up an idea of the industrial environment in which the patients worked before the war. Unfortunately, however, much of the material depends on uncorroborated human recollection, as records of timbers, glues, and other materials used and dates of installation of exhaust ventilation for that period have long since been destroyed. Most of the patients had worked in small firms with fewer than 50 woodworkers and all had been exposed to conditions where there was a great deal of dust. Without exception all had worked in their early years without exhaust ventilation, this having been installed in the larger firms shortly before the war, and in the others more recently. All the Buckinghamshire men had been exposed to beech, one patient (Case 010) almost exclusively, but the exposure of the Banbury and Oxford patients to this material was minimal.

Wood dust in machine shops and making-shops in the furniture trade probably contains not only particles of wood but also, on occasion, particles of silica and carborundum in the vicinity of sanding machines, and of glue where material is sawn after veneering. The presence of these is thought to be too irregular for them to be of significance. Extensive inquiries were made to determine whether woods used before the war were treated with insecticides (in particular those containing arsenic), fungicides, or seasoning agents before being processed; all replies to these inquiries were negative.

Adenocarcinoma of the nasal cavity and sinuses in woodworkers in the furniture industry is almost certainly due to the inhalation of particles of wood. It seems likely that at least one, perhaps more, of the common European hardwoods is involved. At present there is no evidence to incriminate a substance or substances added to the timber before or during its processing in the machine shops or making-shops. Varnishes, lacquers, and polishes cannot be incriminated. If our provisional interpretation of the secular trend is borne out by future events and the outbreak of cases has now reached its peak, it may be that the period of maximum exposure was when mechanization of wood-processing was advanced but exhaust ventilation had not yet been introduced in most places. During this period exposure to dust may have been maximal. This situation existed during the period 1920–39.

Occupational cases of nasal cancer have been reported in three other industries. Doll (1958) reviewed the situation in the nickel industry. Up to 1958 62 cases of nasal cancer had been reported in workers in the Mond Nickel Company, Swansea. Most of these were squamous tumours (L. G. Morgan, personal communication). The relative risk of dying of this cancer for workers in the factory was about 150 times normal in 1948-56. Weil, Smyth, and Nale (1952) described four cases in a plant manufacturing isopropyl alcohol, one of which was an adenocarcinoma. In neither of these industries has the carcinogen been identified, but the risk has been abated by changing or enclosing the process. Nasal cancers have also been reported in workers who used radium to paint on watchdials, and may have been due to inhalation of this substance (Hems, 1967). It is difficult to see how any of these groups can provide a clue to the origin of cases in the furniture industry.

Snuff was commonly taken by furniture workers in this area before the war to clear their noses of wood dust and also, perhaps, because smoking was generally forbidden within the factories. However, as only 3 of the 11 men with adenocarcinoma in whom the appropriate history could be elicited had ever taken snuff, this is unlikely to be relevant.

It is our opinion that adenocarcinoma of the nasal cavity and accessory sinuses in woodworkers and other workers in the furniture industry should now be prescribed as an industrial disease. The natural frequency of this particular type of tumour is so low that it can be assumed that all such tumours in persons who are working or have worked in the furniture industry are occupational in origin.

It is also recommended that a register of woodworkers in the High Wycombe furniture industry at least should be set up so that the situation may be kept under surveillance. The question of the advisability of regular nasal examination of woodworkers in the furniture industry should be studied in the light of the average annual incidence rates shown in Table VII. In the meantime measures should be taken to reduce so far as possible the amount of wood dust present in the air of furniture factories, and workers should be encouraged to wear masks.

Finally, it may be helpful to try to place the risk of adenocarcinoma in woodworkers in the furniture industry in perspective. An annual incidence of 0.7 per 1,000 is similar to that experienced by males from carcinoma of the bronchus or by females from carcinoma of the breast in the Oxford Region (Oxford Regional Hospital Board, 1966). Though the latter figures include persons of all ages, and are therefore not strictly comparable, it is reasonable to conclude that in High Wycombe, at any rate, the risk of adenocarcinoma of the nasal cavity and ethmoid in woodworkers in the furniture industry is of the same order of magnitude as is the risk of the two commonest tumours prevailing in the general population. The effect of the substance or substances concerned is thus to make what is naturally an extremely rare tumour a common one in a restricted group of men.

#### **Summary and Conclusions**

The material reported here shows beyond reasonable doubt that cabinet makers, chair makers, and wood machinists in the furniture industry in the southern part of the Oxford Hospital Region are at special and substantial risk in respect of adenocarcinoma of the nasal cavity and sinuses, especially the ethmoid. The risk is not limited to woodworkers in the Buckinghamshire chair-making industry but is present at the same order of magnitude in persons engaged in the manufacture of general furniture in Buckinghamshire and Oxfordshire. The risk is also present in woodworkers in the furniture industry in other parts of Southern England, but no final opinion can yet be given on whether the magnitude of the risk there is as high as within the Oxford Region. The risk also extends to a significant but lesser extent to persons employed in other trades exposed to dust in the furniture industry. In this area carpenters and joiners never employed in the furniture industry have either no increased risk or an increase in risk which is not detectable in a population of this size. The best estimate of the latent period of the tumour is 39 years, but tumours may develop after as little as five years' exposure in persons who have left the industry.

It is concluded that a constituent or constituents of wood dust which is inhaled and is present in such commonly used hardwoods as oak and beech is aetiologically related to the development of these tumours. Polishes, lacquers, and varnishes are unlikely to be incriminated. The factor concerned was present in the industry in this area as early as 1920 and at least as recently as 1940. It is not known whether it is still present, but from the temporal distribution of the cases, and bearing in mind the long latent period of the tumour, it is likely that further cases will occur in any event.

It is recommended that the epidemiological situation should be watched carefully. The condition should be prescribed, and further efforts be made to reduce the concentration of wood dust in furniture factories and to encourage the use of masks.

ADDENDUM.—Since this was written a further case of adenocarcinoma of the nasal cavity, diagnosed in December 1967, has come to light. This man (Case 041) had left High Wycombe but had been employed in the furniture industry there as a chair maker from 1919 to 1947.

#### **Appendix**

Excerpts of occupational histories in males which were taken from the patients or their relatives.

## Thirteen Patients with Adenocarcinoma who Worked in Furniture Industry

Case 001.—Entered the furniture industry in 1928, working as a band sawyer in two firms in High Wycombe until 1950, when he became a vacuum sweep, continuing in this business until an adenocarcinoma was diagnosed in 1958. Never took snuff. This was the only case in which there had been exposure to dust other than wood dust (soot). He always wore a mask as a sweep.

Case 002.—Entered the furniture industry in 1917 as an apprentice cabinet maker. The whole of his working life was spent in the furniture industry in Oxford. From 1917 to 1929 he made sectional bookcases, principally out of American oak. From 1929 to the beginning of his illness in 1962 he worked for a small firm making school and educational furniture, for which the principal woods used were American and Austrian oak, Parana pine, Douglas fir, Spanish chestnut, Cyprus pine, and a very small quantity of beech. According to his sister-in-law, with whom he lived, he used to come home at night covered with dust. He was never exposed to smoke, and was not a snuff taker.

Case 003.—Started work in 1924 as an apprentice Frenchpolisher at a furniture factory in Banbury. Left this trade in 1933 because of "polisher's rash," Moved to Oxford and entered the motor industry, where he worked until the onset of his illness in 1960. During his time in the motor industry he worked principally in the wiring department and was not exposed to dust. He never took snuff.

Case 004.—Started work in 1923 as an errand boy for a firm of dyers and cleaners. In 1925 he transferred to the same furniture factory in Banbury as Case 003, where he worked as a wood machinist up to 1936. He then left for an aluminium foundry, where he worked as a furnaceman until 1964, when his adenocarcinoma was diagnosed. He never took snuff. During the war he was in the Auxiliary Fire Service for the works, but attended few fires and was not exposed to much smoke.

Case 005.—Started work in 1920 in an iron foundry but left in 1922. From 1922 to 1960 he worked as a wood machinist, principally on a dimension saw, until the diagnosis of his adenocarcinoma in 1960, in the same factory in Banbury as Cases 003 and 004. The principal woods used before the war were oak, mahogany, and sapele. No beech was used. Canadian fir and spruce were used for air-frame manufacture during the war. An extractor has been in use in the machine shop in this factory since before the war, but the conditions at that time were described as very dusty. This worker was also in contact with glue, as some of the work was veneered before it was sawn up. He was never a snuff taker.

Case 006.—Started work in 1915, working for a few months as a goldsmith and silversmith, and later learning the craft of cabinet making in Ipswich. In 1923 he moved to High Wycombe, where he worked as a cabinet maker and table maker until the onset of his illness in 1963. Throughout this period of 40 years he worked in one firm which employed about 200 men. After the war he became an inspector in the same firm. He never worked in chair making or veneering. The principal woods to which he was exposed were oak, mahogany, walnut, beech, and some African woods, the names of which are unknown. Exposure to certain types of mahogany used to bring on coryza, known as "mahogany told." According to his wife he brought home a certain amount of dust on his clothes, and this used to stain his handkerchiefs. He was a very occasional snuff taker.

Case 007.—Started work in 1918 as an apprentice cabinet maker. Worked in a number of different firms in this trade in High Wycombe. Before the war he was exposed to mahogany, oak, and a certain amount of beech. Always worked in the cabinet-making shop, never on chairs. As the previous patient (Case 006) he also suffered from "mahogany cold." According to his wife his underclothes were often pink with sawdust. He never took snuff.

Case 008.—In 1915 started work in grocery trade, in which he worked until 1918. After one year in the bakery business he entered the furniture industry in High Wycombe in the same factory as Case 006, and worked as a chair maker until an adenocarcinoma

was diagnosed in 1958. He was occasionally concerned with table making and veneer matching. Before the war he was exposed principally to oak and beech, and, to a lesser amount, to mahogany. He suffered from catarrh and sinusitis all his life. He never took snuff

Case 009.—Started work in 1926 in the High Wycombe furniture trade as an apprentice chair maker. Worked in a number of firms, always as a chair maker, and before the war was exposed to mahogany, beech, and oak. He had never suffered from nasal trouble before the onset of his illness in 1961. He occasionally took smiff before the war.

Case 010.—This man had nasal diphtheria as a child. He entered the furniture industry in 1935 as a wood machinist in a small firm with 10 employees; here he worked until the onset of his illness in 1963. Until 1947 this firm did not have any dust extraction except over the bandsaw. The machines this man worked principally were the bandsaw, the spindle moulder, the tenoner, and the borer. The timber used was almost exclusively local beech from the Cotswolds and Chilterns seasoned in their own yard. Very small quantities of oak and African cherry were also used. His habits with regard to snuff are not known.

Case 011.—Started work in 1914 as an errand boy at a chemist shop. After service in the Army he entered the High Wycombe furniture trade in 1920 as a machinist, working principally with the mortising machine and fretsaw. From 1920 to 1929 he worked in a small firm of chair makers who had no extraction plant. From 1929 to the onset of his illness in 1964 he worked on a fretsaw at a larger firm of chair makers which had effective exhaust ventilation from the beginning of his time there. Before the war he was exposed to mahogany, African walnut, beech, and English and Russian oak. His habits with regard to snuff are not known.

Case 031.—Started work in 1915 as a general wood machinist in the sawmill of a firm of seat makers in High Wycombe. He worked later in two other firms, neither of which had any extraction plant. He was exposed to beech, plywood, mahogany, oak, and walnut. He remained in the business until the onset of his symptoms in 1965. He never took snuff.

Case 032.—Entered the High Wycombe furniture business as a French-polisher in 1926, and remained in this trade, working at various firms until the onset of his illness in 1966, apart from a period in the Army between 1940 and 1946. Up to 1957 he worked with French polish only on elm, beech, mahogany, and some African woods. Since 1957 he has used various synthetic lacquers and sprays. Throughout his career he has used sandpaper after sealing or damping on polish with a wad of cotton. This creates some fine dust. This man was a very occasional snuff taker.

# Patient with Squamous Carcinoma Exposed to Wood Dust outside Furniture Industry

Case 024.—This man also suffered from a hereditary form of cataract, for which he had operations between 1933 and 1935. Started work as a butcher's errand boy in 1930 until his vision failed in 1933. Worked as a storeman in an Army depot from 1941 until 1954. From 1954 until diagnosis of squamous carcinoma in 1964 worked as rip-sawyer and crate maker in sawmill in Army depot. This was dusty work, as for several years there was no extraction plant. Low-quality soft woods only were used. He never took snuff.

#### Five Other Cases (not Shown in Table VIII)

Case 201.—Born in 1896. Started work as a farm labourer in 1910. From 1912 to 1922 served in the regular Army. From 1922 to 1926 worked as a porter and shunter on the railway. In 1926–7 worked loading and unloading cement at a cement works. This was extremely dusty. For the remainder of his career until an anaplastic carcinoma was diagnosed in 1960 he worked as a storeman at an R.A.F. station. He never took snuff.

Case 202.—Born in 1911. Started work as a shipbreaker in 1926. Moved to road work (tarring) 1930–2. Later worked as a steel erector. From 1938 to diagnosis of squamous carcinoma in 1962 worked as slinger and crane driver at steel works. Denies exposure to dust. Never took snuff.

Case 203.—Born in 1911. Started work as a carrier in 1924 for a firm loading lime, cement, tea, and sugar. In 1926 moved to work as storeman to a builder's merchant. Again exposed to lime and cement dust. From 1936 to 1964 worked principally as a plasterer. The histology of this tumour is not known. He never took snuff.

Case 204.—Born in 1898. After working as a packman's boy (1910-12) and an apprentice slaughterer (1912-14), and serving in the Army (1914-17), he worked as a labourer for grain merchants (1917-36) and was exposed to the dust of cereals and beans. From 1946 to 1961, when an anaplastic carcinoma was diagnosed, he worked in the quarry of a cement works but was not much exposed to dust. He never took snuff.

Case 205.—Born in 1896. Started work as a baker's assistant in 1908 and was exposed to flour dust until he left to work at an Army depot in 1914. After service in Army worked at a power station unloading coal trucks (1919-25) and later as boilerman, stoker, and storeman (1925-60), when he was exposed to coal dust and ash. A transitional cell tumour was diagnosed in 1960. He never took snuff.

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## Brucella abortus in Fresh Cream and Cream Products

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Several samples of cream were examined in addition to milk from herds of cows and goats when seeking the source of brucella infection in two children. The source of these infections was not established, but the samples of heat-treated creams were of poor bacteriological quality. They reduced methylene blue rapidly, contained large numbers of faecal coliform organisms, and showed considerable phosphatase activity. Bacteriological examination of further samples of cream gave similar results. In view of these findings a survey of fresh cream and cream products was undertaken. The laboratory results obtained in this survey, in which 1,161 samples of cream were examined bacteriologically for keeping quality and hygiene, have been described elsewhere (Barrow and Miller, 1967). During the survey Brucella abortus was isolated from routine samples of cream or cream products from five different processing dairies. Though it is well known that brucella organisms may be present in milk, and that they usually become concentrated in the cream, we are not aware of any investigations into the presence of Br. abortus in cream or cream products. In view of this, and of the current interest in brucellosis in both man and animals, we describe in this paper the circumstances associated with these isolations of Br. abortus from creams, together with details of the subsequent investigations.

This account illustrates some of the public health hazards associated with cream, and indicates the need for more frequent sampling, for minimum bacteriological standards, and for improved legislation similar to that at present required for milk and ice-cream.

#### **Bacteriological Methods**

Hygiene and Keeping Quality.—All the samples of cream were examined by the methylene blue reduction test and the phosphatase test: the numbers of viable bacteria, coliform organisms, and faecal coli present were also determined. The methylene blue reduction test, volumetric method, described for

cream by a working party of the Public Health Laboratory Service (1958) was used. Phosphatase activity was determined by the Aschaffenburg-Mullen method, as described in the Milk (Special Designation) Regulations, 1963 (Great Britain, 1963). Quantitative estimations of the numbers of coliform organisms and of Escherichia coli type 1 present, if any, were made by both surface and pour-plate methods with MacConkey agar medium. Most of the strains of presumptive faecal coli were confirmed as E. coli type 1 by their biochemical reactions. The total numbers of bacteria present were estimated by the method of Miles and Misra (1938) with blood agar medium. Further details of the techniques used are given by Barrow and Miller

Direct Culture for Brucella abortus.—Because of poor initial results with the tests of keeping quality and hygiene, all samples of creams, irrespective of heat treatment, were cultured for Br. abortus, usually within one hour of receipt in the laboratory. The samples of cream were stirred with cotton-wool swabs or with wooden spatulae, previously sterilized in bulk. Each swab was rubbed over the surface of one plate of brucella agar medium in a suitably vented Petri dish. The inoculated plates were incubated in an atmosphere of CO<sub>2</sub> at 37° C. for four to five days. The selective brucella agar medium used and the procedure for incubation in CO2 were the same as those described by Barrow and Peel (1967). Colonies of brucellae were identified by their appearance, sensitivity to dyes, and slide agglutination reaction with antiserum. They were confirmed subsequently by type-identification; all were Br. abortus type 1.

Enrichment Culture and Guinea-pig Inoculation.-In addition to direct culture for brucella organisms, some of the

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