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CONSISTENCY OF REPORTED HEALTH EFFECTS
OF AIR POLLUTION

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Abstract:

Three different studies have considered the relation between various mortality rates and air pollution levels. This paper evaluates the consistency of these studies and finds some evidence of a constant proportional effect on both males and females. Two of the studies indicate a relation between air pollution and total mortality rate but differ in the estimate of the effect by a factor of fifteen. The studies generally find little relation between pollution and lung diseases and lung cancer; they disagree on heart disease and total cancer findings.

I. Introduction

Concern about the harmful health effects of air pollution has produced a set of papers studying the relations between pollution levels and mortality rates. This paper surveys the results of three sets of authors, gives some reanalysis of previously published results, and evaluates the entire group of studies for consistency. The Lave and Seskin study^{1,2} uses regression analysis to determine whether the mortality rate of a Standard Metropolitan Statistical Area (SMSA) can be predicted by the pollution levels at one sampling station in the SMSA. The Winkelstein analysis of mortality in Buffalo³⁻⁸ and the Nashville study⁹⁻¹² group census tracts

with similar pollution levels, as measured by a group of sampling stations, and similar median family income or socioeconomic class. While the Buffalo study uses SO_2 and total suspended particulates (TSP) as pollutants, only TSP seemed to show a relationship with mortality. The Nashville study measured sulfation, dustfall, soiling index and SO_2 . Detailed mortality data was reported only for one pollutant, either soiling or sulfation. Both analyses of the data use informal tabular procedures rather than linear model methods. One goal, then, is to analyze these data with regression or analysis of variance techniques and to compare the results with the Lave and Seskin findings.

This paper compares the results on total mortality rates and on cause-specific mortality rates. It then evaluates the entire group of studies for consistency. A null hypothesis to be considered is whether there is evidence of a constant proportional increase in the mortality rates for males and females. Another concern is to determine those diseases which have a strong relationship with pollution.

II. Total Mortality

In their study of 1960-1961 mortality rates¹, Lave and Seskin find some strong relations between pollution and total mortality rates. Sulfate pollution appears to be more important than particulate. They have since redone their study using 1969 data², and their new results are in reasonable agreement with their previous ones. Lave and Seskin use the 1960 regression results to define three new index variables: one for the sulfate measures, another for

the particulate measures, and the third for the social and economic variables. These indices are the new regressors in the analysis of the 1969 mortality rates. If the regressors in an index are important, then the coefficient of the index from the new data should be one. Based on data from 65 SMSAs, the coefficient of the sulfate index is 1.7, and its 95% confidence interval includes 1. This regressor, then, retained its validity. By definition, the 1960 sulfate index is 1 based on 117 SMSAs. When the two coefficients are weighted by the square root of the number of SMSAs used to determine the coefficient, the revised estimate of the effect of a change in sulfate levels on mortality (deaths/ 10^5) is

$$5.2 (\Delta_{\min} \text{SO}_4) + 3.25 (\Delta_{\text{ave}} \text{SO}_4) + 0.07 (\Delta_{\max} \text{SO}_4),$$
where SO_4 levels are measured in units of $\mu\text{g}/\text{m}^3$. Note that Lave reports sulfate levels in units of $10\mu\text{g}/\text{m}^3$.

Since the Buffalo analysis did not use regression techniques in its data analysis, reanalysis of the reported data is necessary to compare these results with the Lave-Seskin analysis.

The mortality ratios reported in references 3 and 4 are based on unadjusted mortality rates for a wide age group (50-69 years). We obtained a listing of the census tracts included in each group from the Erie County County Health Department and indirectly standardized the rates using 1960 census data (see Exhibit 1).

Our reanalysis of the Buffalo data uses the model:

$$Y_{ijk}^{SEP} = \mu + \alpha_1 I_i^S + \alpha_2 E_{jk}^{EP} + \alpha_3 P_k^P + \alpha_4 E_{jk}^{EP} I_i^S + \alpha_5 P_k^P I_i^S + \epsilon$$

where I_i^S is an indicator variable equal to 1 for male values, 0 for females, E_{jk}^{EP} is 10^4 /median family income for the census tracts in the Winkelstein study, P_k^P is the log of the particulate level, and Y_{ijk}^{SEP} is either the log of the unadjusted mortality rate or the square root of the standardized mortality ratio.

Both transformations are indicated by the nature of the data. The square root transformation is a variance stabilizing transformation for Poisson random variables. Logarithmic transformation of the raw rates changes the proportionate effects to additive effects and so is also natural. Finally, examination of the data indicates that the increase in the transformed rates is nearly linear in the log of pollution level. The regression analysis of the data consists of fitting the non-controversial variables (I_i^S , E_{jk}^{EP} , $E_{jk}^{EP} I_i^S$ and then adding P_k^P and $P_k^P I_i^S$ subsequently.

At first glance, the change in the pattern of total female mortality is substantially different from the change in the male mortality. In fact, the proportionate increase in both is nearly the same. The test of the coefficient $\alpha_5=0$ is a test of whether the pollution effect is proportional for men and women. A low F supports this hypothesis. Exhibit 2 contains the ANOVA tables for the analyses of the square root of the indirectly standardized mortality ratios and of the log of the unadjusted rates. Note that both analyses support the hypothesis of

proportionate increases in men and women. This tends to discount the possibility that the male pattern reflects occupational exposures and to strengthen the case for the ambient air pollution as a cause of increased mortality.

(Insert Exhibits 1 & 2 about here).

It is difficult to compare the Winkelstein and Lave-Seskin studies of total mortality because they study slightly different quantities using different regressors. Lave and Seskin study total white mortality for ages 45-64 while Winkelstein studies white mortality for ages 50-69. Winkelstein examines the relationships between mortality and total suspended particulates (TSP) and mortality and SO_2 separately. He finds essentially no relation with SO_2 . Lave examines the relation between mortality and TSP and sulfur pollutants in combination. Further, the economic and social variables are different.

A second model, linear in the pollution variable, is fitted to the Winkelstein data to make the comparison easier. The model is

$$SMR = \alpha_0 + \alpha_1 S + \alpha_2 E + \alpha_3 E \cdot S + \alpha_4 P + \alpha_5 P \cdot S + \epsilon$$

where SMR is the indirectly standardized mortality ratio and

the pollution P is the actual estimated pollution rather than the log of the pollution. Only E and P have significant coefficients, and these are overwhelmingly non-zero. The fitted equation using significant variables only is

$$\text{SMR} = -0.51 + (0.6 \pm 0.08) E + (0.007 \pm 0.001) P$$

where $E = 10^4/\text{family income}$, P is the TSP in $\mu\text{g}/\text{m}^3$. Since the base mortality rate in the Winkelstein data was 200 deaths/ 10^4 (males and females age 50-69), the effect of an increase in TSP of $1\mu\text{g}/\text{m}^3$ on total mortality is

$$200 \cdot 0.007 = 1.4 \text{ deaths}/10^4.$$

The 95% confidence interval for the effect is 1.0 to 1.8 deaths/ 10^4 .

For this comparison, Lave ran an analysis of age 45-64 mortality with average TSP as the only pollution regressor.¹³ The average of the white male and female regression equations is:

$$\begin{aligned} \text{MR} = & 93 + 0.09 P + .014 (P/M^2) \\ & + 0.11 (\%>65) + 0.022 (\% \text{ NW}) \\ & + 0.13 (\% \text{ Poor}) - 0.028 (\log \text{ Population}) \end{aligned}$$

The estimated effect of an increase in mean TSP of $1 \mu\text{g}/\text{m}^3$ is 0.090 deaths/ 10^4 with a 95% confidence interval of 0.04 to 0.14 deaths/ 10^4 . This effect is lower than the derived Winkelstein effect by a factor of 15.6. A test of whether this difference is due to the considerable uncertainty in the estimates is whether the 95% confidence interval of the difference of the two estimates includes zero: this interval

is 1.3 ± 0.6 . Clearly the difference is more than statistical fluctuation. The factor of 15 may be too high, though; for example, the ratio of the lower end of the Winkelstein confidence interval and the upper end of the Lave-Seskin estimate is roughly seven.

There are many possible explanations. The mortality-pollution relation may be non-linear and more steeply rising with greater pollution so that Buffalo, a city with high pollution, may have a much greater mortality pollution effect than a typical city.

Another possibility is that one or both of the studies have left out an important variable and thus are not valid. A third partial explanation is that the error in the Winkelstein pollution estimates is substantially smaller than the error in the Lave-Seskin pollution values. This greater accuracy in the regressor variable reduces the underestimation of the true coefficient.¹⁴ Further, there is a bias in the Lave-Seskin estimates caused by using center city numbers; this bias has an unknown and possibly large magnitude.

III. Cause Specific Mortality Rates

The procedure of this section is to reexamine the reported mortality rates of the intra-city studies and compare the

results to the Lave-Seskin studies. Since the analysis of the logarithm of the reported total mortality is so successful for the Buffalo data, this transformation is used on the disease specific data where indirect age standardization would be difficult. Since some of the cells are based on rates determined from small populations, in cases where the significance was marginal we used weighted least squares regression. The significance reported here is the statistical significance of the reanalysis and not necessarily that reported by the original authors. This criterion emphasizes consistency of effect for males and females, white and nonwhites rather than the simple statistical difference of raw rates. A disease is alleged to be affected by air pollution if and only if it produces consistent effects for different sexes and races.

Exhibits 3-6 are summary tables of the three studies' findings for specific disease categories. The two studies examining infant mortality, Lave and Seskin¹ and the Nashville study,⁹ come to generally different conclusions. The significance of the Lave-Seskin estimated effects is much greater than the Nashville study's. Further, the Nashville study found the effects to be different on whites and nonwhites, suggesting that other variables are more important.

One would expect the effects of air pollution to be apparent in respiratory disease mortality. The Lave and Seskin and the Nashville study both find strong pollution effects on the tuberculosis mortality rate. Neither study finds a significant effect on bronchitis mortality.

Winkelstein examines male deaths for which asthma, bronchitis, or emphysema is mentioned on the death certificate. The ability to examine respiratory disease as both a contributing and underlying cause of death is a more powerful way of detecting possible air pollution effects. Our reanalysis of these data, however, does not find a significant effect. The Nashville data shows an effect on the asthma rate which the Lave-Seskin study does not find. Other respiratory rates are only considered in one study; none of these is significant.

The three studies have a rough consistency in their analysis of cancer mortality rates. The Nashville study finds a stronger pollution effect on total cancer mortality than the Lave-Seskin study. None of the studies shows data representing a statistically significant relation of pollution with lung or bronchial cancer. The Nashville data shows a large effect on nonwhites in the middle and high polluted areas and a much smaller effect on whites. While the original Nashville papers report a relation, the lack of consistent effects across races and pollution levels is such that our reanalysis does not find a significant relation. The Buffalo data shows a significant relation between air pollution and prostatic cancer mortality.

All three studies report on stomach cancer mortality. The Lave-Seskin study finds an effect in 1960 that is not repeated in 1961. While the original Buffalo papers report a relation between stomach cancer and air pollution, our statistical reanalysis of the data does not confirm this finding. Our reanalysis includes the percent Polish and percent foreign born as non-controversial regressors in the stomach cancer regression only. With these two regressors included, the contribution of the pollution variable falls below the point of significance. Similarly, the Nashville data reported an extreme difference of stomach cancer mortality rates between high pollution areas and low pollution areas for males, whites and nonwhites. The female difference, however, is small; and the white and nonwhite differences are not consistent. The statistical reanalysis does not find any significant pollution contributions for stomach cancer mortality. The relation with stomach cancer mortality is unproven on the basis of these studies.

There is substantial disagreement about the relation of pollution to various classes of heart disease mortality. Lave and Seskin find very strong effects on total cardiovascular, hypertensive heart disease, and heart disease mortality rates. The Nashville study does not find any relation with total cardiovascular mortality and finds a racial effect for hypertensive heart disease. In fact, the Nashville data indicate no significant pollution effects on heart disease mortality. Winkelstein finds an almost

significant effect on arteriosclerotic heart disease. The overall picture for heart disease rates is quite unclear.

Exhibit 7 contains the analysis of variance tables of the data in the Buffalo studies. Winkelstein's data shows a marginally significant effect (at 5%) of pollution on the cerebrovascular mortality rate and a strong pollution effect on cirrhosis of the liver.

IV. Conclusions

All three studies find a significant and substantial effect of pollution on total mortality rate. The estimate of the effect from the Lave-Seskin study is a factor of fifteen lower than the estimate from the Winkelstein data. On statistical grounds, the Lave-Seskin coefficients should be lower than the Winkelstein coefficients; however, the observed factor is disconcertingly large. An increase in pollution may be associated with an equal percentage increase in the male and female mortality rates. This similarity of effects argues against occupational exposure of men as a cause of the observed effects.

There is some consistency in the findings for specific causes of death. Lave and Seskin and the Nashville study find a strong association of tuberculosis mortality with pollution

level. In general, there is agreement that most cancer and lung disease mortality rates are not related to pollution levels.

There are also substantial disagreements. Lave and Seskin find a moderately strong relation between pollution and infant mortality which is not replicated in Nashville. They do not find any relation for influenza and pneumonia while the Nashville study does. The Nashville study finds a strong relation for total cancer mortality while Lave and Seskin find only a weak relation. The most substantial disagreement is for the heart disease rates: Lave and Seskin find strong relations for total cardiovascular mortality and hypertensive heart disease; the Nashville data indicates no significant relationships; and the Winkelstein data indicates a nearly significant relation for arteriosclerotic heart disease.

Replication of the intercity and intracity studies is necessary to evaluate the causes of the discrepancies among the studies. One cause of these discrepancies is that the pollutant each study cites may be presumed to have the strongest relation with air pollution of the pollutants available. This pollutant may not have the strongest effect in other studies. At this point, it is impossible to say whether or not the Buffalo effects are exceptionally large or whether they are typical of other cities. The intracity methodology is simple, and the studies are not expensive. The repetition of these studies is an important step in assessing the existence and magnitude of the pollution-mortality relationship.

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Acknowledgements

The authors special thanks go to Professor Lester Lave for making available copies of his draft manuscript, for making a special regression run, and for his discussions. Beth A. Schecher performed extensive computer calculations; Gregory Cianflone also assisted with the Nashville calculations; Debra Shenk read and criticized the paper.

EXHIBIT 1.

INDIRECTLY ADJUSTED MORTALITY RATIOS FROM WINKELSTEIN

Buffalo, Whites 50-69+, 1959-61 Males

Pollution Level
Increasing Pollution →

	1	2	3	4
1	---	1.57	1.67	2.11
2	1.03	1.15	1.28	1.53
3	---	1.04	1.17	1.43
4	0.89	0.98	1.19	---
5	0.79	0.94	0.94	---

Buffalo, Whites 50-69+, 1959-61 Females

	1	2	3	4
1	---	1.13	1.40	2.34
2	0.92	0.97	1.29	1.49
3	---	0.98	1.24	1.39
4	1.04	0.84	1.15	---
5	0.88	0.92	0.69	---

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EXHIBIT 2

ANALYSIS OF VARIANCE TABLES WINKELSTEIN BUFFALO DATA^{3,4} TOTAL MORTALITY FOR 50-69

SOURCE	SQUARE ROOT INDIRECTLY STANDARDIZED MORTALITY RATIO				LOG UNADJUSTED RATE			
	SUM OF SQUARES	DEGREES OF FREEDOM	MEAN SQUARE	F*	SUM OF SQUARES	DEGREES OF FREEDOM	MEAN SQUARE	F
Non-controversial	4.525	3	1.508	----	0.925	3	0.308	----
Pollution	1.736	1	1.736	39.5	0.113	1	0.113	37.7
Pollution • Sex	0.017	1	0.017	0.4	0.002	1	0.002	0.7
Residual	1.157	26	0.044	----	0.068	26	0.003	----

*F.₀₁(1,26) = 7.72

EQUATIONS:**

$$(10 (\text{SMR}_{ijk}))^{\frac{1}{2}} = -2.6 + (0.73 \pm 0.15) E_{jk}^{EP} + (2.32 \pm 0.37) P_k^P \\ + (-0.13 \pm 0.36) I_i^S + (0.13 \pm 0.21) E_j^{EP} I_i^S$$

$$\log MR_{ijk} = -0.38 + (0.21 \pm 0.038) E_{jk}^{EP} + (0.59 \pm 0.09) P_k^P \\ + (0.18 \pm 0.09) I_i^S + (0.05 \pm 0.05) E_{jk}^{EP} I_i^S$$

** Coefficient is reported as estimated coefficient $\pm$ its standard error.

EXHIBIT 3

INFANT MORTALITY SUMMARY OF PUBLISHED RESULTS* SOURCE: 1,9

SOURCE	FETAL	<28 DAYS	UNDER 1 YR
Lave-Seskin 1960	0.14 (2.7) Mean S	0.08 (1.6) Mean P	0.37 (2.8) Min P
Lave-Seskin 1961	0.14 (1.8) Mean S	0.12 (2.2) Mean P	0.32 (2.1) Min P
Nashville White	NS	F = 11.44 (p<0.005) dustfall	F = 30.5 (p<<0.005) sulfation
Nashville Non-White	F = 9.08 (p<0.01) dustfall	NS	NS

*NS: no statistically significant relation. Lave-Seskin findings are coefficient (t-value of coefficient) pollutant for 1960 and 1961 respectively that was found to be most significant statistically. A t-value of roughly 2 is significant at the 5% level.

EXHIBIT 4

RESPIRATORY MORTALITY SUMMARY AND REANALYSIS OF PUBLISHED RESULTS SOURCE 1,3,10

SOURCE	TOTAL RESPIRATORY	SELECTED RESPIRATORY	INFLUENZA & PNEUMONIA
Lave-Seskin 1960	NA	NA	0.001 (1.2) mean S Influenza 0.003 (.7) min P Pneumonia
Lave-Seskin 1961	NA	NA	0.001 (2.2) mean S Influenza 0.002 (.5) min P Pneumonia
Nashville M-F (sulfates only)	NS: F = 9.2 (2,2)	NS: F = 8.2 (2,2)	S: F = 82.0 (2,2)
Nashville Race (sulfates only)	NS: F = 1.4 (2,2)	NS: F = 1.1 (2,2)	NS: F = 3.3(2,2)
Winkelstein	NA	NA	NA
	BRONCHITIS & EMPHYSEMA	TUBERCULOSIS	ASTHMA
Lave-Seskin 1960	0.0001 (1.1) max P	0.002 (3.6) mean P	0.001 (2.6) mean P
Lave-Seskin 1961	0.00004 (0.5)	0.002 (4.0)	0.001 (1.6)
Nashville M-F (sulfates only)	NS: F = .5 (2,2)	S F = 48.7 (2,2)	NA
Winkelstein	NS: F =2.4 (1,13)	NA	NA

Notes: NA: study did not consider this disease

NS: not statistically significant at 5%

S: statistically significant at 5%

Lave-Seskin data are coefficient (t-value) pollutant found to be most significant.

Nashville data are significance code: F value from ANOVA of log of mortality rate and degrees of freedom

EXHIBIT 5

CANCER MORTALITY
SUMMARY AND REANALYSIS OF
PUBLISHED RESULTS
SOURCES: 1,5,8,12

SOURCE	ALL CANCER	LUNG & BRONCHIAL CANCER	ESOPHAGUS	BUCCAL & PHARYNX
Lave-Seskin 1960	0.009(2.1)min S 0.006(2.0)mean S	0.006 (2.0) means S	NA	0.001 (1.1) min P
Lave-Seskin 1961	0.004(0.8)min S 0.005(1.6)mean S	0.001 (1.1)		0.0004 (.7)
Nashville M-F(soiling)	S: F = 427 (2,2)	NS: F = 5.0 (2,2)	NS: F = .5 (2,2)	NA
Nashville Race(soiling)	S: F = 40 (2,2)	NC	NC	NA
Winkelstein (TSP)	NA	NS: F = 0.6 (1,13)**	NA	NA
	STOMACH CANCER	BLADDER CANCER	LARGE INTESTINE	BREAST
Lave-Seskin 1960	0.006(2.5)min S 0.001(2.3)max S	NA	NA	0.0003 (1.7) max S
Lave Seskin 1961	0.003(1.1)min S 0.0003(.5)max S	NA	NA	0.0003 (1.9) max S
Nashville M-F(soiling)	NS: F = 2.0 (2,2)	NS: F = 2.4 (2,2)	NS: F = 5.1 (2,2)	NA
Nashville Race (soiling)	NS: F = 1.3 (2,2)	NS: F = 0.8 (2,2)	NS: F = 0.5(2,2)	NA
Winkelstein (TSP)	NS: F = 3.0 (1,24)	NA	NA	NA
	PROSTATE			
Lave-Seskin 1960	NA			
Lave-Seskin 1961	NA			
Nashville Sex (soiling)	not valid			
Nashville Race (soiling)	NS: F = 1.8 (2,2)			
Winkelstein (TSP)	S: F = 48.4 (1,5)			

Notes NA: study did not consider this disease
 NC: missing cells in data; no calculation made on table
 NS: not statistically significant at 5%
 S: statistically significant at 5%
 Lave-Seskin data are coefficient (t-value) pollutant found to be most significant.
 Nashville data are significance code: F value from ANOVA of log of mortality rate and degrees of freedom

**Malignant neoplasms of bronchus, trachea, and lung, males only.

EXHIBIT 6

HEART DISEASE
SUMMARY AND REANALYSIS OF PUBLISHED RESULTS
SOURCES: 1,6,11

SOURCE	TOTAL CARDIOVASCULAR	GENERAL ARTERIOSCLEROSIS	ARTERIOSCLEROTIC HEART DISEASE
Lave-Seskin 1960	0.058 (3.1) min S, 0.028 (2.6) mean S	NA	NA
Lave-Seskin 1961	0.050 (2.9) 0.030 (3.0)	NA	NA
Nashville(soiling)*	NS: F = 1.0 (2,3)	NS: F = 0.01 (2,3)	NS: F = 1.7 (2,3)
Winkelstein (TSP)	NA	NA	NS: F = 4.0 (1,26)
	HEART	ENDOCARDITIS	OTHER MYOCARDIAL DEGENERATION
Lave-Seskin 1960	0.013 (2.0) max S, 0.066 (2,3) min S	0.002 (1.9) max P	NA
Lave-Seskin 1961	0.012 (3.5) 0.042 (2.9)	0.002 (1.5) max P	NA
Nashville (soiling)	NA	NA	NS: F = 9.1 (2,3)
Winkelstein	NA	NA	NA
	HYPERTENSIVE HEART DISEASE	RHEUMATIC HEART DISEASE	
Lave-Seskin 1960	0.003 (3.2) max S	NA	
Lave-Seskin 1961	0.003 (3.6)	NA	
Nashville (soiling)	NS: F = 4.3 (2,3) (significant race pollution interaction)	NS: F = 3.4 (2,3)	
Winkelstein	NA	NA	

Notes: NA: study did not consider this disease

NS: not statistically significant at 5%

S: statistically significant at 5%

Lave-Seskin data are coefficient (t-value) pollutant found to be most significant.

Nashville data are significance code: F value from ANOVA of log of mortality rate and degrees of freedom.

* Mean square for error in Nashville analysis is the sex-race interaction plus the sex-race-pollution interaction.

EXHIBIT 7

ANOVA TABLES OF LOG MORTALITY RATES REANALYSIS OF BUFFALO STUDIES SOURCES: 6,7,8

	LOG STOMACH CANCER			LOG ARTERIOSCLEROTIC HEART DISEASE		
	SUM OF SQUARES	DEGREES OF FREEDOM	F	SUM OF SQUARES	DEGREES OF FREEDOM	F
Non-controversial	2.128	5		7.899	3	
Pollution	0.193	1	3.0	0.775	1	4.0
Pollution x Sex	0.002	1	0.03	0.007	1	0.04
Error	1.538	24		4.980	26	

	LOG CIRRHOSIS OF LIVER			LOG CEREBROVASCULAR		
	SUM OF SQUARES	DEGREES OF FREEDOM	F	SUM OF SQUARES	DEGREES OF FREEDOM	F
Non-controversial	3.033	3		2.660	3	
Pollution	1.046	1	38.6**	1.193	1	4.3*
Pollution x Sex	0.001	1	0.04	0.015	1	0.05
Error	0.705	26		7.179	26	

*SIGNIFICANT AT 5%

**SIGNIFICANT AT 1%