

Ventilatory Lung Function and Chronic Chest Symptoms among the Inhabitants of Urban Areas with Various Levels of Acid Aerosols: Prospective Study in Cracow

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The analysis carried out earlier in Cracow showed that the high level of SO₂ and particulate matter (PM) alone cannot be responsible for an excess of chronic chest symptoms and faster lung function deterioration in the population at large. To check the hypothesis that acid aerosols present in the urban air may cause substantial damage of the lungs, data from a 13-year follow-up survey of chronic chest diseases in Cracow (1968-1981) have been reanalyzed. In the plan of the analysis, three areas of the city with various levels of sulfate and sulfur transformation ratio (STR) in the urban air have been defined. In each of the defined areas, the prevalence of chronic chest symptoms, as well as lung function decline, have been studied. In total, the lung function study group consisted of 1414 persons (584 males and 830 females). Those men who lived in the area with the higher sulfate and STR had lower FEV₁ levels by about 151 mL than did the residents of the other areas, and this was equivalent to the effect of smoking. In females, the pattern was generally the same. In men, the FEV₁ decline rate (mL/year) over the 13-year period was significantly faster by about 11 mL/year in the areas with higher and intermediate STR, which was again equivalent to the effect of smoking. It was found that the level of SO₂ and PM in the urban air correlated with the symptom prevalence in women; however, it had no clear impact on lung function deterioration.

Introduction

The effect of atmospheric air pollution on chronic respiratory diseases has been studied in numerous epidemiological surveys (1-5). Extensive cross-sectional studies showed a gradation of symptoms across the levels of pollution; however, as pollution was measured in different ways in each of the studies, it is difficult to deduce any quantitative relationships with sulfur dioxide (SO₂) and particulate matter (PM). Some of the studies indicated that increased cough and phlegm production were associated with air pollution but decreased lung function was not.

The impact of SO₂ and PM in the urban air on chronic respiratory problems has been studied earlier in Cracow, and the prevalence of chronic chest symp-

toms persistent over 5 years (1968-1973) was related to the air pollutants (5,6). The air pollution level in Cracow (SO₂ and PM) was then monitored daily by a network of 20 sampling stations covering the entire area of the city. The data on air pollution enabled us to select the part of the city having the highest air pollution level during the study period. This proved to be in the city center, where the mean daily concentration of PM was 180 µg/m³ and of SO₂ was 114 µg/m³. In the remaining areas the daily mean concentration of PM was 109 µg/m³ and of SO₂ was 53 µg/m³. Only occasionally did the air pollution in these areas exceed that in the city center. The findings of this study in Cracow showed that air pollution increased the prevalence of persistent chest symptoms and reduced FEV₁ level only in persons who were exposed to occupational hazards. The results showed that even the high level of SO₂ and PM alone cannot be responsible for significant excess in chest symptoms and lung function deterioration and, therefore, other pollutants should be taken into consideration.

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The association shown in Japan between sulfuric acid (H_2SO_4) and respiratory morbidity (7) gives support to the hypothesis that acid aerosol is a very harmful component of urban air pollution. This is consistent with the results of mortality in major cities in the U.S. (8) that indicate that sulfates are better predictors of mortality than any of the gravimetric indices that have been used previously. To check this intriguing hypothesis, the data from the 13-year follow-up survey of chronic chest diseases in Cracow (1968–1981) have been reanalyzed. In the plan of the analysis, three areas of the city with various levels of sulfate and sulfur transformation ratio in the urban air have been defined. In each of the areas, the prevalence of chronic chest symptoms and lung function decline rate in the study sample have been measured over the study period.

Materials and Methods

The study area covers the city of Cracow in which about 740,000 inhabitants live. The town is situated in the valley of the Vistula River where long inversion phenomena occur, as well as a great number of foggy and misty days (up to 150 days during a year). The urban area, especially the city center, is characterized by little wind and frequent periods of stillness. The rapidly growing industry and a great number of chimneys of houses with insufficient central heating until recent times are the main sources of air pollution in Cracow.

The spatial distribution of the daily mean level of SO_2 shows that the highest concentrations over the whole year are present in the central area of the city. Mean daily levels of SO_2 concentrations in the cold months are two or even three times higher than in summertime. About 50% of SO_2 measurements in the city center are in the range of 151 to 350 $\mu\text{g}/\text{m}^3$. The spatial distribution of PM less than 20 μm is similar to that of SO_2 . In the cold months (October–March) the dust concentrations are three to four times higher than in months with mild weather (April–September), and the frequency of the measurements above 200 $\mu\text{g}/\text{m}^3$ is very high, especially in the city center (Table 1).

Annual mean concentration of SO_4^{2-} in the Cracow

region is 19.2 $\mu\text{g}/\text{m}^3$. The highest values (23.3 $\mu\text{g}/\text{m}^3$) have been found in the eastern part of the region around the metallurgical complex. In order to assess transformation rates of SO_2 to sulfite (SO_3) in the atmosphere, the relative content of sulfate sulfur versus total sulfur has been measured. It appeared that in the center of the city this ratio was 10%; however, in the area around the metallurgical complex, it was found to be 40%. It is supposed that the faster transformation process of SO_2 could have been caused by oxidation processes on catalytic particulate matter aerosols emitted by the metallurgical complex and cement factory situated in the eastern part of the city (9).

On the basis of measurements of SO_2 , PM, and sulfur transformation ratio (STR), the three pollution areas have been defined: area A, eastern part of the city, around the metallurgical complex, with lower SO_2 and PM level, but high STR (40%); area B, western part of the region, with intermediate levels of SO_2 , PM, and STR (15%); and area C, central part of the city, including the old city center, with very high SO_2 and PM level, but low STR (10%).

The data on health status of the Cracow inhabitants were derived from the Cracow 13-year prospective study on chronic obstructive lung disease (10). The first survey was completed in 1968, the second survey in 1973, and the third in 1981. In the first survey, the standardized interviews and spirometric testing were performed in 3047 persons, i.e., in 70% of the random sample of Cracow adult residents. The whole group that completed the spirometric testing and interviews in all three surveys had, in total, 1747 persons. The analysis presented deals only with those subjects who at least over the last 8 years of the follow-up (since 1973) did not change their residence area. In total, this study group consisted of 2357 persons (951 males and 1406 females); however, only 1414 persons (584 males and 830 females) completed all three spirometric tests.

To estimate the eventual selection bias, the characteristics of the group under study were compared with those of the dropouts. There was an excess in the prevalence of wheezing and attacks of breathlessness in area C men who were interviewed in 1968. This could eventually blur the differences across the city areas. An excess in chronic cough and phlegm between the study group and the dropouts known to be alive was about the same size in every city area, and it should have no

Table 1. Mean daily concentrations of particulate matter and SO_2 in various areas of Cracow.^a

Mean daily concentration, $\mu\text{g}/\text{m}^3$	Winter season	Nonwinter season	Annual
Particulate matter			
Area A	110	44	72
Area B	135	46	82
Area C	177	58	110
SO_2			
Area A	103	61	79
Area B	124	59	85
Area C	135	63	94

^aBased on data from 1976 to 1986.

impact on the conclusions. In females, there was a bigger excess in the prevalence of wheezing, attacks of breathlessness, and chronic cough in area B than in area A, and this might inflate the difference in the frequency of symptoms observed.

Comparing FEV₁ level in 1968 (standardized by age and height) between those who were included in the study sample and those who were not followed in the various city areas, one can see that the differences across the areas were not statistically different between examined and dropouts known to be alive (Table 2). Hence, the selection factor could not bias the lung function results.

The assessment of the health status of the population sample included standardized interviews and spirometric measurements (11). The questionnaire was based on the British Medical Research Council questionnaire for studies of chronic, nonspecific respiratory diseases. The spirometric testing was performed with the Vitalograph nondigital wedge spirometer. On all three occasions examined persons repeated the maximal forced expiration effort five times, and the highest tracing to estimate FEV₁ was chosen and later corrected to body temperature pressure saturated (BTPS).

Chronic cough or chronic phlegm was diagnosed in those persons who reported the symptoms usually during day or night at least over 3 consecutive months. The diagnosis of chronic wheezing has been made if the symptoms occurred on most days independent of cold or chest infections. Dyspnea on effort was diagnosed in respondents who reported that they were troubled by shortness of breath when climbing up the stairs and had to rest at the first or second floor. Also, persons who confirmed that they were short of breath when walking fast or walking up a slight hill were inserted in this category. Attacks of breathlessness were diagnosed in the case when respondents reported that they had attacks of shortness of breath independent

of effort. To analyze the relationship between the symptom prevalence and area of residence, log-linear models were used with age and smoking habit as possible confounders.

The rate of FEV₁ change expressed in milliliters per year was estimated for each person in the 13-year follow-up period (1968–1981), as well as in the 8-year period (1973–1981). The analysis was carried out separately for men and women. Along with the dummy variable indicating the residence area, age, smoking habit, and mean FEV₁ level were included as confounders in the multiple linear regression of FEV₁.

Results

In males the prevalence of chronic chest symptoms in the end point survey (Table 3) was not in fact different across the city areas, but in females there was an excess in prevalence of chronic phlegm, chronic cough, and dyspnea on effort among those persons who lived in area C (high SO₂ and PM). Odds ratio (OR) standardized to age and smoking habit by the log-linear model confirmed the statistically significant excess in risk for chronic cough, chronic phlegm, and wheezing only in females.

FEV₁ level in 1981 was slightly lower in females who lived in the city center (area C); however, FEV₁ decline in the 13-year follow-up (1968–1981) expressed in percentage of the mean level was significantly faster in both sex groups that lived around the metallurgical complex (Table 4). Mean age differed significantly among residents of various areas and that was taken into consideration in the following analysis.

In Tables 5 and 6 the data from the linear regression analysis of FEV₁ level (1981 survey) on age, height, smoking, education, and area of residence are presented. One can see that the FEV₁ level in males depended not only on age, height, smoking, and education, but also on the residence area. Those who lived

Table 2. Lung function level in 1968 among those who were included in the study sample and in dropouts by city areas.^a

Subjects	n			%			FEV ₁ , L ^b		
	A	B	C	A	B	C	A	B	C
Males									
Study sample	181	141	262	57.6	33.1	43.0	3.97	4.09	3.97
Dropouts, alive	105	203	235	33.4	47.7	38.5	4.13	4.04	3.97
Dropouts, died (1968–1981)	28	82	113	8.9	19.2	18.5	3.93	3.47	3.56
Total (spirometric testing in 1968)	314	426	610	100.0	100.0	100.0	4.02 (0.3) ^c	3.95 (0.3)	3.89 (0.3)
Females									
Study sample	217	199	414	58.0	37.8	52.2	3.32	3.24	3.24
Dropouts, alive	142	285	305	38.0	54.2	38.3	3.29	3.26	3.29
Dropouts, died (1968–1981)	15	42	75	4.0	8.0	9.5	2.81	2.73	2.65
Total (spirometric testing in 1968)	374	526	793	100.0	100.0	100.0	3.29 (0.2)	3.21 (0.2)	3.21 (0.2)

^aStudy sample subjects completed three spirometric tests and did not change residency since 1973.

^bStandardized by age and height.

^cNumbers in parentheses are SEs of the mean.

in area A had a lower FEV₁ level by about 151 mL than the residents of the city center (area C), and this was equivalent to the effect of smoking. In females, the pattern was generally the same, i.e., the effect of living in the neighborhood of the metallurgical complex on

FEV₁ level was negative, though slightly weaker than that observed in men.

In men, the mean FEV₁ decline rate (mL/year) over the 13-year period was significantly faster by about 11 mL/year in the areas with high and intermediate

Table 3. Chronic chest symptoms reported in end point survey and odds ratios estimated by log-linear models fitted to age, smoking, and residency.^a

City area	n	Dyspnea on effort		Wheezing		Attacks of dyspnea		Chronic cough		Chronic phlegm	
		%	OR ^b	%	OR	%	OR	%	OR	%	OR
Males (n = 951)											
Area A	283	19.8	1.22	9.9	0.99	10.6	1.06	37.1	1.20	40.3	1.09
Area B	222	17.6	0.87	9.0	0.90	8.6	0.85	29.3	0.86	38.7	1.00
Area C	446	18.6	0.93	10.8	1.10	10.8	1.10	31.4	0.97	36.8	0.92
p (χ ² for symptoms/area)		0.8	0.30	0.7	0.7	0.6	0.6	0.10	0.20	0.6	0.5
Females (n = 1406)											
Area A	348	33.3	0.90	5.7	0.67	8.6	0.79	14.7	0.73	14.4	0.68
Area B	303	40.6	1.01	10.6	1.36	12.5	1.22	22.8	1.32	22.8	1.24
Area C	755	42.4	1.09	8.6	1.08	10.9	1.46	18.7	1.03	21.7	1.17
p (χ ² for symptoms/area)		0.02	0.30	0.08	0.05	0.20	0.20	0.03	0.02	0.01	0.01

^aAmong those who did not change residence area since 1973.

^bOdds ratio.

Table 4. Mean FEV₁ level, FEV₁ changes, mean age, and smoking habit by residency area.^a

City area	n	FEV ₁ , 1981	% FEV ₁ , 1968-1981	% FEV ₁ , 1973-1981	Age, 1981	Permanent smokers, %
Males						
Area A	181	3.15	25.7	15.5	53.1	48.6
Area B	141	3.22	24.4	14.7	55.3	46.1
Area C	262	3.16	20.5	12.2	56.1	43.5
Total	584	3.17*	23.1‡	13.9*	55.0†	45.7*
Females						
Area A	217	2.39	24.3	13.3	49.7	14.8
Area B	199	2.25	22.6	14.0	54.6	20.1
Area C	414	2.26	20.6	13.4	56.0	17.4
Total	830	2.29†	22.0†	13.5*	54.0‡	17.3*

^aLung function tested three times in subjects who did not change residency since 1973.

†p < 0.05.

‡p < 0.01.

*Nonsignificant, p > 0.10.

Table 5. Linear regression of FEV₁ level among persons who did not change residency since 1973—males.

Independent variables	FEV ₁ , 1981		t
	Regression coefficient	SE of regression coefficient	
Area A ^a	-150.9	63.9	2.36
Area B ^a	-36.0	68.7	0.52
Age, years, 1981	-50.0	2.6	18.98
Height, m	36.0	4.6	7.81
Permanent smokers			
< 15 cigarettes	-172.3	92.6	1.86
≥ 15 cigarettes	-257.7	67.1	3.84
Exsmokers	-144.4	77.4	1.87
Education	-138.8	56.5	3.84
Constant		41.0	
R ² , %		47.66	

^aVersus area C = 0.

STR, and this was again equivalent to the effect of smoking (Table 7). No interaction was found between smoking and the quality of air in the residence area. As regards the FEV₁ decline rate over an 8-year period (1973–1981), the effect of living in an area with high or moderate STR was similar to that for the 13-year period. In women, the FEV₁ decline rate estimated over the 13-year period (1968–1981) in area A was about the same as in men, but in area B it was slightly slower (Table 8). In area A, the effect of residency around the metallurgical complex was even more pronounced

than that of smoking. For the FEV₁ decline rate in women over the 8-year period (1973–1981), the effects of residency and smoking turned out to be rather small and insignificant.

Discussion

The main message that comes from the analysis presented is the conclusion that the high level of SO₂ and PM correlated with the symptom prevalence in women; however, it had no impact on lung function

Table 6. Linear regression of FEV₁ level among persons who did not change residency since 1973—females.

Independent variables	FEV ₁ , 1981		
	<i>b</i>	SE _{<i>b</i>}	<i>t</i>
Area A ^a	- 88.8	41.2	2.16
Area B ^a	- 64.1	41.1	1.56
Age, years, 1981	- 36.2	1.5	24.61
Height, m	0.1	0.6	0.22
Permanent smokers			
< 15 cigarettes	-155.3	63.1	1.83
≥ 15 cigarettes	- 82.3	56.4	1.46
Exsmokers	-127.4	71.3	1.79
Education	-125.5	34.4	3.65
Constant		4353.95	
R ² , %		46.62	

^aVersus area C = 0.

Table 7. Linear regression of FEV₁ decline rate among males who did not change residency since 1973.

Independent variables	FEV ₁ , mL/year (<i>n</i> = 584)					
	1968–1981			1973–1981		
	<i>b</i>	SE _{<i>b</i>}	<i>t</i>	<i>b</i>	SE _{<i>b</i>}	<i>t</i>
Area A ^a	11.7	3.7	3.20	11.1	6.0	1.85
Area B ^a	11.9	4.0	2.99	12.6	6.5	1.95
Height, m	159.8	28.7	5.55	141.4	46.3	3.05
Mean FEV ₁	-10.6	2.1	5.05	-14.3	3.4	4.22
Permanent smokers						
< 15 cigarettes	14.0	5.3	2.62	5.0	8.7	0.50
≥ 15 cigarettes	8.4	3.8	2.23	10.0	6.1	1.63
Exsmokers	14.6	4.5	3.26	7.6	7.3	1.03
Constant		-190.2			-152.11	
R ² , %		10.5			4.6	

^aVersus area C = 0.

Table 8. Linear regression of FEV₁ decline rate among females who did not change residency since 1973.

Independent variables	FEV ₁ , mL/year (<i>n</i> = 830)					
	1968–1981			1973–1981		
	<i>b</i>	SE _{<i>b</i>}	<i>t</i>	<i>b</i>	SE _{<i>b</i>}	<i>t</i>
Area A ^a	11.8	2.4	4.96	2.43	3.9	0.62
Area B ^a	3.9	2.4	1.60	4.30	4.0	0.83
Height, m	6.5	3.3	1.98	11.2	5.4	2.06
Mean FEV ₁	-5.8	1.7	3.51	-4.2	2.7	1.58
Permanent smokers						
< 15 cigarettes	6.1	3.7	1.63	10.0	6.1	1.62
≥ 15 cigarettes	8.7	3.3	2.62	-4.5	5.5	0.83
Exsmokers	0.8	4.2	0.19	-0.1	6.9	0.10
Constant		39.1			28.7	
R ² , %		4.8			1.3	

^aVersus area C = 0.

deterioration over time. We found that FEV₁ decline rates over the follow-up were much faster in both sex groups in those persons who lived in area A. We assume that the effect of SO₂ and PM on lung function must have been negligible, as in area A rather low levels and in area C rather high levels of these pollutants were observed. The study supports the hypothesis that pollutants other than SO₂ or PM presumably present in the urban air are responsible for lung function damage. It suggests that acid aerosols may be the cause of the observed lung disorders because FEV₁ decline was faster in the area with higher sulfate content and STR. The high sulfur transformation ratio is considered to be an indirect measure of the oxidation processes of SO₂ in the atmosphere, which in certain circumstances may lead to the appearance of acid aerosols and H₂SO₄ (9). Atmospheric H₂SO₄ is a secondary contaminant arising from the chemical transformation of gaseous SO₂.

The measured SO₄²⁻ includes strong acids (H₂SO₄ and NH₄HSO₄), as well as the fully neutralized salt, (NH₄)₂SO₄. Because the H⁺/SO₄²⁻ ratio is highly variable in time and location and is often close to zero, we have to acknowledge that SO₄²⁻ is a rather poor surrogate for acid aerosols concentration. The opinion that SO₄²⁻ is a better indicator of the active component of fine particles (FP) than is PM, or total suspended particles (TSP) does not necessarily make it an accurate indicator of acid aerosols but does lend support to the hypothesis that H⁺ may be the active agent (12-14). Unfortunately, in our epidemiological study, we had no possibility to relate the health status of the population to acidity (i.e., H⁺ ion concentration of respirable particles).

Our findings fit well with the data of the cross-sectional mortality study in U.S. (1), where predictors of mortality due to air pollution were expressed in terms of four aerosol pollutant surrogates, i.e., TSP, inhalable particles < 15 μm (IP), FP < 2.5 μm, and SO₄²⁻. In order of magnitude of coefficient and level of significance between mortality rate and pollutant surrogate, only FP and SO₄²⁻ had statistical significance as predictors of response.

In our study we found that the presumed chronic effect of higher sulfate content and higher STR in the urban air on lung function was rather strong and comparable with the detrimental consequences of smoking. As no interaction was found between smoking and the air pollutants, we can assume that both factors develop their harmful effects in the lung tissue independently. It was evident from the additional analysis that the effect of sulfate pollutants and STR could not be confounded by occupational hazards.

The excess in the prevalence of chronic chest symptoms in persons who lived in the areas with high SO₂ and PM but with lower sulfate content may be explained by the fact that SO₂ is mainly absorbed in the nose and upper respiratory tract, and only minimal amounts reach the lower respiratory tract. Also, in the upper respiratory tract, most of total suspended par-

ticulate matter is deposited, and this part of lung airways react to environmental insults with chronic chest symptoms (cough, phlegm, etc). The more pronounced symptoms in women may be caused by the fact that women tend to spend much more of their time than men in the residence area.

The strong effect of acid aerosols on the lung function may result from the deposition pattern in the respiratory tract. It is supposed that H₂SO₄ and (NH₄)₂SO₄ aerosols are deposited largely in the small airways. The deposition pattern within the respiratory tract is dependent on the size distribution of the ambient droplets and humidity (12,15). Acidic ambient aerosols typically have a mean median aerodynamic diameter of 0.3 to 0.6 μm. Thus, even with hygroscopic growth in diameter in the respiratory airways, acid aerosols remain within the fine particle range and deposit preferentially in the distal lung airways and airspaces. It is possible that sulfate salts can promote release of histamine or other mediators of bronchoconstriction in the distal airways.

Certainly, the clearance of sulfuric acid, bisulfite, sulfite, and sulfate from the lung is influenced by the cations present in the aerosols inhaled simultaneously. Because urban air is such a complex mixture of these aerosols, the assessment of harmful effects of ambient aerosols is very difficult. Metal sulfates may alter mucociliary clearance, which is responsible for clearing the lung of particles and responsible for host defense ability. Therefore, an accurate estimate of the toxicity of complex aerosols occurring in urban air based on its sulfate contents or STR may not be possible, since the metallic ions often associated with them may also be harmful and we do not know to what extent they could change the health response at the population level. Hence, further studies in this direction should be taken into careful consideration.

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REFERENCES

1. Lawther, P. J., Waller, R. E., and Henderson, M. Air pollution and exacerbations of bronchitis. *Thorax* 25: 525-539 (1970).
2. Lunn, J. E., Knowelden, J., and Handyside, A. J. Patterns of respiratory illness in Sheffield infant schoolchildren. *Br. J. Prev. Soc. Med.* 21: 7-16 (1967).
3. Pollution Atmosphérique et Affections Respiratoires Chroniques—Groupe Coopératif. Pollution atmosphérique et affections respiratoires chroniques ou à répétition. *Bull. Eur. Physiopathol. Resp.* 18: 87-116 (1982).
4. Ware, J. H., Thibodeau, L. A., Speizer, F. E., Colome, S., and Ferris, B. G., Jr. Assessment of the health effects of atmospheric sulfur oxides and particulate matter: Evidence from observational studies. *Environ. Health Perspect.* 41: 255-276 (1981).
5. Wojtyniak, B., Krzyzanowski, M., and Jedrychowski, W. Importance of urban air pollution in chronic respiratory problems. *Z. Erkr. Atmungsorgane* 163: 274-284 (1984).
6. Jedrychowski, W., Krzyzanowski, M., and Wojtyniak, B. Con-

- fronting the effects of smoking and air quality on the development of chronic respiratory diseases. *Tokai J. Exp. Clin. Med.* 10 (4): 323-330 (1985).
7. Kitagawa, T. Cause analysis of the Yokkaichi asthma episode in Japan. *J. Air Pollut. Control Assoc.* 34: 743-746 (1984).
 8. Evans, J. S., Tosteson, T., and Kinney, P. L. Cross-sectional mortality studies and air pollution risk assessment. *Environ. Int.* 10: 55-83 (1984).
 9. Kasina, S. Processes of transport, transformation and removal of sulfur compounds from the atmosphere. *Panst. Zakl. Graf.* (in Polish) (1981).
 10. Krzyzanowski, M., Jedrychowski, W., and Wysocki, M. Factors associated with the change in ventilatory function and the development of chronic obstructive pulmonary disease in a 13-year follow-up of the Cracow study. *Am. Rev. Respir. Dis.* 134: 1011-1019 (1986).
 11. Sawicki, F., and Lawrence, Ph. S., Eds. Chronic nonspecific respiratory diseases in the city of Cracow. National Institute of Hygiene, Warsaw, 1977.
 12. Lippmann, M., Schlesinger, R. B., Leikauf, G., Spektor, D., and Albert, R. E. Effects of sulphuric acid aerosols on respiratory tract airways. *Ann. Occup. Hyg.* 26: 667-690 (1982).
 13. Schlesinger, R. B. Comparative irritant potency of inhaled sulfate aerosols—effects on bronchial mucociliary clearance. *Environ. Res.* 34: 268-279 (1984).
 14. Schlesinger, R. B., Naumann, B. D., and Chen, L. C. Physiological and histological alterations in the bronchial mucociliary clearance system of rabbits following intermittent oral or nasal inhalation of sulfuric acid mist. *J. Toxicol. Environ. Hlth.* 12: 441-465 (1983).
 15. Lippmann, M., Yeates, D. B., and Albert, R. E. Deposition, retention, and clearance of inhaled particles. *Br. J. Ind. Med.* 37: 337-362 (1980).