Ten-year mortality in a sample of an adult population in relation to air pollution

MICHAŁ KRZYŻANOWSKI AND BOGDAN WOJTYNIAK

From the Department of Medical Statistics, National Institute of Hygiene, Warsaw, Poland

SUMMARY The 10-year mortality in a sample of adult inhabitants of Cracow, Poland, was analysed according to the levels of air pollution in the area of residence. Smoking habit and several social and occupational factors were considered in the analysis, which was carried out with the use of a multivariate method for categorical variables. Among men the main effect of air pollution was marginally significant, but there was a significant interaction between air pollution and smoking. Among women no such relation could be detected. Also, the association between female mortality and smoking was not significant. From other factors considered in the analysis, only exposure at work to dust, high humidity, and variable temperature was related to mortality in both men and women. In addition among women higher mortality was related to a lower level of education.

The effects of air pollution on health in an urban environment continue to be the subject of discussion.^{1,2} Various studies indicate that the possible effect of air pollution on mortality is relatively small and may be influenced by several confounding factors, such as smoking habit and occupational or socioeconomic conditions that also generally affect mortality.^{3–5} One way to assess the effects of air pollution on health, taking into account these confounding factors, is a longitudinal epidemiological study of populations exposed to various levels of air pollution. This is laborious to organise and conduct but affords relatively exact data on individuals who may be followed up for many years. In this paper we present the mortality after 10 years in a random sample of residents of the higher and lower polluted areas of Cracow.

Population and methods

In 1968 a sample of the adult population of Cracow, a city inhabited at that time by 555 000 people, was examined to estimate the prevalence of chronic non-specific respiratory diseases.⁶ The sample was selected at random from permanent residents of Cracow aged from 19 to 70 years by using the register of dwellings as a sampling frame. Standard interviews were obtained from 4355 subjects who formed 94% of the random sample. The interviews included questions on respiratory symptoms, smoking habits, education, place of birth, housing conditions, type

and place of work, and possible exposure to occupational hazards.

Independently of the population study, during 1968–73, measures of ambient air pollution were collected by 20 samplers placed at regular intervals of about 2.5 km in the town area. The devices for continuous measurements were similar to those designed by Warren Spring Laboratory in Britain. The aspirated air passed through a Whatman No 1 filler clump, and bubbled through a fluid to absorb sulphur compounds. The paper filters and bubblers were collected daily except on Sundays and holidays. Concentrations of suspended particulate matter of diameter less than 10 μm were determined by an EEL smoke stain reflectometer and concentrations of SO_2 by a Specol electrophotocolorimeter. With this data we established that in the five-year period the central part of the city had the highest atmospheric pollution; the mean daily concentration of particulate matter was 180 $\mu\text{g}/\text{m}^3$ and of SO_2 114 $\mu\text{g}/\text{m}^3$. In the remaining areas the daily mean for particulate matter was 109 $\mu\text{g}/\text{m}^3$ and for SO_2 53 $\mu\text{g}/\text{m}^3$, and only occasionally did air pollution in these areas exceed those in the centre of the city (figs 1 and 2). There were 658 people living in the centre (15% of the study sample), and we regarded these people as a group exposed to high atmospheric pollution.

In 1978, 10 years after the primary study, we returned to the study population to identify who were alive, who were dead, and if dead what had caused their death.

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Fig 1 Monthly averages of air pollution with suspended particulate matter in the centre (—) and in the remaining area of Cracow (---) during 1968-73.

METHOD OF ANALYSIS

The method of analysis has been described in detail by Freeman, Wojtyniak, and Krzyżanowski (submitted for publication) and is based on that of Grizzle *et al.*⁷ Linear models (similar to those in the analysis of variance) were constructed in which age-adjusted mortality rates were explained by various social and environmental factors. The significance of the effects of particular factors was tested by the statistic Q, which has a χ^2 distribution with degrees of freedom equal to the number of parameters tested simultaneously. Coefficient $\hat{b}$ estimated in the model can be interpreted as the differential effect of the respective category of given factor measured relative to the average mortality level $\hat{m}$.

If the age-standardised mortality rates were computed for every combination of categories of each possible factor the frequency of these combinations would be small; this limits the number of factors that can be analysed simultaneously.

Consequently, we did not estimate one general model including all factors, but tested several models each containing one, two, or three confounding factors in addition to air pollution and smoking, which were the factors in which we were most interested. Table 1 shows the list of factors included in the analysis and the groupings used.

We calculated the probability of death in the 10-year period due to all causes except accidents using the formula:

$$q = \frac{d}{n - w - 0.5a}$$

where q = probability of death

d = number of deaths in 10 years excluding accidents

n = number of persons in the study at 1968

w = number of persons lost to follow-up at 1978

a = number of deaths due to accidents in the 10 years

Fig 2 Monthly averages of air pollution with sulphur dioxide (SO_2) in the centre (—) and in the remaining area of Cracow (---) during 1968–73.

The proportion of deaths due to accidents was 10.9% for men and 3.2% for women; the proportion of the study population lost to follow-up was 1.4% for men and 1.3% for women.

We then calculated age-adjusted mortality rates using indirect standardisation with the total examined group as standard.⁸

Results

We were able to identify 4296 individuals (98.6% of the study population, of whom 308 (7.2%) had died in the 10-year period (table 2). Only 59 (1.4%) of the original population had been lost to follow-up.

The basic linear model of age-standardised mortality on air pollution and smoking habit in men confirmed the highly significant effect of smoking (table 3). The exposure to higher levels of air pollution increased mortality, but this effect was most clearly seen in smokers as the interaction term between air pollution and smoking significantly improved the fit of the model.

We extended this model to consider the influence of place of work and type of work at the time of the interview. The resulting model (table 4) confirmed the influence of increased air pollution on mortality in smokers and showed that non-industrial places of work had a raised risk of mortality, but there was little difference between manual and non-manual

type of work. There was an important interaction between raised air pollution and non-industrial place of work.

Exposures to occupational hazards were also considered in the analysis (table 4), and while the effect of chemicals or irritating gas did not increase mortality, the various other hazards (dust, temperature, or humidity) did have an effect.

When place of birth was included in the analysis the interaction effect of air pollution and smoking diminished, and the model indicated that men born in Cracow and in rural areas are more susceptible than immigrants born in towns outside Cracow to the high air pollution.

Dwelling conditions did not affect mortality in men, nor did level of education.

Since these confounding factors were themselves interrelated we took into the basic model of air pollution and smoking two or three other factors at a time which had been shown to have some effect—place of work, exposure to various occupational hazards, and place of birth. On the basis of this analysis we estimated a final model that included only three factors (table 5). This showed that the effects of smoking and various occupational exposures as well as the effect of air pollution in smokers are significant. The main effect of air pollution itself was just on the border of statistical significance.

Table 1 Categories used to classify the study population

Air pollution	High	A ₁	Residents in 1968 of the defined central area
	Low	A ₀	Resident in 1968 elsewhere in Cracow
Smoking category	Male smokers	S ₁	Current smokers who had smoked at least one cigarette a day in 1968
		S ₀	Non-smokers (never smoked) and ex-smokers—that is, those who had either never smoked regularly or who had given up smoking for at least one month before interview
	Female smokers	S ₁	Smokers (current smokers and ex-smokers)
		S ₀	Never smoked
Place of work	Industrial	W ₁	Place of work—industrial
	Non-industrial	W ₀	Place of work—commercial, administrative, or other
Type of work	Manual	M ₁	Manual workers
	Non-manual	M ₀	Non-manual workers
Occupational hazards	Chemical	CH ₁	Exposed at work to chemicals or irritating gases for at least five years
		CH ₀	Not exposed
	Various	V ₁	Exposed at work to various hazards, such as dusts, variable temperature, or high humidity, to one or more, each for at least five years
		V ₀	Not exposed to any of above
Birth place	Cracow	B ₀	Born in Cracow
	Other town	B _T	Born in another town
	Rural	B _R	Born in a rural area
Dwelling conditions	Crowded	C ₁	Living area less than 10 m ² per inmate
	Less crowded	C ₀	Living area more than 10 m ² per inmate
Education	University	E ₃	Education level over 15 years of school
	High	E ₁	11–14 years of school
	Elementary	E ₀	Education level under 11 years of school

Table 2 Number of men and women in each category in 1968, number died 1968–78, and age-standardised mortality

Category	Men			Women		
	No of cases	No dead	ASM*	No of cases	No dead	ASM
A ₁	255	42	122.0	389	21	35.4
A ₀	1645	141	82.8	2007	104	52.1
S ₁	1258	133	112.4	711	40	60.3
S ₀	642	50	61.8	1685	85	44.3
W ₁	825	71	86.9	767	29	51.5
W ₀	999	110	96.6	1263	61	47.0
M ₁	1066	123	100.2	1039	61	48.4
M ₀	758	58	81.0	991	29	36.9
CH ₁	439	42	91.6	223	10	48.8
CH ₀	1461	141	91.6	2173	115	48.3
V ₁	769	106	109.9	503	40	64.4
V ₀	1131	77	73.8	1893	85	43.2
B ₀	707	69	110.1	875	39	46.8
B _T	394	40	78.3	579	33	44.0
B _R	799	74	87.1	942	53	52.7
C ₁	969	79	91.3	1148	53	54.5
C ₀	931	104	92.0	1248	72	44.7
E ₃	288	21	82.9	196	2	14.5
E ₁	766	61	88.0	940	31	40.3
E ₀	846	101	96.2	1260	92	55.0
Total No of cases analysed	1900	183	91.7	2396	125	48.3
Cases lost to follow-up	25	—	—	34	—	—

*ASM = Age-standardised mortality per 1000 population.

Table 3 Analysis of variation of age-standardised mortality rates in men: basic model

Source of variation	Hypothesis	p-value	Estimates*	
			b	SE _b
Air pollution	A ₁ = 0	0.077	14.92	8.42
Smoking	S ₁ = 0	0.000	37.20	8.42
Air pollution in smokers	A ₁ × S ₁ = 0	0.032	18.07	8.42
Model	A ₁ = S ₁ = A ₁ × S ₁ = 0	0.000	—	—
Goodness of fit — perfect — saturated model				

*Estimated overall mean $\bar{m} = 96.95$, $SE_{\bar{m}} = 8.42$; deaths per 1000 population.

In a similar analysis for women the definition of the smoking categories differed; for women ex-smokers were joined with the current smokers to form a group of “ever smoked” in comparison to “never smoked.” In women the basic linear model comprising two factors, air pollution and smoking, was not significant, although it fitted the data satisfactorily (table 6). Smoking had no effect and air pollution was on the border of significance, $p = 0.053$, but the higher mortality was in the less polluted areas. This strange effect became even more pronounced when

Table 4 Results of fitting various models to age-standardised mortality rates in men

Model*	Effects	b†	p‡	Goodness of fit
Ma (place and type of work)	A ₁	10.79	0.151	Q _c = 4.79 df = 9 p = 0.852
	S ₁	39.98	0.000	
	A ₁ × S ₁	21.95	0.003	
	W ₀	15.91	0.020	
	A ₁ × W ₀	13.15	0.061	
	M ₁	7.75	0.187	
Mb (occupational exposures)	A ₁	10.90	0.147	Q _c = 12.62 df = 10 p = 0.245
	S ₁	29.25	0.000	
	A ₁ × S ₁	13.54	0.071	
	V ₁	17.02	0.008	
	CH ₁	-7.45	0.291	
Mc (place of birth)	A ₁	8.48	0.278	Q _c = 3.73 df = 4 p = 0.444
	S ₁	32.60	0.000	
	A ₁ × S ₁	13.33	0.094	
	B _C	20.16	0.073	
	B _T	-29.57	0.007	
	A ₁ × B _C	13.66	0.245	
	A ₁ × B _T	-33.15	0.003	

*Confounding factors in parentheses.

†Deaths per 1000 population.

‡Probability of Q_c for the given effect (H₀: b = 0).

Table 5 Analysis of variation of age-standardised mortality rates in men: final model

Source of variation	Hypothesis	p-value	Estimates*	
			b	SEb
Air pollution	A ₁ = 0	0.051	15.79	8.09
Smoking habit	S ₁ = 0	0.000	34.96	8.31
Air pollution in smokers	A ₁ × S ₁ = 0	0.029	17.74	8.12
Various occupational exposures	V ₁ = 0	0.023	13.94	6.13
Model	A ₁ = S ₁ = A ₁ × S ₁ = V ₁ = 0	0.000	—	—
Goodness of fit		0.785	—	—

*Estimated overall mean $\bar{m}$ = 98.17, SE $\bar{m}$ = 8.16; deaths per 1000 population.

Table 6 Analysis of variation of age-standardised mortality rates in women: basic model

Source of variation	Hypothesis	p-value	Estimates*	
			b	SEb
Air pollution	A ₁ = 0	0.053	-8.49	4.38
Smoking habit	S ₁ = 0	0.168	6.59	4.79
Model	A ₁ = S ₁ = 0	0.079	—	—
Goodness of fit		0.152	—	—

*Estimated overall mean $\bar{m}$ = 46.38, SE $\bar{m}$ = 4.99; deaths per 1000 population.

the occupational exposures were considered; the mortality of women exposed to various hazards (dust, temperature, and humidity) was higher than those not exposed, whereas exposure to chemicals or gases reduced mortality (table 7).

Introducing education into the model had a significant effect in the expected direction—that

Table 7 Results of fitting various models to age-standardised mortality rates in women

Model*	Effects	b†	p‡	Goodness of fit
Wa (occupational exposures)	A ₁	-10.66	0.006	Q _c = 14.64 df = 9 p = 0.101
	S ₁	0.69	0.852	
	V ₁	16.99	0.000	
	CH ₁	-19.59	0.000	
Wb (education)	A ₁	-5.22	0.204	Q _c = 7.80 df = 7 p = 0.350
	S ₁	4.68	0.185	
	E ₀	24.11	0.000	
	E ₁	8.77	0.077	
Wc (place of birth)	A ₁	-6.32	0.181	Q _c = 5.65 df = 5 p = 0.342
	S ₁	4.44	0.315	
	B _C	7.68	0.311	
	B _T	-10.12	0.086	
	A ₁ × B _C	14.74	0.051	
	A ₁ × B _T	-10.98	0.063	

*Confounding factors in parenthesis.

†Deaths per 1000 population.

‡Probability of Q_c for the given effect (H₀: b = 0).

Table 8 Analysis of variation of age-standardised mortality rates in women: final model

Source of variation	Hypothesis	p-value	Estimates*	
			b	SEb
Air pollution	A ₁ = 0	0.024	-8.94	3.97
Smoking habit	S ₁ = 0	0.154	6.23	4.37
Education	E ₀ = E ₁ = 0	0.001	—	—
Elementary	E ₀ = 0	0.000	19.38	5.50
High	E ₁ = 0	0.093	9.67	5.77
Various occupational exposures	V ₁ = 0	0.049	9.43	4.80
Model	A ₁ = S ₁ = E ₀ = E ₁ = V ₁ = 0	0.000	—	—
Goodness of fit		0.564	—	—

*Estimated overall mean $\bar{m}$ = 33.57, SE $\bar{m}$ = 5.83; deaths per 1000 population.

women with higher education have lower mortality. Of the other confounding factors, only the place of birth modifies to some extent the relation between air pollution and mortality in that natives of Cracow have a somewhat increased mortality in the polluted central area. The opposite effect is seen in migrants born in other towns.

It was not possible to proceed to a model for women that considered simultaneously the two main factors and three other confounding factors, as the number of deaths was too few to construct the many cells of the contingency table. We therefore constructed two separate models with two additional confounding factors in each—educational level with one type of occupational exposure at a time.

In the model considering air pollution, smoking, education, and exposure to chemicals or irritating gases no regular pattern of mortality rates could be detected, and models with first-order interactions were not fitted. When the various hazards were substituted for the chemical hazards, more regular

patterns could be observed (table 8). There was increased mortality in women living in the areas of lower air pollution, in those with less education, and in those exposed to various hazards,

Discussion

In most studies of long-term effects of air pollution on mortality the authors correlated the mortality rates in different territories with the characteristics of air pollution in these areas.⁹⁻¹¹ The main problems in such studies are connected with a measurement of confounding factors, and the only values it is possible to use in the analysis are usually crude characteristics of the area. In a prospective epidemiological study information on every individual is available, but the method of data collection limits the number of people who can be examined, which diminishes the validity of observed death rates. Relatively small numbers of deaths in the follow-up period and qualitative data on confounding factors also restrict the number of factors that can be considered simultaneously in the analysis.

Nevertheless, the data used in our study enabled us to detect some associations that are difficult to find in an analysis based on aggregated data. One is the interaction effect of air pollution and cigarette smoking observed in men, indicating that men who smoke have a higher susceptibility to pollutants. This finding differs from the conclusion of Morris *et al.*¹² who could not show more than an additive effect of air pollution and smoking on 13 years' mortality.

We checked if the interaction observed in our data did not result from differences in the habit of residents of the more and less polluted areas who smoked. The distributions of duration of smoking and of the amount of cigarettes smoked daily, however, were similar in both groups.

For women the results of our study are similar to the observations of Morris¹²: in both studies there was no excess mortality in women residing in more polluted areas. Opposite results have been reported by Brozzo *et al.*,¹³ who had found an association between female mortality and the level of air pollution. The strength of that correlation, however, had been 2.5 times smaller than that observed for men.

The lack of interaction between smoking and air pollution in women may be partly explained by the much smaller effect of smoking on mortality in women than in men observed in our study as well as in some previous ones.¹⁴ This weak influence of the habit may be a reason why air pollution has no effect even in a smoking group.

The relatively small number of deaths in the group followed up for 10 years severely restricted the

possibility of cause-specific analysis. For men, however, we could detect that the greatest excess of mortality in the residents of the highly polluted area was due to deaths caused by malignant neoplasms of the respiratory system, arteriosclerosis, pulmonary heart disease, and chronic bronchitis, emphysema, and asthma. This only partly supports the findings of Brozzo *et al.*¹³ The higher overall mortality in women residing in areas with lower air pollution was due to diseases of the circulatory system.

We could not identify which of the two types of air pollution we considered (suspended particulates or SO₂) is responsible for the excess of mortality in men living in the more polluted area, since the levels of both pollutants were strongly correlated.

Among confounding factors only exposure to various occupational hazards appeared to be significantly related to mortality in men and women. All three types of hazards (dust, variable temperature, and high humidity) have been strongly intercorrelated, so it was impossible to distinguish the specific effect of one of them. In general, their presence probably describes burdensome conditions at work. In contrast, mortality is not related to exposure to chemicals and irritating gases at work. Perhaps this may be due to the selection of healthier workers and to some preventive measures taken when severe hazards are known to be present. Similar selection may be related in part to the somewhat lower mortality in the industrial employees than in other workers. Such effect of selection of workers has been noticed also in other studies.¹⁵

The relation of socioeconomic conditions to mortality has also been observed.¹⁶⁻¹⁸ In our analysis these factors have been represented by the type of occupation, dwelling conditions, and the level of education. Only the latter variable is clearly related to mortality in Cracow and only in women.

We could not draw any definite conclusions concerning the relation between mortality and the place of birth, nor its influence on the relation between mortality and air pollution. These problems need further study in a larger sample or in a sample observed for a longer time.

As the final result of the analysis we may state that the raised mortality observed in men living in the more polluted part of Cracow, which is especially pronounced in smokers, is additional to the effects of smoking habit, social conditions, or occupational factors. The real effect of air pollution may be even stronger than that observed in the study, since the residents of the higher polluted area could spend part of the day in the less polluted one and vice versa.

It is difficult to explain the inverse relation observed in women, despite the smaller reaction of women to smoking. The question is: Are the above

observations the result of influence of some uncontrolled factors, changing the real relationship between air pollution and mortality in women, or do they reflect the smaller susceptibility of women to this type of environmental factor. To answer these questions further studies are necessary, carried out in other populations of men and women.

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