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Morbidity and Pollution: Model Specification Analysis for Time-Series Data on Hospital Admissions[1]

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Time-series analysis of effects of pollutants on emergency hospital admissions indicates important synergistic interactions among pollutants and to a lesser degree nonlinearities in effects of single pollutants. Comparisons of alternative econometric specifications are made to determine the appropriateness of incorporating nonuniform pollution impacts. The data substantially support the existence of synergisms among pollutants with high levels of sulfur dioxide, SO_2 (particulates), increasing the impact of particulates (SO_2) on emergency hospital admissions. Marginal effects of either pollutant are, however, small at current ambient air quality levels. These results indicate that damage estimates were likely to be understated during the 1960's when pollution levels were high, while, at current levels of those pollutants considered here, marginal damages are lower than would be estimated in studies failing to incorporate synergistic and nonlinear impacts.

I. INTRODUCTION

Damages associated with air pollution take many forms, with economists often finding damages to materials to be of the same order of magnitude as damages to human health. Yet, the motivation underlying legislative initiatives continues to be concern for human health. Mortality damages, largely premature death, have received extensive documentation as, for example, in the recent Lave and Seskin [3] volume. However, 20 years suffering from bronchitis might only be captured as a death premature by just a few days in a mortality study. As a consequence, morbidity damages may take on potentially greater importance when analyzing the impact of environmental pollutants on human health.

Previous work has typically dealt with average damages per microgram of pollutant (implicitly assuming damage linearity), virtually ignoring the extent of nonlinearities and synergisms in effects of pollutants on human health in formal analysis. As a result, policymakers have tended to distrust the estimated damage coefficients, believing them to be biased downward due to these omissions. One may conjecture that the frequent recent findings that proposed legislation involves marginal costs

[1] The research results presented in this paper provide the empirical foundations for Graves and Krumm [2]. We acknowledge the American Enterprise Institute and the Institute of Behavioral Science at the University of Colorado for partial funding of this research. Neither of these organizations is, however, responsible for our conclusions. We thank R. Bear, N. Kiefer, A. Zellner, and an anonymous referee for helpful comments on an earlier draft and P. Zellner for helpful research assistance. The usual disclaimer applies.

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orders of magnitude greater than marginal environmental benefits (see, e.g., many of the studies reported in Tolley and Graves [9]) stem fundamentally from past methodologies felt by decision-makers to be suspect.

Laboratory experiments (see Shy *et al.* [8] for a review) with specific pollutants are subject to criticism, relative to epidemiological studies, for failing to simulate ambient air in which a number of pollutants are present. In casual policy discussion the presence of, for example, particulates is argued to increase the damage associated with sulfur dioxide and its higher order sulfates, although formal substantiation of these claims has heretofore been lacking.

The concern of this paper is with morbidity damages, measured by emergency hospital admissions, for these relate to sulfur dioxide (SO_2) and the coefficient of haze (COH), a rough particulate measure. The analysis takes into account nonlinearities of effects of single pollutants (whose importance was noted in Marin [4]) and synergisms in effects among these major pollutants. The approach to measuring the morbidity effects of pollutants taken here is described in Section II. Log–Taylor expansions are used to approximate the underlying relationship among cardiac and respiratory hospital admissions and pollution and climate variables. This allows for examination of the extent to which effects of single pollutants are constant over the sample range and the extent to which these effects are changed due to variation in levels of other pollutants. This approach is important especially in studies, like the present, in which theoretical guidance in specification is lacking.

Classical least-squares estimates are presented in Section III that illustrate the sensitivity of damage estimates to model specification. More refined analysis of the time-series structure of the model is presented in Section IV, allowing for preliminary model selection. Diagnostic specification checks based on estimation under alternative error structure assumptions are utilized to lend credence to the appropriateness of alternative specifications. Based on this preliminary screening of models, posterior odds ratios are used to determine point estimates of pollutant effects that are most consistent with the data. Damage functions associated with pollutants, based on the informational content of the data regarding the appropriateness of the remaining specifications, are presented and the implications for policy analysis are discussed.

II. EMPIRICAL APPROACH

In this section a modeling procedure is outlined which applies to examination of immediate or short-term impacts of changes in local pollution measures on morbidity as captured by total cardiac and respiratory (TCR) emergency room admissions to Cook County Hospital in Chicago, Illinois.[2] Low-dose cumulative effects are not explicitly considered. However, since the analysis is confined to one very small geographic region, in the absence of significant migration in and out of the area of people who might be affected differently by pollution, the cumulative long-run exposure to low-dose pollution will not affect the empirical modeling. Low-dose cumulative impacts may then be captured in part by the constant term in the

[2]Differences between results disaggregated by sex and by cardiac and respiratory category are presented in Graves and Krumm [2]. Although some differences are apparent the qualitative nature of the results are not sensitive to the aggregation used here.

regression analysis, a term which would differ among areas depending on characteristics of the population and area involved. Only to the extent that pollution levels in the sample period differ dramatically from those of the past will they affect low-dose cumulative impacts in a manner other than through the constant term. Moreover, if the distribution of economic and demographic characteristics of the population remains unchanged over the sample period aggregate data analysis can lead to useful insights into the nature of the relationship between morbidity and pollution, although application of the results to other areas where these characteristics may differ requires caution.

While morbidity is only one component of human health, emergency room hospital admissions in turn represent only one component of morbidity with other admissions and illness treated at home also being important. To obtain a full estimate of the impact of pollution upon human health an analysis akin to the methodology presented here could usefully be applied to these other health dimensions. The effects of pollution on emergency hospital admissions may be analyzed in the context of a health production function whereby a variety of actions may be taken by persons in response to variations in the level of pollution, with sicknesses due to pollution reflecting the net result of the interaction of changing pollution levels and avoidance practices. Emergency hospital admissions then measure only the more extreme cases resulting from pollution health damages.

The framework utilized here takes emergency hospital admissions as one output of a health production function with pollution measures and other climatic controls as exogenous inputs. Clearly, socioeconomic characteristics affect the overall level of emergency hospital admissions and the potential impact of changing pollution levels on hospital admissions in any given area. However, the constancy of such factors over relatively short periods of time avoids many of the problems associated with comparisons of pollution effects on human health among regions (see, e.g., Lave and Seskin [3]) and over long periods of time.[3]

The production function approach facilitates the study of interactions among pollutants in their net effects on human health and provides a common way of interpreting coefficients associated with different variables. For example, with morbidity level held constant the change in one pollutant needed to offset a change in another pollutant is analogous to measuring the degree of substitution between (negative) inputs in production, which leads to potentially important reasons for differential pollution controls. Similarly, measurement of the percentage increase in morbidity due to equal percentage increases in all pollutants provides a measure of scale effects that is most relevant to discussions of threshold effects and important in analyzing overall pollution level policies. Additionally, synergisms serve to modify these relationships in that the mix of pollutants might alter the magnitudes of impacts with, for example, marginal effects of a pollutant on human health depending on levels of other pollutants.

Closely related to the preceding are changing levels of climatic control variables which may be usefully interpreted as factors shifting the production function much the same as technological change in the more usual production analysis. Climate

[3]One of the benefits of using time-series data in a single area as opposed to cross-section data based on different geographic areas is that systematic sorting of individuals among areas due to spatial variation in air quality and climatic conditions is less likely to call for the need of more complicated estimation strategies to take this response into account.

variables might directly affect emergency admissions as well as through the indirect channel of altering the degree of damage from a given level of pollution. Humidity is perhaps the most well documented of the climate variables with respect to its effect on pollution damages (see McJilton *et al.* [5], but sunlight and temperature may also have effects on marginal pollution damages as well as direct seasonal effects on morbidity (associated perhaps with "flu season" and general systemic weakness associated with very high temperatures).

Building on this background, let emergency hospital admissions be a function of a vector of pollutants, $P = (P_1, P_2, \ldots, P_n)$, and a vector of production shift variables, $S = (S_1, S_2, \ldots, S_n)$,

$$M = f(P, S) \tag{1}$$

where the form of $f(\cdot)$ depends in part on the nature of aggregation. The general relationship in (1) does not specify the nature in which pollutants and climate control variables affect morbidity. The explicit manner in which pollutants, in particular, affect hospital admissions is essential for policy analysis, with some specifications greatly limiting the form of the relationship. Below, a variety of general relationships is examined providing a framework for examining the functional form of $f(\cdot)$.

A second-order logarithmic Taylor's expansion of (1) may be used as an approximation to the exact but unknown functional relationship between M and the elements in P and S.[4] The extent to which nonlinearities, synergisms, and "economies of scale" may be captured by such a procedure is very important. When (1) is expanded the true functional relationship $f(\cdot)$, is approximated by

$$\ln M = \ln M_0 + \sum_{i=1}^{n} \gamma_i \ln p_i + \sum_{i=1}^{m} \alpha_i \ln s_i + \tfrac{1}{2} \sum_{i=1}^{n} \sum_{j=1}^{n} \beta_{ij} \ln p_i \ln p_j + \tfrac{1}{2} \sum_{i=1}^{m} \sum_{j=1}^{n} \delta_{ij} \ln s_i \ln p_j + \tfrac{1}{2} \sum_{i=1}^{m} \sum_{j=1}^{m} \nu_{ij} \ln s_i \ln s_j + \mu. \tag{2}$$

In (2) the γ and α terms are the parameters associated with the first derivative of $f(\cdot)$ with respect to changes in the logarithms of elements in P and S. If no other terms were included on the right-hand side of (2), this would represent a first-order logarithmic Taylor's expansion of (1). In such a case synergisms and nonlinearities in proportionate effects of increasing pollution levels on morbidity are not allowed. The β, ν, and δ terms are the parameters associated with the second derivative of $f(\cdot)$ with respect to the elements in P and S. Specifically, the β_{ij} terms capture the interactions among various pollutants (nonlinearities and synergisms) in production of morbidity, the ν_{ij} terms represent the interactions among elements in S, and the δ_{ij} terms capture interactions between elements in P and those in S. The μ term

[4] Taylor expansions of $f(P, S)$ up to a given order in either variable levels or their logarithms can be viewed as special (extreme) cases of a still more general framework which allows for a "combination" of level and logarithmic relationships along the lines suggested by Box and Cox [1]. A more complete analysis could incorporate such considerations although more complicated relationships could also be obtained by higher order expansions of Eq. (1). Our purpose is to examine the extent to which choice of functional form affects policy implications of the results and to illustrate that care needs to be taken in blindly applying empirical results that are very sensitive to such considerations.

TABLE I
Variable Definitions and Summary Statistics

Variable name	Definition	Mean	Standard deviation
TCR	Total cardiac and respiratory admissions	64.8	28.7
SO_2	SO_2 concentration (ppm × 100) at day of admission	2.5	1.4
COH	COH concentration (per 1000 linear ft × 100) on the day of admission	68.2	26.4
AT	Average temperature (F°)	47.9	19.4
ARH	Average relative humidity (%)	69.5	11.4
SC	Sky cover (tenths)	6.9	3.5

represents errors resulting from third and higher order terms in full logarithmic Taylor series representation of (1).

In the following sections empirical results are discussed and analyzed based on the framework presented above. There is a need to examine the extent to which models based on simplifications of (2) and possible extensions to include higher order terms are warranted by the data.

Data

The data employed here were collected under the direction of Professors T. Namekata and B. Carnow of the School of Public Health, University of Illinois. The Namekata and Carnow [6] study describes the data and its limitations fully. In brief, the morbidity data are the number of emergency room admissions from midnight on Monday of each week to midnight on Tuesday at Cook County Hospital in Chicago for the 18-month period beginning in mid-September 1971 and ending at the end of March 1973 (in total 81 observations). The pollutants (elements of P) considered are sulfur dioxide SO_2 and coefficient of haze COH since data on other pollutants were unreliable or missing.[5] Meteorological controls (elements of S) employed are average temperature AT, average relative humidity ARH, and percent of sky cover SC, variables whose importance has already been discussed. Definitions and summary statistics associated with the variables employed are presented in Table I. For the remainder of this work, logarithms of these variables are designated by the prefix L.

III. CLASSICAL LEAST-SQUARES ESTIMATION RESULTS

Model 1: No Interactions

A very restrictive assumption is that all β, δ, and ν parameters in (2) are zero. In such a situation nonlinearities and synergisms in the effects of percentage changes in pollutant levels on percentage changes in morbidity are excluded. Under this assumption the specification in (2) becomes a first-order logarithmic Taylor expan-

[5]It is assumed that the air quality affecting emergency room patients at Cook County Hospital is measured by the five monitoring stations surrounding the hospital. The monitored air quality data from these stations were averaged, giving equal weight to each station.

sion of (1), with some unknown error resulting from omission of second and higher order terms in the complete Taylor expansion.

In Table II, column 1 we present classical least-squares estimates of the parameters of (2) under these restrictions for total combined cardiac and respiratory hospital admissions (TCR). This estimation technique assumes the error terms to be independent and identically distributed over time.

Although the Durbin–Watson statistic suggests that the classical least-squares error structure assumption is violated, even under these conditions the parameter estimates are unbiased if the structural model is correctly specified. On the other hand, the reported standard errors are not useful in reflecting the precision of the estimates. The coefficient associated with LSO_2 is positive though small in magnitude with the coefficient associated with LCOH negative, contrary to *a priori* expectations. The effects of average temperature and sky cover are negative with that for LAT being larger in magnitude. The coefficient on LARH is positive and similar in a magnitude to that for LAT. A negative effect of both LAT and LSC and a positive effect of LARH is consistent with their posited impacts.

Model 2: Pollutant Interactions

To more carefully examine nonlinear and synergistic effects of proportionate changes in pollutant levels on the percentage change in TCR admissions, consider the case where the β_{ij} terms in (2) are not assumed to be zero, with possible error resulting from the noninclusion of other terms in the full logarithmic Taylor series expansion. Interaction effects among elements of S and P are still not allowed nor are those among elements of S.[6] Classical least-squares estimates of the parameters in (2) under these assumptions are presented in column 1 of Table 2. Although the Durbin–Watson statistic again suggests violation of the error structure assumptions, if the model is correct the parameter estimates themselves are still unbiased. The coefficients associated with climatic variables are not sensitive to the inclusion of β_{ij} terms. Based on the point estimates in the second column of Table II, for each pollutant at the sample mean, 1% increases in either SO_2 or COH have negligible effects on morbidity. However, for the other pollutant two standard deviations above its sample mean the effects become more substantial with a percent increase in SO_2 leading to a 0.25% increase in morbidity and a percent increase in COH leading to a 0.33% increase in morbidity. Effects of increasing pollution levels on morbidity are increased by the interaction term, LSO_2LCOH, while effects are decreased by the squared individual pollutant variables. The former effect dominates only slightly with 1% increases in both pollutants increasing the combined effects of 1% changes in both pollutants on morbidity by 0.02%.

Model 3: Interaction among Pollutant and Climate Variables

Comparison of results in column 1 with those of column 2 in Table II illustrates the sensitivity of estimated human health effects of pollution to the form of the

[6]The negative coefficient for LCOH in the first column in Table 2 is a priori not acceptable and suggests that the linear model is not capable of adequately capturing effects of pollutants on human health. The approach taken here is to expand on the linear specification step by step, in the hopes of preserving simplicity without adding needless complications in the process of determining an appropriate relationship.

TABLE II

Classical Least-Squares Estimates of Eq. (2) under Alternative Model Specifications[a]

Independent variables	Model specification			
	1	2	3	4
C	4.40	0.51	−11.89	−14.03
	(1.09)	(3.15)	(15.6)	(28.06)
LSO_2	0.036	−1.54	−4.75	−1.67
	(0.067)	(0.66)	(2.50)	(0.68)
LCOH	−0.04	2.16	5.76	12.92
	(0.09)	(1.53)	(4.11)	(20.84)
LAT	−0.38	−0.36	−0.082	−0.37
	(0.07)	(0.068)	(1.01)	(0.07)
LARH	0.32	0.28	3.29	0.285
	(0.24)	(0.24)	(3.62)	(0.25)
LSC	−0.05	−0.04	−0.70	−0.041
	(0.05)	(0.05)	(0.77)	(0.05)
LSO_2LCOH		0.41	0.354	0.441
		(0.17)	(0.195)	(0.17)
LSO_2^2		−0.10	−0.080	−0.213
		(0.07)	(0.096)	(0.63)
$LCOH^2$		−0.30	−0.278	−2.92
		(0.19)	(0.206)	(5.12)
$LSCLSO_2$			(0.107)	
			(0.158)	
LSCLCOH			0.181	
			(0.199)	
LATLCOH			−0.061	
			(0.249)	
$LATLSO_2$			−0.037	
			(0.206)	
$LARHLSO_2$			0.889	
			(0.604)	
LARHLCOH			−0.908	
			(0.934)	
LSO_2^3				0.065
				(0.08)
$LCOH^3$				0.210
				(0.41)
R^2	0.33	0.39	0.41	0.39
SEE	0.30	0.29	0.30	0.30
D.W.	1.08	1.11	1.21	1.11

[a]Reported standard errors are in parentheses.

model. Allowing for nonlinearities and synergisms among pollutants greatly alters the estimated marginal impacts of changes in pollution levels on measures of morbidity. To further pursue this sensitivity, consider the case where further interactions are allowed, with effects of pollution on human health depending on levels of other climatic control variables in a more complicated manner, with the δ_{ij} terms not restricted to be zero.

Classical least-squares estimates of (2) under these conditions are presented in column 3 of Table II. Again the Durbin–Watson statistic suggests that the error

structure assumptions associated with this estimation technique are not valid. Moreover, inclusion of these additional variables does not lead to substantial reductions in the standard error of the estimates, which suggests that more simple models may be appropriate. However, the results stemming from this specification can be used to examine the sensitivity of earlier results to this more complicated relationship. Estimates of the effects of percentage changes of climatic control variables on TCR at the means of the pollution variables are presented in column 1 of Table III. These afford a comparison with the single-parameter estimates for these variables in Table II, the results not differing substantially between specifications. Based only on the point estimates in column 3 of Table II, increases in LSO_2 lead to decreases in the effect of sky cover and, though less precisely, average temperature, and increases in the effect of average humidity. Increases in LCOH lead to increases in the impact of sky cover and decreases in the impact of average temperature and average relative humidity.

Of particular policy relevance is the sensitivity of estimated pollution impacts on cardiac and respiratory admissions to model specification. Comparison of results of Models 2 and 3 suggests that the qualitative nature of effects of pollutants on hospital emergency admissions is only slightly affected by inclusion of additional pollution–climate interaction possibilities. When all variables are at their sample means effects are very small. For the other pollutant two standard deviations above its sample mean, however, a percent increase in SO_2 leads to a 0.23% increase in morbidity and a percent increase in COH leads to a 0.27% increase in morbidity. These results are quite similar to those based on estimates in column 2 of Table II and illustrate the insensitivity of estimated synergistic effects to the inclusion of the climatic interaction variables. Shifts in climate control variable levels modify the results somewhat although taken at their sample mean values, results of Models 2 and 3 are surprisingly consistent. For policy purposes this means that estimated pollution effects are not dramatically altered by including additional second-order interaction effects, making the implications of the simple model more reliable.

Model 4: Higher Order Taylor Expansions

The second-order logarithmic expansions presented above all suggest that the partial effects of a single pollutant on morbidity declines (the second-order pollution-effects term is negative) as the level of the pollutant increases. This most likely

TABLE III

Percentage Changes in Total Cardiac and Respiratory (TCR) Admissions Due to Percentage Changes in Climatic Control Variables at Mean Levels of SO_2 and COH Pollutants

	1	2
Average temperature	−0.37	−0.160
Average relative humidity	0.027	−0.206
Sky cover	−0.033	0.031

represents the flattening of the pollution damage function for higher levels of pollution in the sample. However, this relationship is unlikely to hold for still higher levels of pollution where it might be expected that the partial effects increase again.

To deal with this possibility more fully, two third-order terms of the Taylor expansion are included in addition to the second-order terms of Model 2.[7] Classical least-squares estimates of these parameters are presented in column 4 of Table II. The Durbin–Watson statistic again suggests the error structure assumptions of classical least-squares estimation are not satisfied, while the standard error of the estimates is not substantially changed in comparison with that associated with the results in column 2 which does not include the third-order terms. The signs of all parameter estimates, however, remain essentially the same as before.

Effects of changing SO_2 at first decline and then increase at higher levels of SO_2 with the impacts lowest around the mean of the sample. Effects of COH are similar to those stemming from the quadratic specification generally decreasing over the sample space with the cubic terms only serving to modify the diminished impacts at higher levels of COH. Estimated effects of percent increases in either pollutant at their sample means are slightly negative, but with the other pollutant two standard deviations above its sample mean a percent increase in SO_2 leads to a 0.20% increase in morbidity and a percent increase in COH leads to a 0.24% increase in morbidity, which is similar to that of previous estimates. Synergisms between pollutants and the increasing effect of SO_2 on hospital admissions at higher levels of SO_2 dominate the declining impact of COH at higher levels of COH so that percent increases in both SO_2 and COH lead to an increased effect of pollutants on morbidity by almost 0.29% at the sample means.

Based on classical least-squares point estimates the implications of these models for calculating effects of changes in pollutant levels on hospital admissions vary, with the inclusion of third-order single-pollutant variables changing the qualitative nature of the results most. However, for each model the error structure assumptions associated with classical least-squares estimates are violated.

IV. ERROR STRUCTURE ANALYSIS AND MODEL CHOICE

The results presented in Section III suggest the importance of examining alternative specifications in analysis of morbidity effects of pollutants, with the qualitative and quantitative nature of the results often sensitive to specification choice. A finding common to all specifications, however, is that the error structure does not conform to classical least-squares estimation assumptions. In this section the residuals are analyzed allowing for more complete examination of the models presented above. Joint estimation of structural parameters and error process parameters provides a useful diagnostic tool, allowing for examination of model misspecification and sensitivity of results to estimation methodology.

[7]Additional analysis of the second-order expansion in (2) to include estimates of the ν_{ij} terms provided no further insights and is not presented here. In addition to the two third-order terms examined here more complicated expansions could be investigated. The approach taken in this work is to attempt to adequately approximate the true underlying relationship given the variation available in the data and not to identify it exactly. There is no assurance that the specification analysis presented here yields information regarding potentially more complicated relationships that could be determined given more data.

Analysis of Residuals

The Durbin–Watson statistics reported in Section III measure the extent to which the error terms follow a first-order autoregressive process as opposed to being independent over time. While these calculations are useful as a first step in examination of residual structure, they result from the imposition of a first-order autoregressive process on the errors, the calculation of which may actually result from higher order autoregressive and/or moving average process. Especially with weekly data it is possible that more complicated error structures are appropriate. Such possibilities require explicit examination in order that estimation of the structural model parameters in conjunction with those characterizing the error process can lead to useful insights into the nature of the actual pollution–health relationship.

Important in the analysis is identification of the appropriate error structure. We consider here a variety of reasonable combinations of autoregressive and moving average (ARMA) error processes. Investigation of the autocorrelation and partial autocorrelation functions of the residuals suggests that an ARMA(1,0) process is likely. Overfitting is used in this section to more fully examine other possibilities. The current analysis focuses on three alternative structures:

$$\begin{aligned} &\text{ARMA}(1,0): \quad \epsilon_t = \phi_1 \epsilon_{t-1} + \mu_t \\ &\text{ARMA}(2,0): \quad \epsilon_t = \phi_1 \epsilon_{t-1} + \phi_2 \epsilon_{t-2} + \mu_t \\ &\text{ARMA}(1,1): \quad \epsilon_t = \phi_1 \epsilon_{t-1} + \mu_t + \theta_1 \mu_{t-1} \end{aligned} \tag{3}$$

where ϵ_t is the residual from the classical least-squares results and μ_t is an independent and identically distributed error term. The ARMA(1,0) process is embedded in either the ARMA(2,0) process (with the restriction that $\phi_2 = 0$) or the ARMA(1,1) process (with the restriction that $\theta_1 = 0$). Hence, a test of the appropriateness of an ARMA(1,0) error structure is to consider more complicated processes that include the ARMA(1,0) as a special case. If estimation of an ARMA(2,0) or ARMA(1,1) process leaves the estimate of ϕ_1 unchanged with estimates of either ϕ_2 or θ_1 close to zero, this supports the more simple ARMA(1,0) assumption.[8]

Estimates of the ARMA models in (3) for each model are presented in Table IV.[9] The estimate of ϕ_1 is quite precisely estimated and in all cases its magnitude is insensitive to the inclusion of other autoregressive and moving average terms which are small and imprecisely estimated. These results strongly support an ARMA(1,0) error process which is assumed to be the case in the analysis that follows.

[8]Expansion of the order of both the autoregressive and moving average components of the error structure can lead to parameter redundancy. For example, if the true error process is ARMA(1,0) $\epsilon_t = \phi_1 \epsilon_{t-1} + \mu_t$ and $\epsilon_{t-1} = \phi_1 \epsilon_{t-2} + \mu_{t-1}$. Subtracting the second expression from the first results in $\epsilon_t = (1 + \phi_1)\epsilon_{t-1} - \phi_1 \epsilon_{t-1} + \mu_t - \mu_{t-1}$ which is ARMA(2,1) in form although subject to parameter restrictions. Under these parameter restrictions, however, this is neither an invertible nor stationary process.

[9]The Box–Jenkins routine in the Econometric Software Package (ESP) was used in these computations. This requires input of initial parameters estimate. These were based on the methodology suggested in Nelson (7).

TABLE IV
Time Series-Structure of Residuals from Table 2[a]

Model specification	Type of Structure	Parameter estimates ϕ_1	ϕ_2	θ_1
Model 1	ARMA(1, 0)	0.456		
		(0.099)		
	ARMA(2, 0)	0.438	0.041	
		(0.112)	(0.110)	
	ARMA(1, 1)	0.507		0.064
		(0.205)		(0.240)
Model 2	ARMA(1, 0)	0.416		
		(0.101)		
	ARMA(2, 0)	0.419	−0.008	
		(0.113)	(0.114)	
	ARMA(1, 1)	0.407		−0.011
		(0.247)		(0.245)
Model 3	ARMA(1, 0)	0.390		
		(0.102)		
	ARMA(2, 0)	0.372	0.050	
		(0.112)	(0.110)	
	ARMA(1, 1)	0.457		0.077
		(0.246)		(0.279)
Model 4	ARMA(1, 0)	0.445		
		(0.099)		
	ARMA(2, 0)	0.464	−0.045	
		(0.112)	(0.109)	
	ARMA(1, 1)	0.394		−0.064
		(0.224)		(0.245)

[a]Standard errors in parentheses.

Joint Estimation of Structural and Error Process Parameters

A fruitful means of choosing among the specifications presented earlier is to jointly estimate the structural and error process parameters. The error processes resulting from the models estimated in Section III could be due to omission of important variables in the specifications or might stem from sources that are of little consequence for the structural relationships involved. Comparison of parameter estimates from Section III with those incorporating the error structure process determined in the previous section can shed light as to the source of error structure characteristics. If a model is correctly specified in terms of the structural variables, the parameter estimates should not be dramatically altered by these two estimation techniques. These issues are now addressed, allowing for preliminary model selection.

Model 1. In column 1 of Table V we present estimates of the model in column 1 of Table II incorporating first-order autocorrelation of the error terms. Comparison of these results suggests this model is inappropriate for capturing the effects of pollution on morbidity. The coefficient on LSO_2 changes sign to become negative, which is contrary to a priori expectations although very imprecisely estimated. The

TABLE V

Classical Least-Squares Estimates of Eq. (2) under Alternative Model Specifications, Corrected for First- Order Autocorrelation[a]

Independent variables	Model specification			
	1	2	3	4
C	0.279	0.052	0.010	−0.006
	(0.273)	(0.244)	(0.244)	(0.300)
LSO_2	−0.064	−1.42	0.310	−0.940
	(0.060)	(0.496)	(1.47)	(0.508)
LCOH	0.185	2.05	0.951	3.79
	(0.086)	(0.41)	(0.596)	(0.817)
LAT	−0.128	−0.146	1.28	−0.156
	(0.081)	(0.073)	(0.624)	(0.072)
LARH	0.911	0.177	−0.025	−0.054
	(0.120)	(0.207)	(0.743)	(0.216)
LSC	−0.146	−0.052	−0.153	−0.037
	(0.043)	(0.044)	(0.477)	(0.042)
LSO_2LCOH		0.347	0.162	0.22
		(0.132)	(0.138)	(0.134)
LSO_2^2		−0.027	−0.033	−0.006
		(0.061)	(0.073)	(0.120)
$LCOH^2$		−0.263	0.037	−0.95
		(0.056)	(0.124)	(0.288)
$LSCLSO_2$			0.067	
			(0.114)	
LSCLCOH			0.029	
			(0.124)	
LATLCOH			−0.342	
			(0.145)	
$LATLSO_2$			0.004	
			(0.147)	
$LARHLSO_2$			−0.262	
			(0.338)	
LARHLCOH			0.014	
			(0.193)	
LSO_2^3				0.006
				(0.055)
$LCOH^3$				0.079
				(0.033)
ρ	0.60	0.65	0.76	0.71
R^2	0.79	0.85	0.88	0.86
SEE	0.28	0.24	0.23	0.23
D.W.	2.07	1.87	1.63	1.84

[a]Standard errors in parentheses.

coefficient on LCOH changes sign to become positive. The coefficients on the climatic control variables remain the same although they change in magnitude. The dramatic qualitative differences in estimated pollutant impacts on morbidity between columns 1 of Tables II and V and the fact that the effect of one pollutant is positive in each case indicate that this specification is seriously incomplete.

Model 2. In column 2 of Table V we present estimates of the model in column 2 of Table II. It is important for current concerns that the parameter estimates do not

differ greatly between the two estimation techniques with the robust nature of the estimates suggesting a reliable model specification. To the extent that the results differ, decreased nonlinearities in conjunction with smaller synergistic interaction among pollutants stem from the second estimation technique. Percent effects of single pollutants with all variables at their sample means are not substantial. However, with the other pollutant two standard deviations above its sample mean, a 1% increase in SO_2 leads to a 0.20% increase in morbidity and a 1% increase in COH leads to a 0.40% increase in morbidity. The effect of percent increases in each pollutant leads to an increase in their combined effect by over 0.10%, indicating that synergisms outweight nonlinearities in combined pollutant impacts.

Model 3. In column 3 of Table V we present estimates of the model in column 3 of Table II incorporating a first-order error autoregressive process. Estimated percentage effects of changes in climate variables on emergency hospital admissions are presented in column 2 of Table III for all variables at their sample means. In comparison with calculations presented in column 1, the effect of average relative humidity switches sign. Although the magnitude associated with effects of sky cover remains small it switches sign to become positive.

In comparison with results using classical least-squares estimation techniques, coefficient estimates in column 3 of Table V often change signs. However, estimates of the effects of pollutants on hospital admissions stem from combinations of these estimates, which taken together do not differ qualitatively from the estimates of Section III. For example, for the other pollutant two standard deviations above its sample mean, a 1% increase in SO_2 leads to a 0.07% increase in morbidity and a 1% increase in COH leads to a 0.32% increase in morbidity. While these results do not differ substantially from those based on the estimates in the third column of Table II, they depend crucially on the coefficients associated with the climatic interaction variables which are often relatively large in magnitude but quite imprecisely estimated. This suggests that variation in climatic conditions may lead to substantial but essentially unknown impacts on the pollutant effects.

Model 4. In column 4 of Table V we present parameter estimates of the model in column 4 of Table II incorporating a first-order autoregressive error structure. Parameter estimates associated with average temperature are smaller in magnitude while those for sky cover remain the same compared with the classical least-squares results. However, effects of average relative humidity (although imprecisely estimated) switch sign when one corrects for first-order autocorrelation. The coefficient on the interaction term between SO_2 and COH decreases in magnitude, while the coefficients on the squared and cubic pollution variables decrease in magnitude. The latter estimates are imprecise for SO_2 as evidenced by the high standard errors of the coefficients. However, based on the point estimates, for the other pollutant two standard errors above its means, a 1% increase in SO_2 leads to a 0.12% increase in morbidity and a 1% increase in COH leads to almost a 0.50% increase in morbidity. Percent increases in both SO_2 and COH increase their combined effects on morbidity by almost 0.56%.

Summary. The nature of effects of pollutants on morbidity based on the joint estimation of structural and error process parameters suggests that the linear model is likely a misspecification. Further, comparison of effects based on estimates in this section and Section III indicate that the qualitative nature of the results for the interaction models are not dramatically sensitive to estimation technique. However, the results differ qualitatively among alternative interaction models. This difference

is most evident between the quadratic and cubic pollution specifications where the latter suggests an increase in pollutant effects at high pollutant levels, not reflected in either quadratic specification. In addition, the quadratic model that includes pollutant–climatic interactions often yields imprecise estimates of these interactions. If these interactions are an important element in determining mobidity effects of pollutants, the imprecision of the estimates yields little in the way of guidance as to the magnitude or sign of the effects. To determine the potential importance of the various effects captured by the different interaction models we turn now to the statistical analysis of model selection.

Model Selection

Empirical results for a variety of functional forms suggest that, while extensions of a quadratic model with interactions among pollutants and climate variables do not substantially alter the form in which pollutants affect emergency hospital admissions, more complex nonlinearities among pollutants (in terms of the cubic approximation) do alter implications for measuring health impacts.

The variety of models estimated above are embedded in more complex models, the most general a cubic logarithmic Taylor expansion of (1). For current purposes it is sufficient that model 1 is a special case of model 2 which in turn is a special case of model 3 or 4. However, neither model 3 nor 4 is embedded in the other. While the implications for policy analysis of either model 2 or 3 are similar for climatic conditions at the sample mean, they differ if there is substantial climatic variation. The implications of both of these models differ from those of the cubic representation of model 4. There is a need to analyze the extent to which the data support one of these models over the others. The approach taken here is to calculate posterior odds ratios to facilitate in model comparison. Following along the lines of Zellner and Siow [10] it is desirable to present such comparisons among models under conditions where there is little prior information as to which model is appropriate.[10]

We consider two models at a time with one model not including interaction terms that are included in the other model (which makes one model a special case of the other). Equal prior probabilities on each model are used in conjunction with a Cauchy probability density function centered at zero for the parameters associated with the interaction variables not included in the simpler model. Under these conditions posterior odds ratios reveal information as to the extent to which the data suggest one model is more "probable" than another. In terms of the models presented above, an approximation to the posterior odds ratio for the simpler relative to the more complicated model derived in Zellner and Siow [10] is

$$K_{s,c} \doteq \gamma(\nu_c/2)^{k_2/2} \Big/ \left[1 + (k_2/\nu_c)F_{k_2,\nu_c}\right]^{(\nu_c-1)/2} \tag{4}$$

where $\gamma = \pi^{1/2}/\Gamma[(k_2 + 1)/2]$; k_2 is the number of additional variables in the more complicated model; $\nu_c = n - k - 1$ where k is the number of variables in the more complicated model and n is the number of observations; and F_{k_2,ν_c} is the usual F statistic. Given the sample size and number of variables in each model, $K_{s,c}$ is a

[10]This approach is useful because we may not wish to accept or reject one model over another and really have no null hypothesis to begin with.

simple function of but not proportional to the usual F statistic. As ν_c increases, a larger F statistic is required to maintain the same $K_{s,c}$ which corresponds to the lowered "significance level" applicable to F_{k_2,ν_c} as ν_c increases.

The posterior odds ratio for models 2 and 3 is over 16,000 while for models 2 and 4 it is 2.98.[11] These results imply that the model including interaction terms between pollutants and climate control variables does not fare well against the simpler model including only pollutant interaction variables. The large posterior odds ratio for model 2 relative to model 3 suggests that in the absence of any prior reason for choosing one specification over the other, model 2 dominates model 3. Since the implications for policy analysis do not differ greatly among these specifications for climatic variables at their sample mean levels, no further attention is focused on model 3.

While the odds ratio for model 2 relative to model 4 lends support to the quadratic relative to the cubic specification, the policy implications of these models differ. Moreover, the data does not lend overwhelming support to the quadratic model and it would be incomplete to merely ignore the implications of the cubic model results. For present purposes, it seems that costs of undervaluation as well as of overvaluation of health effects of pollution are similar from a policy perspective and we consider a quadratic loss function associated with evaluation of the hospitalization effects of pollution in analyzing models 2 and 4. This implies weighting the results of each model according to the posterior odds ratio.[12]

Effects of Pollutants on Hospital Admissions

In Fig. 1 we present estimates of the percentage effect on hospital admissions of a percentage change in COH for SO_2 levels at the mean and two standard deviations above its sample mean. These estimates are based on the posterior odds weighted point estimates of models 2 and 4. While partial effects decrease with higher COH levels they are exacerbated by higher SO_2 levels. Similarly, in Fig. 2 we show estimates of percentage effects of changes in SO_2 for COH levels at the sample mean and two standard deviations above the mean. Analogous qualitative results apply. These findings suggest that calculation of the benefits associated with air quality improvements is not simplistic and that the benefit associated with a particular pollutant standard is not independent of the levels of other pollutants in ambient air.

In Fig. 3 we present estimates of the percentage effect on hospital admissions of equal percentage changes in both SO_2 and COH for various levels of the two pollutants in the sample. Nonlinear impacts of each pollutant as well as synergistic interactions between pollutants are combined in these calculations. Synergisms outweigh nonlinear effects of single pollutants leading to increasing percentage impacts as both pollutants increase.

[11] For simplicity we consider the two-way comparisons as analytically developed by Zellner and Siow [10]. With a set of η comprehensive options, posterior probabilities could be calculated for each, not independent of the others. For current purposes, because the odds of model 3 relative to model 2 are extremely small and because the odds of model 2 relative to model 4 are not low, inclusion of model 3 in determination of posterior model probabilities is not likely to be of substantial importance if all three models are considered jointly.

[12] In essence, the cost associated with choosing the implications of one model over the other when the other is indeed correct is treated symmetrically with respect to each model. The choice of other loss functions would modify these results.

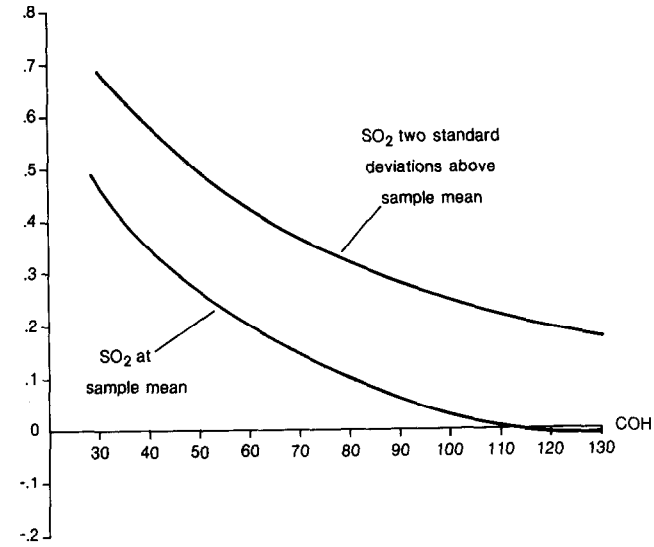

FIG. 1. Percentage effects on total cardiac and respiratory hospital admissions resulting from percentage changes in COH.

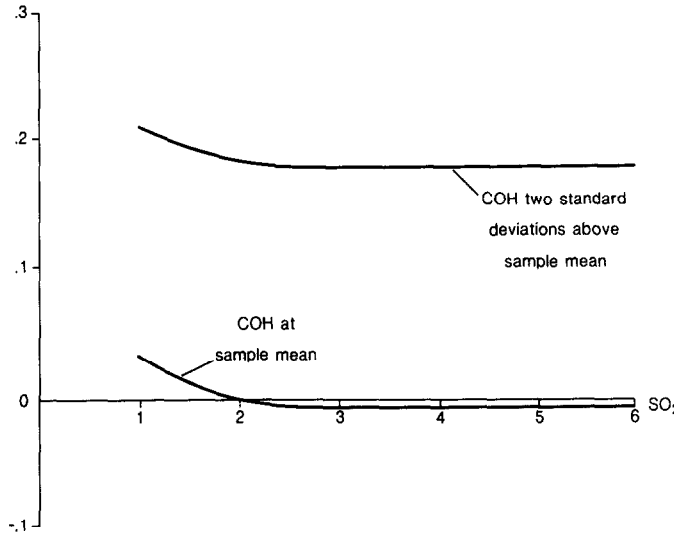

FIG. 2. Percentage effects on total cardiac and respiratory hospital admissions resulting from percentage changes in SO_2.

V. CONCLUSION AND POLICY IMPLICATIONS

The analysis presented here suggests the importance of synergisms and nonlinearities in effects of pollutants on hospital admissions. An implication, if the nature of these results holds up for other damage categories, is that marginal damage estimates that do not take these factors into account do not provide the relevant information needed for appropriate policy decision making. A further implication is that a spatial nonuniformity in control stringency is even more strongly indicated than had

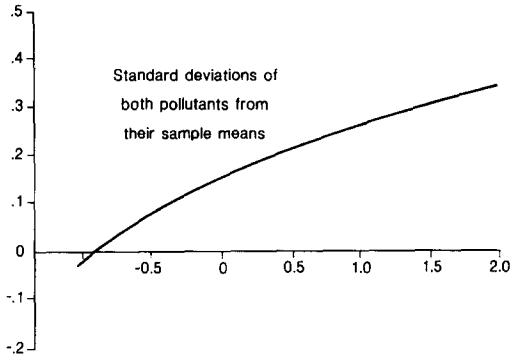

FIG. 3. Percentage effects on total cardiac and respiratory hospital admissions resulting from equal percentage changes in SO_2 and COH.

been heretofore thought. The contribution of the paper, however, lies not so much in the area of specific damages examined or the empirical findings themselves. Rather, a methodology is utilized that, when theoretical guidance regarding functional form is lacking, provides an objective approach to evaluation of alternative models (or mix of models), that have quite different policy implications.

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