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Atmospheric Pollution and Lung Cancer

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Lung cancer is consistently more common in urban areas than in rural. The excess cannot be accounted for by specific occupational hazards but some of it might be due to the presence of carcinogens in urban air. The excess cannot be wholly due to such agents, because the excess in nonsmokers is small and variable. Cigarette consumption has also been greater in urban areas, but it is difficult to estimate how much of the excess it can account for. Occupational studies confirm that pollutants present in town air are capable of causing lung cancer in man and suggest that the pollutants and cigarette smoke act synergistically. The trends in the mortality from lung cancer in young and middle-aged men in England and Wales provide uncertain evidence but support the belief that atmospheric pollution has contributed to the production of the disease. In the absence of cigarette smoking, the combined effect of all atmospheric carcinogens is not responsible for more than about 5 cases of lung cancer per 100,000 persons per year in European populations.

Urban-Rural Gradient

The idea that lung cancer might be caused by agents in the ambient atmosphere has provided a stimulus for research ever since it was realized that the mortality attributed to the disease was consistently higher in towns than in the countryside. The differences observed were not in general large, being commonly about two-fold, but it is notable that the mortality increased progressively with the size of town. Some typical data for both mortality and incidence are shown in Tables 1-3 and elsewhere in this issue (*1*).

The excess urban mortality could be an artifact due to greater ease of access to hospitals and better diagnosis, but this is unlikely to be the explanation for much of the difference as it has persisted despite the more even spread of medical services. Moreover, case-control studies have shown that patients with lung cancer tend to have lived in towns for longer periods than matched control patients with other diseases (*5*). Some small part could be due to specific occupational hazards, but this also is unlikely to be a major factor as the known and suspected hazards (Table 4) affect only a small proportion of the total urban population. It seems, there-

Table 1. Mortality from lung cancer in England and Wales, 1950–1973, by sex and place of residence (standardized for age).

	Annual death rate per 100,000							
	Male				Female			
Area	1950	1960	1970	1973	1950	1960	1970	1973
Greater London	73.4	117.1	134.8	132.2	10.8	15.9	23.6	25.8
Other conurbations	63.4	101.8	131.7	133.2	9.8	12.3	19.8	22.5
Towns more than 100,000 population	59.6	101.8	117.7	121.1	8.2	12.1	17.9	19.5
Towns 50,000 to 100,000 population	46.1	93.6	108.9	113.1	7.0	10.9	16.8	20.6
Towns less than 50,000 population	42.5	78.5	100.1	101.1	7.1	9.2	16.5	18.0
Rural areas	39.7	63.7	89.9	89.7	6.2	9.2	14.8	16.7

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Table 2. Mortality from lung cancer in U.S.A., 1958: males, by area of residence.[a]

Density of population	Standardized mortality ratio	
	Metropolitan counties	Nonmetropolitan counties
500,000 or more	123	—
150,000–499,999	111	—
10,000–149,999	164	89
2,500–9,999	107	84
Rural, nonfarm	91	80
Farm	—	65

[a]Data of Haenszel et al. (*2*).

Table 3. Incidence of lung cancer in Scandinavia by sex and place of residence.

Sex	Area	Annual incidence per 100,000, 1959-62		
		Norway[a]	Finland[a]	Denmark[b]
M	Capital	39.9	99.8	90.7
	Other towns	23.9	91.6	44.0
	Rural areas	10.0	69.9	20.8
F	Capital	4.1	6.3	11.6
	Other towns			6.5
	Rural areas	3.1	4.3	4.4

[a]Standardized on total population of both countries in 1960 (*3*).
[b]Standardized on European standard population: suburbs of capital omitted (*4*).

Table 4. Occupational causes of lung cancer.

Agent	Industry
Ionizing radiations (radon)	Various mines in GDR, Czechoslovakia, USA, Newfoundland, England, and Sweden
Polycyclic hydrocarbons from combustion of coal	Manufacture of coal gas and coke
Chrome ore	Manufacture of chromates
Nickel ore	Refining of nickel
Asbestos	Asbestos mining, manufacture of asbestos textiles, insulation work, ship-building and breaking etc.
Arsenic	Manufacture of pesticides
Mustard gas	Manufacture of poison gas
Bischloromethyl ether	Manufacture of ion-exchange resins
(?) Vinyl chloride	Manufacture of PVC
(?) Unknown	Rubber industry, manufacture of tires

fore, either that the greater part of the excess must be due to a factor in the general urban environment that causes lung cancer, or that people who live in towns are more likely to develop the disease because of some personal characteristics that distinguish them from people who live in the country.

Carcinogens in the Atmosphere

That some factor in the urban environment might be responsible for the development of the disease appeared likely when it was discovered that the atmosphere contained a variety of substances that caused cancer in animals in laboratory experiments and also caused cancer of the lung in men who were exposed to large amounts in the course of their work. Such substances, which were present in greater amounts in urban air than in rural, included a range of polycyclic hydrocarbons produced by the combustion of fossil fuels, asbestos, arsenic, and radon. The question was, therefore, whether the quantities in the air were sufficient to produce the disease and, if so, how much of the observed mortality they could be responsible for.

Sex Differences in Incidence

That they were unlikely to be responsible for many cases was suggested by the fact that lung cancer was almost everywhere commoner in men than in women, as is shown in Tables 5 and 6. Apart from specific exposure at work, there was no reason why men should be more exposed to atmospheric agents than women, so that, if these agents were responsible for the majority of lung cancers, we should have to postulate either that the disease was overlooked in women or that men were more susceptible to its development. Autopsy studies provide no evidence of gross underdiagnosis in women and there is increasing evidence that women

Table 5. Age-standardized mortality per 100,000 persons from lung cancer in 24 countries, 1966–1967.[a]

Country	Mortality per 100,000	
	Male	Female
Scotland	78.14	11.71
England and Wales	69.66	10.73
Finland	61.00	3.91
Netherlands	56.36	3.42
Austria	50.35	6.09
Belgium	50.09	4.36
U.S.A. (nonwhite)	44.83	7.14
Northern Ireland	43.29	7.14
Germany (Fed. Republic)	42.09	5.10
U.S.A. (white)	39.62	6.70
New Zealand	37.72	5.35
Australia	37.64	4.77
South Africa (white)	37.63	6.93
Denmark	37.34	7.36
Switzerland	37.33	3.33
Canada	34.52	5.36
Ireland	33.54	7.88
Italy	30.23	4.53
France	27.71	3.74
Israel	22.51	7.62
Sweden	17.35	4.34
Chile	15.18	5.59
Norway	14.93	2.97
Japan	13.97	4.86
Portugal	10.91	2.74

[a]Data of Segi and Kurihara (*6*).

Table 6. Age-standardized incidence per 100,000 persons from lung cancer in selected areas since 1970.[a]

Area	Male	Female
England, Liverpool	89.5	14.8
England, Birmingham	77.1	11.5
U.S.A. Detroit (black)	77.1	13.7
Finland	76.5	4.6
Rhodesia, Bulawayo (black)	70.7	3.1
New Zealand (Maori)	67.1	35.4
Switzerland	66.4	7.5
F. R. Germany, Hamburg	63.0	9.2
Poland, Warsaw	60.0	10.2
Singapore (Chinese)	56.9	17.3
GDR	56.2	4.9
U.S.A. Connecticut	53.7	12.2
New Zealand (non-Maori)	48.8	8.0
Cuba	44.7	16.1
Canada, Quebec	41.7	6.0
Denmark	40.2	7.2
Israel (Jews)	29.3	10.4
Brazil, Sao Paolo	25.0	5.1
Spain, Zaragoza	23.5	4.6
Norway	22.2	4.7
Sweden	21.3	5.1
Jamaica, Kingston	21.2	5.0
Japan, Miyagi	20.0	7.0
Colombia, Cali	18.6	5.1
U.S.A. New Mexico (Spanish)	16.7	11.4
India, Bombay	13.5	3.1
Sinapore (Indian)	10.0	7.9
Nigeria, Ibadan	0.8	0.8

[a]IRAC data (*7*).

are perfectly capable of developing the disease if they are exposed to the same extent as men. We may note, for example, that (1) in Maori women, who smoke more than any other female population, the disease is more than twice as common as in other female groups (Table 6), and (2) in women in England and Wales the mortality at ages 45–59 years increased between 1953 and 1973 from 11% of that in men to 27% (men 125.2 and 123.3 per 100,000; women 13.6 and 33.1 per 100,000).

Temporal Correlations

In many countries, the timing of the increase in the mortality from lung cancer is also difficult to associate with the changes in the concentration of atmospheric carcinogens. The increase in the UK and many other European countries occurred so long after the increase in the consumption of coal (*8*) that we cannot attribute any of it to the increase in urban smoke. Nor can it be attributed to pollution by diesel fumes, as diesel engines became common only after the increase in mortality was established. This point is, however, not worth pursuing in detail, as it is impossible to determine with sufficient accuracy how much of the increase in lung cancer is real and how much is attributable to improvements in diagnosis.

Cigarette Smoking

That carcinogens* in the ambient atmosphere are not alone sufficient to cause the current epidemic of lung cancer was finally demonstrated when it was shown that the disease was relatively rare in lifelong nonsmokers and that the incidence increased approximately in proportion to the number of cigarettes smoked per day. For the purpose of the present discussion it will be assumed that the association between the amount smoked and the incidence of the disease is causal, in the sense that, in the absence of cigarette smoking, the majority of cases would not have occurred. (Nearly all the published results agree in showing that the risk of lung cancer is much more closely related to cigarette smoking than to pipe or cigar smoking and, for simplicity, only cigarette smoking will be referred to. Comparisons between the effect of smoking tobacco in different forms are complex and it is possible that different results may be obtained in different countries, because of differences in the type of tobacco used or in the way pipes and cigars are smoked.) The results of one prospective British study are summarized in Table 7, and others are summarized by Friberg and Cederlöf (*1*). These show that the difference between mortality rates in lifelong nonsmokers and in heavy smokers is large (up to 30-fold) and much larger than that commonly observed between the rates in urban and rural residents. Moreover, the mortality in lifelong nonsmokers has invariably been found to be low in comparison with the national mortality rate, irrespective of place of residence. If, therefore, there were any substantial differences in cigarette smoking in town and country, these might account for the urban-rural differences in mortality.

Table 7. Mortality from lung cancer by amount smoked.[a]

Smoking habits	Death rate per 100,000 men, standardized for age
Lifelong non-smoker	7.4
Current cigarette smoker[b]	
1–9 per day	37.1
10–19 per day	78.0
20-24 per day	116.8
25-29 per day	150.0
30–34 per day	212.1
35 or more per day	227.9

[a]Unpublished data from study reported by Doll and Peto (*9*).
[b]Smoking only cigarettes, started under 25 years of age.

That cigarette smoking became common first in large cities is a matter of common observation, but there are very few data that throw any light on the extent to which this could contribute to the "urban factor" in the etiology of the disease. A survey car-

ried out by the Tobacco Research Council (*10*) in the UK showed that men and women in conurbations still smoked twice as many cigarettes as men in "truly rural" parts of the country as recently as 1970 (Table 8). Such figures are, however, inadequate to allow calculations of the extent of the difference in the incidence of lung cancer to be expected from differences in smoking habits, as the incidence of the disease is affected materially by several aspects of smoking other than the number of cigarettes currently smoked, including, in particular, the age at starting to smoke, the duration of smoking, and the manner of smoking (e.g., whether the smoke is inhaled, the number of puffs per cigarette, and the length of the butt thrown away).

Several investigators, from Stocks (*11*) onwards, have tried to allow for differences in smoking habits, using either retrospective studies of cases and controls or prospective cohort studies of men with known smoking and residential histories. The results of two British studies are shown in Tables 9 and 10, and others are summarized by Friberg and Cederlöf (*1*). None reveals any major difference in the incidence of lung cancer among nonsmokers no matter where they live, although several suggest that the effect of smoking a given amount may be greater in large towns than in the countryside. In view, however, of the many aspects of cigarette smoking that affect the incidence of the disease, it may be doubted whether any of the investigators have successfully standardized for differences in smoking habits, by the simple maneuver of comparing men in (say) three categories who smoke different numbers of cigarettes per day.

Table 8. Average cigarette consumption among men and women in urban and rural districts in 1970.[a]

	Average cigarette consumption, cigarettes/day/adult	
Type of district	Men	Women
Conurbations	12	6
County boroughs	11	7
Other urban districts	10	6
Rural districts:		
Total	9	5
Truly rural	5	3
Other rural	8	5
England and Wales	10	6

[a]Data of Tobacco Research Council (*10*).

Table 9. Lung cancer mortality rates for men aged 35–74 years in Liverpool and rural North Wales, by number of cigarettes smoked: standardized for age.[a]

	Death rate per 100,000 per year	
Cigarette consumption	Rural N. Wales	Liverpool
Nonsmoker	22	50
About 10 a day	68	168
About 20 a day	147	248
About 30 a day	232	389
About 40 a day	344	327

[a]Estimated by Stocks (*11*) from retrospective data. Larger differences for nonsmokers are commonly quoted from a paper by Stocks and Campbell (*12*) which presented the preliminary results of the study finally reported by Stocks (*11*). The rates for nonsmokers are likely to be too high because of the method of inquiry, which obtained data about lung cancer patients from relatives after the patients had died.

Interaction of Smoking with Other Agents

If this were the only evidence, we should have to conclude that carcinogens in the general atmosphere have little or no effect on the incidence of lung cancer. The position is complicated, however, by the discovery that several factors may produce a major effect in the presence of another factor when they produce no measurable effect on their own. In particular, there is now evidence to suggest that exposure to either radon or asbestos increases the absolute risk of bronchial carcinoma to a much greater extent in cigarette smokers than in non-

Table 10. Mortality from lung cancer in British doctors by place of residence and amount smoked.[a]

	Current cigarette numbers smoked per day			
Area of residence	0	1–14	15–24	25 or more
Rate, % of rate in all areas				
Conurbations (84)[b]	35	101	109	88
Towns, 50,000 population or over (55)	70	82	114	136
Towns under 50,000 population (62)	257	113	82	104
Rural area (36)	87	98	93	79
Rate per 100,000 men per year				
All areas	10	78	127	251
(237)	(7)	(47)	(77)	(106)

[a]Unpublished data from study reported by Doll and Peto (*9*).
[b]Numbers of deaths in parentheses.

Table 11. Mortality from respiratory cancer in white uranium miners* by cigarette smoking and exposure to ionizing radiations.[a,b]

Smoking habits	Annual death rate per 1000 men for various industrial exposures in "working level months"[c]		
	1–359	360–1799	1800 or more
Nonsmokers	0.2 (1)	0.9 (3)	1.4 (2)
Current smokers			
1–19 cigarettes/day	1.6 (5)	1.1 (3)	8.3 (6)
20 cigarettes/day	1.3 (9)	3.5 (29)	9.4 (30)
More than 20 cigarettes/day	2.7 (8)	4.7 (17)	13.3 (15)

[a]Data of Archer et al. (*13*).
[b]Five or more years after start of mining.
[c]Numbers of cases in parentheses.

Table 12. Deaths from lung cancer in asbestos insulation workers, by smoking habits.[a]

Smoking habits	No. of men	No. of deaths	
		Observed	Expected[b]
History of cigarette smoking	9,590	179	31.60
History of smoking pipe and/or cigars only	609	1	3.11
Never smoked	1,457	1	4.40
Not known	6,144	94	16.76

[a]Data of Selikoff and Hammond (*14*).
[b]From U.S. death rates for white males irrespective of smoking habits.

smokers (Tables 11 and 12). If this is so, the failure to observe any material increase in the incidence of lung cancer in nonsmokers who live in large towns is not conclusive, and the question has to be asked whether the increase in urban heavy smokers reflects subtle differences in smoking habits that have not been taken into account or whether it is due to the interaction of carcinogens in cigarette smoke with others in the ambient atmosphere.

Dose–Response Relationship for Carcinogens in the Atmosphere

The only way we can now answer this question is by quantifying the effect of exposure to a carcinogen in men who have been specifically exposed to large doses in the course of their work and extrapolating back to the effect of the small doses that are inspired from the ambient atmosphere, on the assumption that the incidence of the disease is linearly proportional to the concentration of the carcinogen to which the population is exposed (*15*) and that smoking habits are the same.

Very few sets of data are as yet available to allow such extrapolation. In one study, Doll and his colleagues (*16*) found that men making coal gas suffered a mortality from lung cancer of 306 per 100,000 a year (based on 54 deaths), which was 70% more than that suffered by other men in the same employ who were not so exposed. In another, Hammond and his colleagues (*17*) found that roofers and waterproofers working with pitch and asphalt had 59% more deaths from lung cancer than would have been expected from national mortality rates if they had been members of the relevant Trades Union for at least 20 years (99 deaths against 62.3 expected). Much higher relative risks were found for gas generator workers in Japan (33 to 1) (*18*), and for coke-oven workers in the USA who had been employed for five years or more in occupations which required full-time work above the coke batteries (10 to 1) (*19, 20*). Measurements of the amount of benzo[a]pyrene or other polycyclic hydrocarbons that were likely to have been inhaled were, however, made only for the first two groups (*17, 21*). These are summarized in Table 13, together with figures for urban residents estimated on the assumption that nonmanual workers inhale on average 12 m^3 of air per day (*21, 22*).

Comparison between the workers and urban residents is complicated by lack of precise information about the men's duration of employment, the relative effect of exposure for different periods [which may vary according to the fourth power of the duration (*23*)], and the extent to which atmospheric carcinogens and cigarette smoke interact. If they act

Table 13. Amount of benzo[a]pyrene inhaled by groups of men experiencing different mortality rates from lung cancer.

Group	Mean amount of benzo[a]pyrene inhaled per day, μg
Coal-gas workers (U.K.)	20
Roofing workers (U.S.A.)	17
Urban residents (U.K.)	0.1 to 1.0
Urban residents (U.S.A.)	0.03 to 0.4

synergistically, duration of exposure before the start of cigarette smoking may be relatively unimportant. In this case, town air could hardly be responsible for more than about 10 cases of lung cancer per year per 100,000 men with average smoking habits.

Even fewer data are available for asbestos. The maximum concentration that has been found near building sites where asbestos was being sprayed is three orders of magnitude less than that which has been regarded as an acceptable concentration in the asbestos industry (0.1 mg/m^3), and the amount that is commonly present in town air is still less by another two orders of magnitude (*24*). (Comparison is complicated by variation in the size and shape of air-borne particles and fibers which modify their biological effect.) Unfortunately, we are still uncertain about the size of the risk that is associated with this "accepted concentration." In one study of an asbestos textile factory in England (*25*) it was found that the relative risk of lung cancer among men who had been employed for 20 years in "scheduled areas" (i.e., areas with specific exposure to asbestos dust) decreased progressively as the date of first employment became more recent (Table 14). Present exposure is close to the "accepted concentrations"; but initially it was gross. Unfortunately, detailed dust records were not obtained in a way that enables them to be compared with present data until 1951, by which time the conditions in the factory had been transformed. Even then the mean dust level is likely to have been 3½ times greater than it is now, and we shall have to wait for many years yet before industrial data enable us to make a reasonable estimate of the possible risk associated with the "accepted concentration" of two fibers per milliliter (equivalent to 0.1 mg/m^3).

Estimates of the effect of background radiation can be derived in a similar way from the occupational experience of underground miners exposed to radon. All agree in indicating that the effect is, at the most, small (less than 5 deaths per 100,000 persons per year). Most of the radiation is "natural" (from outer space and the radioactive elements ubiquitously present in buildings, soil, and our own bodies) and only a small part of it can be attributed to pollution from either the combustion of coal or nuclear reactors.

Recent reports of an excess mortality from lung cancer in counties in the USA in which the air is likely to have been polluted with vinyl chloride (*26*) or arsenic (*27*) cannot yet be interpreted. The former substance has not been proved to be carcinogenic in the human lung. The latter has; but the amount in the air under industrial conditions (*28*) has been many orders of magnitude greater than that likely to be present in the general atmosphere.

Trend with Reduction in Pollution

One further source of evidence that could be of value is the trend in the incidence of the disease when pollution is reduced. Any real effect that may have occurred in recent years is less likely to be confounded with the sort of change in diagnostic standards that complicated the interpretation of the previous increase in incidence, but unfortunately it still has to be distinguished from the effect of almost contemporaneous changes in cigarette smoking. These include not only changes in the number of cigarettes smoked by men and women of different ages, but also changes in the extent of use of filters and in the type of tobacco.

In England and Wales, the mortality from lung cancer in men is now declining at all ages under 65 years. The decline began at ages 35 to 39 years around 1955, spread to the next three 5-year age groups 5 years later, to ages 55 to 59 years 10 years later, and to ages 60 and 64 years 15 years later (Table 15). Corresponding changes in the number of cigarettes smoked have been less (Table 16), but the number smoked has decreased since 1954 at all ages under 65 years. Other changes include a change in the type of tobacco smoked and a switch to the use of filter tipped cigarettes, which began in 1950 and continued until 1970, when filter-tipped cigarettes

Table 14. Mortality from lung cancer among men employed in an asbestos textile factory.[a]

Employment history	Number of deaths from lung cancer		Ratio observed to expected deaths
	Observed[b]	Expected	
20 years or more in scheduled area			
10 or more years before 1933	15 (3)	1.58	9.5 to 1
Less than 10 years before 1933	10 (3)	3.05	3.3 to 1
None before 1933	9 (2)	5.56	1.6 to 1

[a]Data of Kinlen et al. (*25*).
[b]Numbers of deaths due to pleural mesothelioma in parentheses.

Table 15. Changes in the mortality from lung cancer in England and Wales, 1950–1973, by sex and age.

Sex	Age	Death rate per 100,000 persons per year				
		1950–54	1955–59	1960–64	1965–69	1970–73
M	35–39	*9.7*	9.2	8.9	7.8	6.1
	40–44	24.6	*25.5*	22.5	21.5	19.2
	45–49	57.9	*59.2*	56.1	53.2	51.7
	50–54	119.4	*125.0*	122.9	116.2	108.0
	55–59	187.1	229.4	*231.3*	222.2	214.2
	60–64	238.6	318.4	365.8	*373.3*	364.1
	65–69	268.5	374.3	463.1	525.0	*527.2*
	70–74	235.9	365.9	479.4	595.2	*684.7*
	75–79	183.5	305.3	435.4	566.8	*700.3*
F	35–39	2.8	2.9	3.2	*3.2*	2.9
	40–44	5.2	5.5	6.9	*7.8*	7.3
	45–49	8.4	10.1	13.0	15.5	*17.9*
	50–54	13.6	16.4	20.1	27.2	*31.3*
	55–59	19.7	23.9	28.9	38.1	*46.4*
	60–64	27.1	31.5	40.5	49.5	*62.6*
	65–69	34.0	37.0	47.7	65.3	*75.0*
	70–74	37.5	43.0	52.5	68.7	*84.2*
	75–79	42.0	46.1	57.2	67.3	*86.2*

Table 16. Changes in cigarette consumption in U.K., 1950–1972, by sex and age.[a]

Sex	Age	Number of cigarettes smoked per person per year					Equivalent number of "constant-tar" cigarettes smoked	
		1950–54	1955–59	1960–64	1965–69	1970–72	1965–69	1970–72
M	35–39	4470	*4800*	4610	4230	4220	3640	2885
	40–44	4470	*4800*	4610	4350	4250	3728	2907
	45–49	4470	*4800*	4610	4060	4580	3501	3122
	50–54	4380	*4650*	4540	4000	4120	3448	2815
	55–59	3900	*4150*	4020	3690	3450	3175	2357
	60–64	3030	*3510*	3500	3130	3430	2701	2348
	65–69	1800	2200	2460	2620	*2700*	2253	1850
	70–74	1360	1670	1870	1900	*2170*	1646	1473
	75–79	1060	1290	1440	1530	*1600*	1346	1087
F	35–39	1460	2040	2350	2510	*2700*	2164	1842
	40–44	1660	2320	2670	2800	*3180*	2411	2175
	45–49	1550	2180	2500	2690	*2850*	2318	1940
	50–54	1160	1580	1900	2310	*2630*	1976	1797
	55–59	1000	1370	1730	2040	*2170*	1753	1490
	60–64	700	880	1120	1290	*1520*	1106	1035
	65–69	510	630	790	890	*1100*	760	783
	70–74	350	450	570	670	*680*	574	470
	75–79	170	210	270	280	*380*	236	262

[a]Data of Todd (*29*).

accounted for over 80% of all cigarette sales. These last two changes together brought about a 41% reduction in the average amount of tar delivered per cigarette between 1965 and 1973 (*29*). Whether any similar but smaller changes took place previously is uncertain. According to the manufacturers they did not; but this is based on the analysis of only one batch of cigarettes made before 1965.

Whether the changes in mortality can be attributed solely to the changes in cigarette smoking or whether they should be regarded as reflecting also the reduction in the amount of pollution in the atmosphere with coal smoke that began in the 1920s (*8*) and gathered speed after the Clean Air Act of 1956 (*30*) cannot be determined without much more precise evidence than we now have about smoking habits and the type of tobacco that used to be smoked in the first half of the century.

According to Todd, Lee, and Wilson (*31*), the limited evidence that is now available suggests that the male cohorts with the highest "cumulative consumption of constant tar cigarettes" were born 5 or 10 years later than those which experienced the highest age-specific lung cancer mortality rates at all ages between 30 and 59 years of age (Table 17). The evidence is based on a variety of assumptions,

Table 17. Male cohorts with highest cumulative consumption of "constant-tar cigarettes" and highest lung cancer rates.[a]

Age	Highest death rate from lung cancer	Cumulative consumption higher than that of cohort with highest lung cancer rate[b]
30–34	1916	1921
35–39	1916	*1921*, 1926
40–44	1916	*1921*
45–49	1911	*1916*, 1921
50–54	1906	*1911*, *1916*, 1921
55–59	1901	1906, *1911*, 1916

[a]Data of Lawther and Waller (*30*).
[b]Cohorts with highest consumption in italics.

several of which may be incorrect. So far as it goes, however, it supports the belief that atmospheric pollution interacts with cigarette smoking to increase the incidence of the disease.

Conclusion

The ambient atmosphere, particularly in towns, contains a variety of substances that are capable of causing cancer of the lung in men who are exposed to large amounts of them in the course of their occupation. General knowledge of carcinogenesis suggests that at low doses the carcinogenic effect is likely to be linearly proportional to the dose received. It should, therefore, be assumed that the small amounts present in the atmosphere contribute to the causation of some of the cases of lung cancer that occur in clinical practice.

Observations on men indicate that by far the most important cause of lung cancer is cigarette smoking and that other factors characteristic of town air have very little effect, except in so far as some of them may interact with it. Extrapolation from occupational studies suggests that this effect is unlikely to be large. In the absence of cigarette smoking, the combined effects of all such atmospheric agents cannot be responsible for more than about five cases of lung cancer per 100,000 persons per year in European populations.

*"Carcinogen" is used to imply any factor that increases the risk of developing the disease, irrespective of whether it is a "complete carcinogen" or a "promoting agent" that acts only in the company of another agent.

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