STATISTICAL ANALYSIS OF DAILY LONDON MORTALITY AND ASSOCIATED WEATHER AND POLLUTION EFFECTS

by

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Abstract

The possible association between three kinds of mortality and several pollution and weather variables is investigated using a general multiple time series regression model on daily data collected during fourteen London winters spanning the time period 1958-1971. The best model for predicting overall mortality, cardiovascular mortality, or respiratory mortality involved using lagged temperature in combination with the logarithms of the same day levels of either sulfur dioxide or black smoke deposits. The pollutants are more important than temperature in predicting changes in overall and respiratory mortality but are less important in predicting cardiovascular mortality. The mechanism indicated by the regression analysis is that pollution acts positively and instantaneously, whereas temperature acts negatively with the strongest component at a lag of two days for cardiovascular mortality and positively as a function of the two-day temperature differential for overall and respiratory mortality. The strongest associations, as measured by the multiple coherence, occur at periods ranging between seven and twenty-one days, implying that pollution and temperature "episodes" must persist in order to influence mortality.

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Table of Contents

	Page
Abstract	2
Acknowledgments	3
Table of Contents	4
List of Figures	5
List of Tables	6
1. Introduction	7
2. The Analysis: Choosing the Approach	8
3. The Input-Output Regression Model	13
4. Determining the Significant Environmental Factors Contributing to Mortality: Frequency Domain Regression	16
5. Describing the Joint Contributions of Temperature and Pollution: The Partial Regression (Impulse Response) Functions	22
6. Conclusions and Recommendations	26
References	28
Appendix A: Summary of Equations Used in Frequency Domain Regression	29
Appendix B: Additional Tables Mentioned in Text	32
Appendix C: Basic London Data Used in Analysis (1958-1971)	37

List of Figures

		Page
Figure 1.	Detrended Daily London Mortality for Winter, 1970.	9
Figure 2.	Detrended Transformed Pollution and Associated Weather Series for the London Winter of 1970.	10
Figure 3.	Impulse and Frequency Response Functions for Two Possible Prefilters	11
Figure 4.	Filtered (high-Pass), Detrended Daily London Mortality for the Winter of 1970.	12
Figure 5.	Filtered, Detrended Cardiovascular Mortality and Associated SO_2 and Temperature Levels for the Winter 1962.	14
Figure 6.	Cross-Correlation Functions Relating Cardiovascular Mortality to SO_2 Levels (log) and Temperature for the Winter of 1962.	15
Figure 7.	Partial Regression Functions (Lagged) Relating Total Mortality to Transformed SO_2 and Temperature for All 14 London Winters.	23
Figure 8.	Partial Regression Functions (Lagged) Relating Cardiovascular Mortality to Transformed SO_2 and Temperature for All 14 London Winters.	24
Figure 9.	Partial Regression Functions (Lagged) Relating Respiratory Mortality to Transformed SO_2 and Temperature for All 14 London Winters.	25

List of Tables

Page

Table 1: Values of Multiple Coherence R^2 Relating Total Mortality to Lagged Regression Models Involving Black Smoke (4), SO_2 (5), Temperature (6), and Relative Humidity (7) for Pooled 14-Year London Data (*indicates significance at $\alpha = .01$ level). 18

Table 2: Values of Multiple Coherence R^2 Relating Cardiovascular Mortality to Lagged Regression Models Involving Black Smoke (4), SO_2 (5), Temperature (6), and Relative Humidity (7) for Pooled 14-Year London Data (*indicates significance at $\alpha = .01$ level). 19

Table 3: Values of Multiple Coherence R^2 Relating Respiratory Mortality to Lagged Regression Models Involving Black Smoke (4), SO_2 (5), Temperature (6), and Relative Humidity (7) for Pooled 14-Year London Data (*indicates significance at $\alpha = .01$ level). 20

Table 4: Residual Analysis of Power for Models Involving Total Mortality (1), Cardiovascular Mortality (2), and Respiratory Mortality (3) as Outputs and Black Smoke (5), Temperature (6), and Relative Humidity (7) as Inputs (*indicates significance at $\alpha = .01$ level). 21

Table B1: Power Spectra of Prefiltered London Data-- Total Mortality (1), Cardiovascular Mortality (2), Respiratory Mortality (3), Black Smoke (4), SO_2 (5), Temperature (6), and Relative Humidity (7). 33

Table B2: Partial Regression Functions Relating Total Mortality to SO_2 and Temperature (see Figure 7). 34

Table B3: Partial Regression Functions Relating Cardiovascular Mortality to SO_2 and Temperature (see Figure 8). 35

Table B4: Partial Regression Functions Relating Respiratory Mortality to Transformed SO_2 and Temperature (see Figure 9). 36

1. Introduction

The investigation of a possible association between pollution levels and short- or long-term adverse health effects in the presence of other confounding environmental factors is a problem of great interest. While it is important to be able to quantify any dose response relation which may exist, it is also essential to be able to account properly for other environmental factors such as temperature and relative humidity. The purpose of this investigation is to focus on modeling short-term mortality fluctuations in terms of pollution and weather effects using a limited data base consisting of daily measurements made over fourteen winters in the immediate vicinity of London.

The London mortality and air quality data have been used previously in attempts to quantify a dose response relation between pollution and daily mortality. A review of a number of such studies, including a groundbreaking British contribution by Martin and Bradley (1960), is given in Ware, et al., (1981) who describe the results in terms of standard regressions relating instantaneous 24-hour mortality to the primary pollutants, black smoke level (BSM) and sulfur dioxide (SO_2), measured in $\mu g/m^3$. This also characterizes the work of Mazumdar, et al., (1980), (1983) using techniques developed by Schimmel (1978) for data collected in New York City.

In all of the above studies, a minimal amount of time series methodology was employed which was confined mainly to eliminating the low frequency "seasonal" effect by using a suitable prefilter; in most cases, the excess over a 15-point moving average was used. A first effort towards resolving some of the lagged effects present in the London data was made by Dawson and Brown (1981) who assumed an autoregressive structure for mortality and found a reasonable correlation with black smoke levels. Wyzga (1978) investigated some specific distributed lag models relating lagged pollution and filtered temperature to daily mortality in Philadelphia.

In this report we will develop a multiple time series model which allows one to estimate an arbitrary lag structure subject to correlation over time. This provides more specific information as to what kinds of pseudo-causal mechanisms might be inferred. The analysis detailed a consideration of (1) temporal patterns in the data including potential lagged effects with pollutants and with temperature and relative humidity, (2) data from more than a single year (examples are Mazumdar, et al., (1980) and Wyzga (1978)), (3) mortality stratified by cause, and (4) possible nonlinear relationships. Furthermore, a stepwise procedure is used to determine which factors contribute significantly to daily mortality fluctuations. Since the analysis is done in the frequency domain, the periods over which the relationship is strongest can be uniquely identified. Although the London data set can still be criticized on the basis that it represents a limited population monitored under rather restrictive environmental conditions, we are still able to extend the analysis to fourteen winters, with mortality stratified according to a limited set of causes.

2. The Analysis: Choosing the Approach

The initial raw data used in this analysis involve overall mortality, cardiovascular mortality, and respiratory mortality measured along with the levels of the two primary pollutants, black smoke (BSM) and sulfur dioxide (SO_2), and two weather series, temperature and relative humidity. The temperature (in degrees C) and relative humidity (percent) are measured at 9:00 a.m. each day in the Metropolitan Office. The smoke and SO_2 levels are the means of seven sites measured in $\mu g/m^3$. These seven series are measured over 112 days, beginning at the forty-eighth week of the current year and ending at the fifth week of the succeeding year, for each of fourteen London winters spanning the years 1958-1971.

The basic raw data over all fourteen winters are plotted in Appendix C. A number of qualitative observations can be made by examining these plots more closely. First of all, the mortality plots have linear trends and in some years will have a low frequency component corresponding to an epidemic. Detrending does not eliminate the low frequency component as can be noted from the plot in Figure 1 which shows the mortality data measured during the winter of 1970. This indicates that a prefilter which eliminates the low frequency should be applied at some stage. The pollution series shown in Appendix C also have a non-stationary appearance which is induced primarily by the occurrence of high level episodes. The idea of transforming to logarithms is appealing for two reasons. First, the transformed data, say for 1970 shown in Figure 2, have a distinctly stationary character which is more consistent with the magnitude of the short-term fluctuations of the mortality series. Secondly, the classical nonlinear dose response relation is often taken to be linear in the logarithms (cf. Ware, et al., (1981) and Dawson, et al., (1981)). The daily temperature and relative humidity data shown in Figure 2 are visually consistent (up to a linear trend) with patterns to be expected with stationary data.

Since linear detrending will not be a completely satisfactory way of eliminating the non-stationarity due to the extremely low frequency components in the years 1961, 1963, 1967, 1969, and 1970 (see Appendix C), we designed a symmetric (phaseless) prefilter with a suitable response. This filtering has been done in the past by subtracting a 15-point moving average from each data value. However, the frequency response of this filter, shown in Figure 3, has some undesirable "ripples" over parts of the high frequency range. A filter designed specifically to reject low frequencies and pass high frequencies is also shown in Figure 3, and we note that a flat response can be achieved by suitably modifying the off-center coefficients. In this analysis, we have detrended each year to eliminate the changes in overall levels due to year-to-year longer term fluctuations. The effects of epidemics over the same year are taken out by filtering. For example, Figure 4 shows the detrended filtered mortality data for the winter of 1970 and the effect of the long-term rise in midwinter has clearly been eliminated.

The preceding arguments and discussion are intended as general support for the notion that each year, after suitable detrending and transformation, we are observing what can essentially be described as a stationary multiple time series and that the years are replicates of this basic underlying series which can be described by the input-output regression model to be described in the next section.

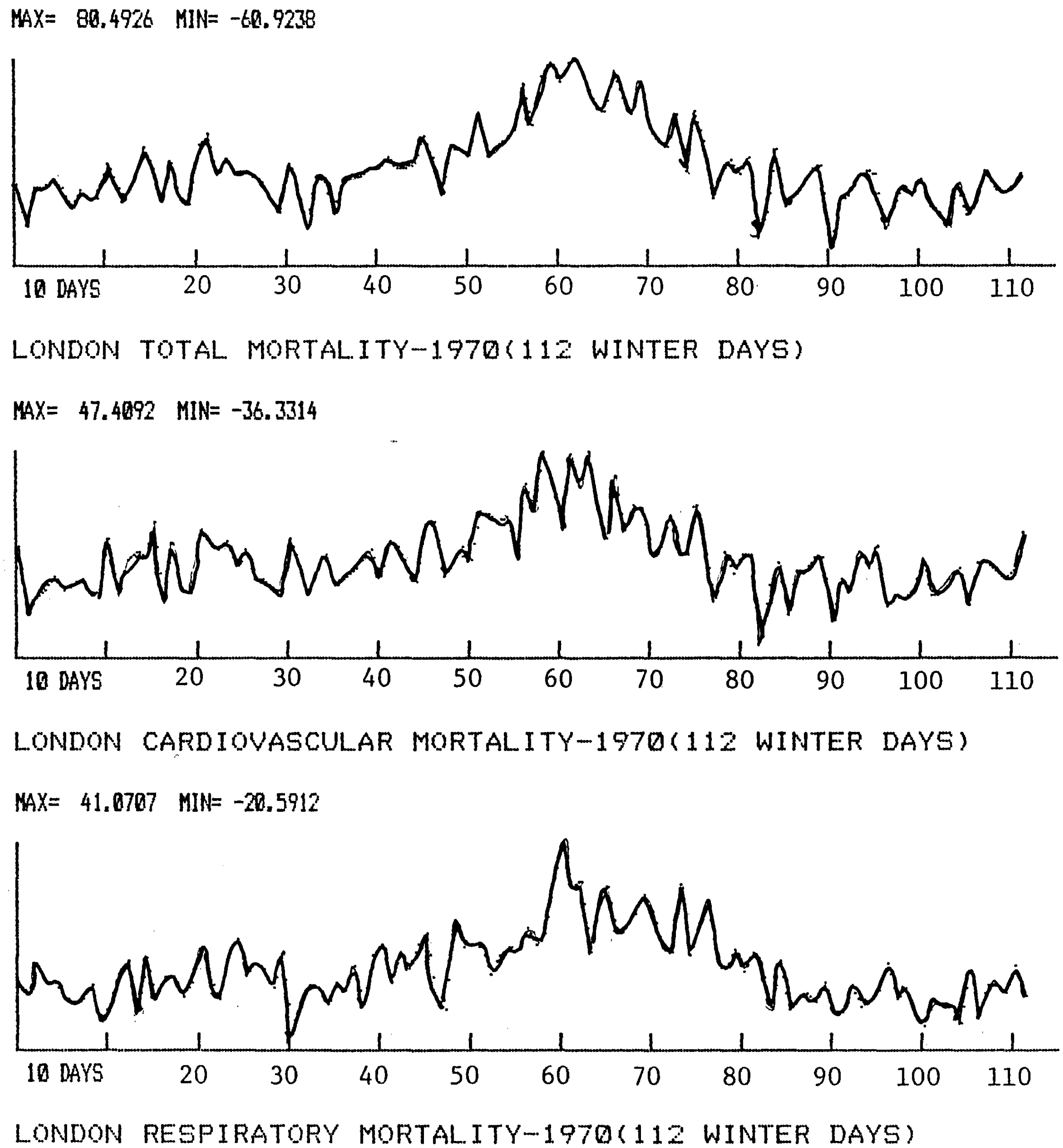


FIGURE 1. Detrended Daily London Mortality for Winter, 1970.

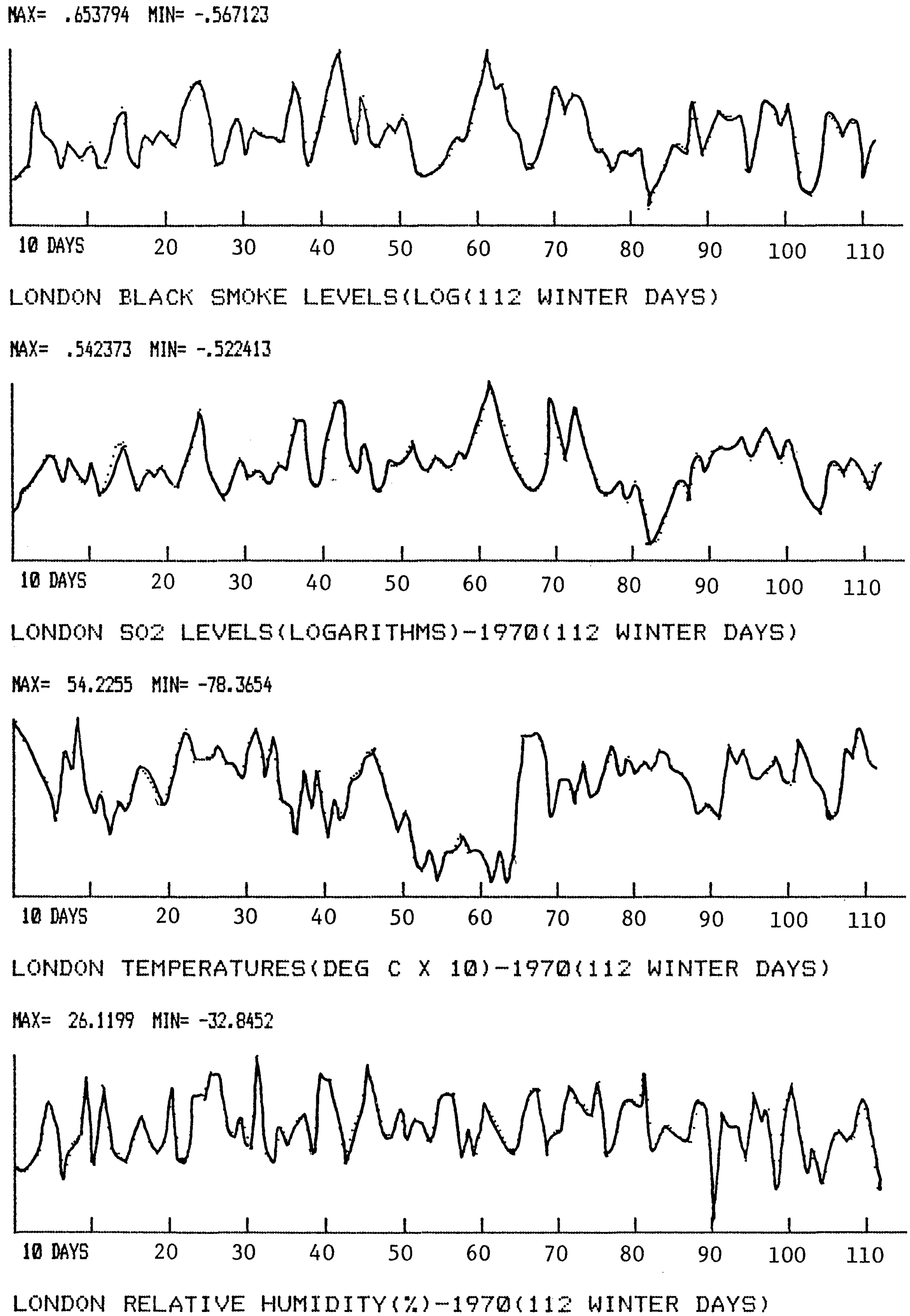
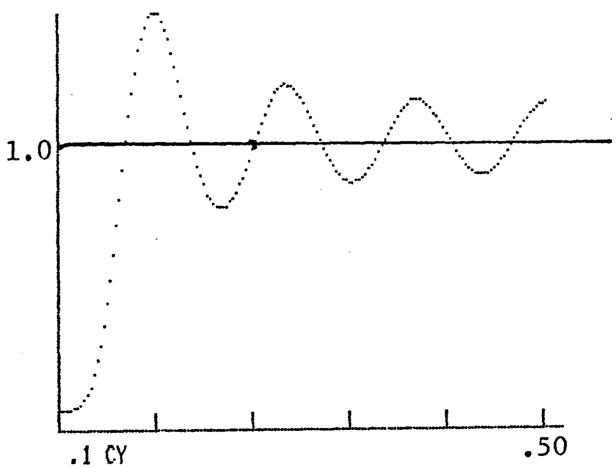


FIGURE 2. Detrended Transformed Pollution and Associated Weather Series for the London Winter of 1970.

SERIES ADJUSTED BY SUBTRACTING 15-POINT FILTER COEFFICIENTS

0	.933
1	-.067
2	-.067
3	-.067
4	-.067
5	-.067
6	-.067
7	-.067

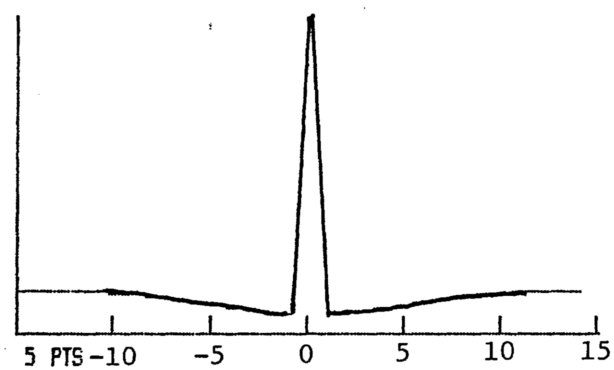
MAX= 1.48064 MIN= 2.49997E-05



FREQUENCY RESPONSE OF FILTER

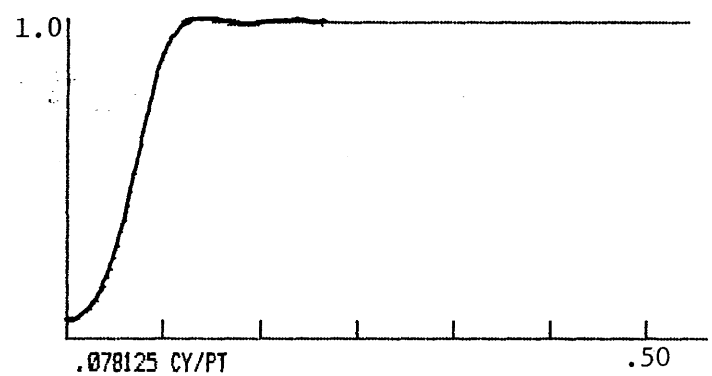
0	.921875
1	-.0765822
2	-.0721008
3	-.065104
4	-.056237
5	-.0462868
6	-.0360857
7	-.0264127
8	-.0179057
9	-.0109979

MAX= .921875 MIN= -.0765822



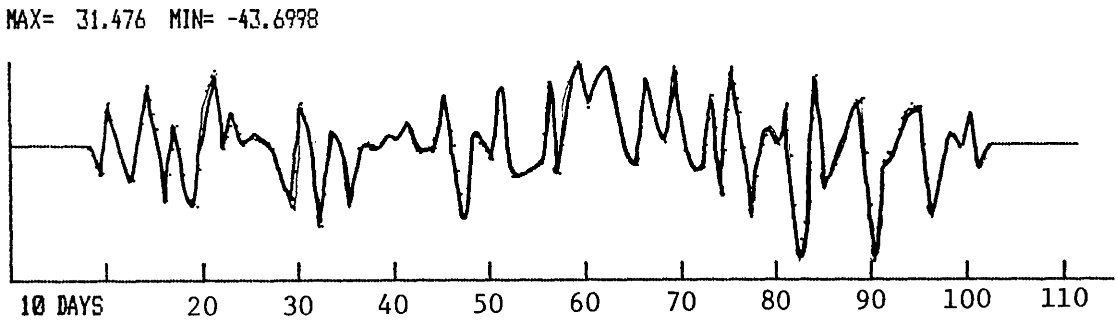
TIME VERSION OF FILTER

MAX= 1.01365 MIN= 7.89228E-03

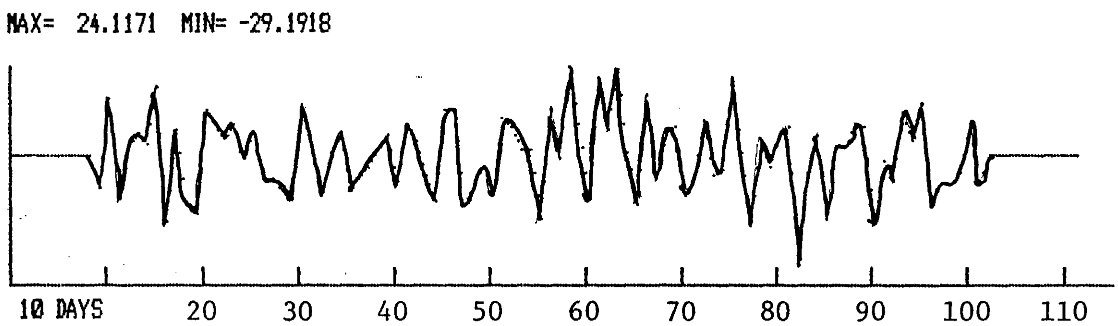


FREQUENCY RESPONSE OF FILTER

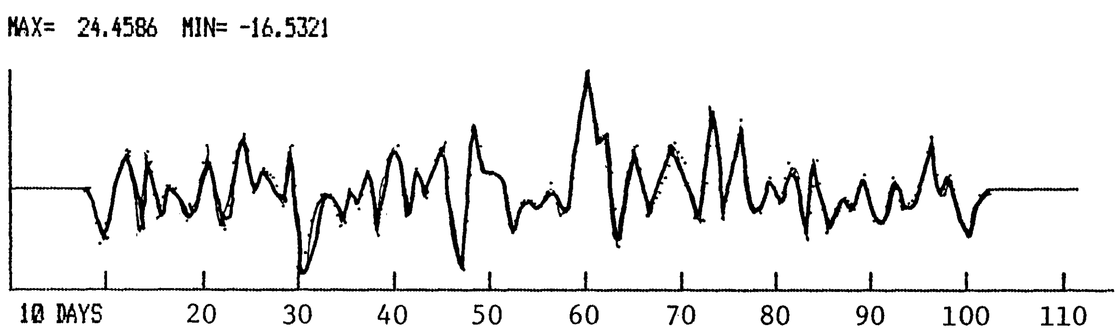
FIGURE 3. Impulse and Frequency Response Functions for Two Possible Prefilters.



FILTERED TOTAL MORTALITY- LONDON WINTER(1970)



FILTERED CARDIOVASCULAR MORTALITY- LONDON WINTER(1970)



FILTERED RESPIRATORY MORTALITY- LONDON WINTER(1970)

FIGURE 4. Filtered (High-Pass), Detrended Daily London Mortality for the Winter of 1970.

3. The Input-Output Regression Model

The assumption that we are dealing with replicates of multiple time series, with the input series taken as the pollution and weather traces and the output series as mortality, implies that we may consider some sort of regression model relating the two. In order to motivate this further we consider 1962 as a year in which there were generally high pollution levels associated with high mortality values and some dynamic variations in temperature levels. For example, at the thirtieth day a high mortality value is associated with high pollution and relative humidity combined with a low temperature value. The same phenomenon occurs to a somewhat lesser extent at the eightieth day. The sample cross-correlation[1] functions shown in Figure 6 confirm the strength of the correlation between pollution and cardiovascular mortality which is maximized (0.62) at lag zero. Cardiovascular mortality is correlated negatively with temperature at a lag of two days. This indicates that a change in temperature tends to precede a change in mortality by about two days for 1962.

The time series regression model provides a basis for investigating possible lagged relations between a collection of p stationary input series $x_{j1}(t),\ldots,x_{jp}(t)$ and a stationary output series $y_j(t)$ for $j=1,2,\ldots,N$ years. In the case under consideration, $y_j(t)$ is one of the mortality series measured on the t^{th} day of the j^{th} winter and $(x_{j1}(t),\ x_{j2}(t),\ x_{j3}(t),\ x_{j4}(t))$ are the input series $\ell n(\mathrm{BSM})$, $\ell n(\mathrm{SO_2})$, temperature, and relative humidity measured at corresponding times. Then, assume that the input and output series are related by the regression model

$$y_j(t) = \sum_{k=1}^{p} x_{jk}(t) * \beta_k(t) + e_j(t) \tag{3.1}$$

where * denotes the convolution

$$x_{jk}(t) * \beta_k(t) = \sum_{u=-\infty}^{\infty} x_{jk}(t-u)\beta_k(u) \tag{3.2}$$

and $e_j(t)$, $j=1,\ldots,N$ are a collection of independent identically distributed stationary time series with a common autocorrelation function

$$R(m) = E(e_j(t+m)e_j(t)) \tag{3.3}$$

for $j=1,\ldots,N$. This model which regards the inputs as fixed is equivalent to

---

[1]The cross correlation between two detrended time series $z_1(t)$ and $z_2(t)$ at lag m is defined as

$$r_{12}^T(m) = \frac{R_{12}^T(m)}{\sqrt{R_{11}^T(0)R_{22}^T(0)}}$$

where

$$R_{12}^T(m) = \frac{1}{T}\sum_{t=1}^{T-m} z_1(t+m)z_2(t)$$

with $R_{12}^T(m) = R_{21}^T(-m)$ at $m=0,\pm1,\ldots,\pm M$ lags.

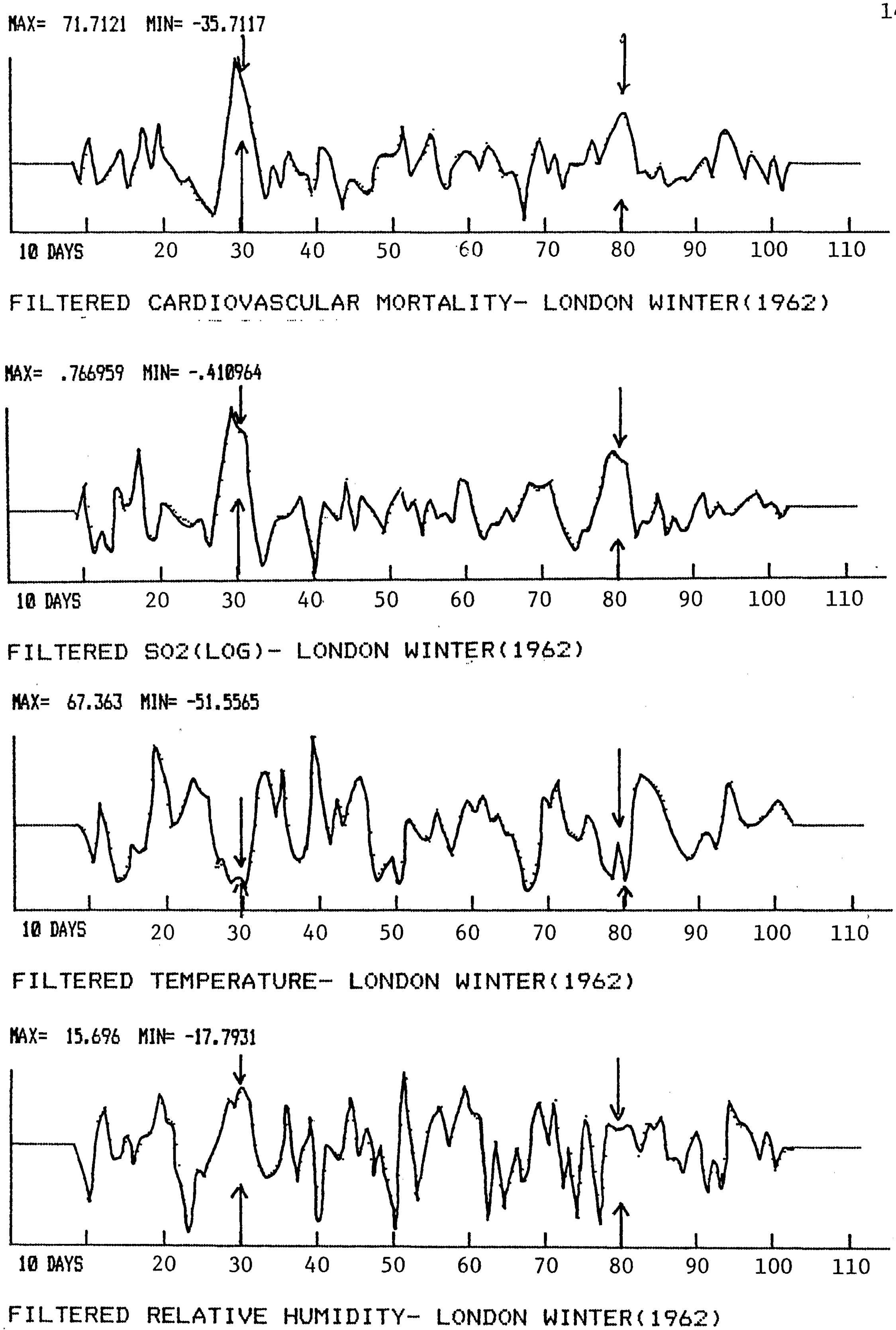
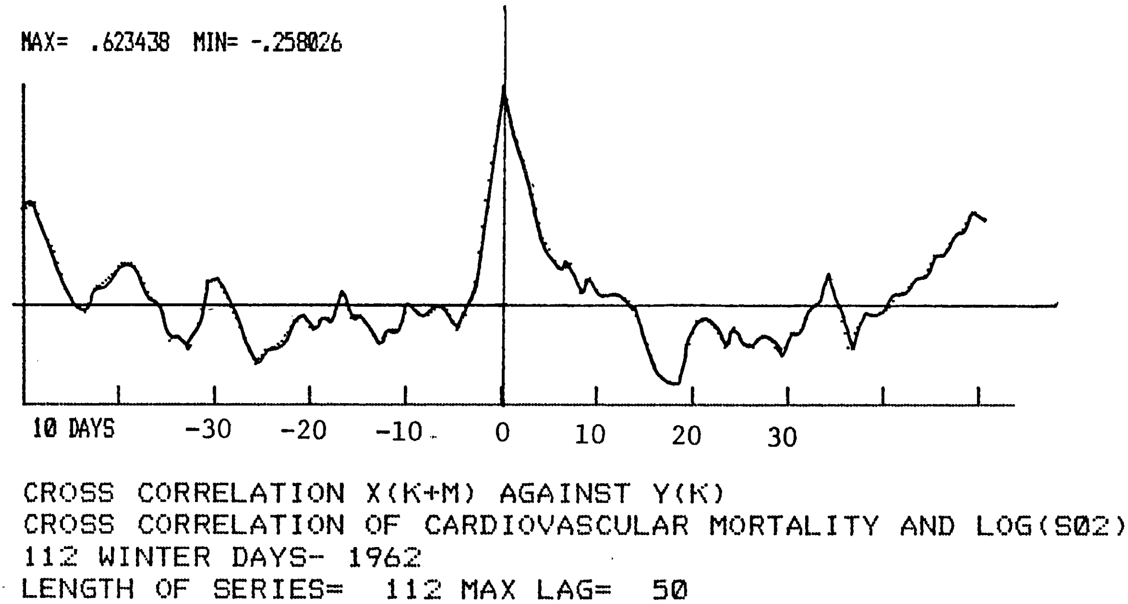


FIGURE 5. Filtered, Detrended Cardiovascular Mortality and Associated SO_2 and Temperature Levels for the Winter of 1962.



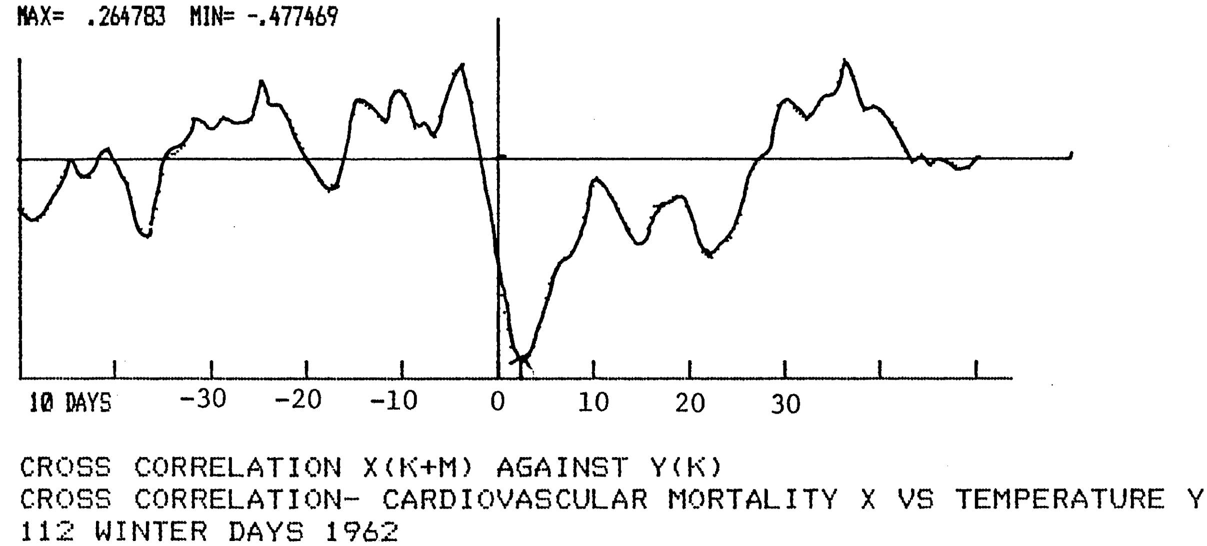


FIGURE 6. Cross-Correlation Functions Relating Cardiovascular Mortality to SO_2 Levels (log) and Temperature for the Winter of 1962.

performing the analysis on the jointly stationary inputs and outputs conditionally on the fixed inputs. We will be interested in estimating the regression functions $\beta_k(t)$ relating the k^{th} input series to the output $y_j(t)$. The model includes the usual instantaneous regression model as a special case since by replacing convolution * by multiplication we obtain

$$y_j(t) = \sum_{k=1}^{p} x_{jk}(t)\beta_k + e_j(t), \tag{3.4}$$

where $e_j(t)$ is now assumed to be independent over time as well so that $R(m) = \sigma^2$ for $m = 0$ and is zero otherwise. The restrictions inherent in this usual model are evident as the β_k does not vary over time, precluding the estimation of lagged effects, and the error is assumed to be independently distributed over time.

The basic problems to be considered for the time series regression model (3.1) are (a) the determination of the significant environmental contributors to mortality and (b) the estimation of the partial regression (impulse response) functions $\beta_k(t)$ for the best model. An arbitrary lagged regression is assumed, so that assumptions are not made about causality.

The analysis is based on the ability to transform all the inputs and outputs to stationary time series by detrending or transformations. This enables us to perform the analysis in the frequency domain using the properties of the discrete Fourier transform (DFT). Another advantage of the frequency domain is that we can isolate the primary coherent period band; the harmonic component may correspond to an identifiable causal phenomenon such as a periodic release of high concentration pollutants.

4. Determining the Significant Environmental Factors Contributing to Mortality: Frequency Domain Regression

In order to determine the joint contributions of the various environmental factors influencing mortality in the lagged regression relation (3.1), we consider first the analog of the standard measure, the squared multiple correlation at frequency ν, say $R^2(\nu)$ as defined in equation (A10) of Appendix A. The values of $R^2(\nu)$, called the multiple coherence function, can be interpreted as the percentage of power accounted for by the given model at a specified frequency ν. The frequency ν is defined in terms of the number of cycles the time series makes in a given unit of time. For example, a series which has a strong component at the $\nu_0 = 0.10$ cycles per day takes $1/0.10 = 10$ days to complete one cycle. One may therefore also quote the regression results in terms of the period, say $T_0 = 1/\nu_0$, which is the length of time needed to complete one full cycle.

It is possible to examine the contributions that each band of frequencies makes to the variance for each series by looking at the power spectra given in Table B1 of Appendix B. The spectra indicate that the frequencies contributing the maximum power are centered on 0.09 cycles per day or a period of about eleven days. The spectrum is an average of the variance over the band from 0.078 to 0.109 cycles per day which corresponds to periods between 9.1 and 12.8 days.

The values of $R^2(\nu)$ for a number of combinations of inputs as related to the possible outputs, overall mortality (Table 1), cardiovascular mortality (Table 2), and respiratory mortality (Table 3) are shown on the next pages. In general, a number of models involving single inputs ($p = 1$), two inputs ($p = 2$), three inputs ($p = 3$), and four inputs ($p = 4$) were studied. The critical values against which R^2 may be compared at $\alpha = 0.01$ are 0.11, 0.15, 0.19, and 0.21, for the $p = 1,2,3$, and 4 cases, respectively.

Table 1 shows that either of the two pollution series or temperature may be considered as primary single contributors to total mortality over a number of frequencies corresponding to seven to 21 days and over the 3.4- to 5-day band for temperature. Table 1 also indicates that overall mortality is predicted best by a model using one of the two pollution values in combination with temperature. Either of the two models ((4,6) or (5,6) in the table) accounts for over 50 percent of the power in the frequency range 0.06 to 0.09 cycles per year corresponding to periods of seven to 21 days. This is somewhat more than can be accounted for in a model any one of the three inputs singly, and no single input appears to be an improvement over the other two. The effect on mortality of the two pollutants appears to be identical in terms of predictable power. The coherence between ℓn(BSM) and $\ell n(SO_2)$ was uniformly high (> 0.80) for all years, bearing out the interpretation that they are acting identically or as surrogates for some other unmeasured phenomenon.

The results for cardiovascular mortality in Table 2 are not as strong but still indicate that over 40 percent of the power can be accounted for in the frequency band 0.03 to 0.09 cycles per day by a model including temperature and one of the two pollutants. A surprising aspect of the cardiovascular mortality is that temperature (6) appears as the best single contributor, accounting for 20 to 40 percent of the power in a somewhat lower frequency band 0.03 to 0.09 cycles per year corresponding approximately to ten- to thirty-day periods.

The respiratory mortality results in Table 3 present a similar picture, although the primary band of interest corresponds to nine- to 21-day periods.

It is clear from Tables 1, 2, and 3 that, in terms of accountable power, the combination of either pollutant with temperature causes the primary effect in a period band corresponding approximately to ten-day cycles. In order to arrive at this model through a more formal statistical analysis, consider the use of F-tests to compare various models, as described in Shumway (1976) or Brillinger (1979). These tests, using equation (A17) in Appendix A, are shown in Table 4 and indicate that if one were to test formally the two-input models against the single input models, they are significantly better in the frequency band 0.06-0.13 cycles per day corresponding to eight- to seventeen-day periods. Furthermore, the models using three or four inputs as predictors are not significantly better than the two-input models.

It is reasonable to comment further on the frequency dependent nature of the relationship between pollution, temperature, and mortality. Since the relation is strongest at seven to 21-day periods, oscillations at higher frequencies in pollution levels will not lead to significant increases in mortality levels. This can be noted in Figure 8 where the low frequency oscillations around the thirtieth and eightieth day in $\ell n(SO_2)$ and temperature have discernible effects on mortality, whereas higher frequency oscillations occurring elsewhere can be tolerated. Since the results in Tables 1 and 2

Model	Frequency (Cycles/Day)																
	0.00	0.03	0.06	0.09	0.13	0.16	0.19	0.22	0.25	0.28	0.31	0.34	0.38	0.41	0.44	0.47	0.50
1 vs 4	0.03	0.09	0.32*	0.37*	0.17*	0.20*	0.12*	0.10	0.16*	0.08	0.13*	0.06	0.08	0.15*	0.19*	0.15*	0.10
1 vs 5	0.03	0.15*	0.36*	0.32*	0.17*	0.19*	0.11*	0.05	0.16*	0.11*	0.09	0.07	0.06	0.12*	0.15*	0.07	0.03
1 vs 6	0.01	0.33*	0.36*	0.30*	0.14*	0.09	0.03	0.21*	0.15*	0.12*	0.05	0.03	0.09	0.00	0.01	0.01	0.01
1 vs 7	0.03	0.08	0.20*	0.12*	0.01	0.03	0.07	0.05	0.07	0.09	0.06	0.02	0.00	0.03	0.01	0.09	0.00
1 vs 4,5	0.03	0.16*	0.39*	0.39*	0.19*	0.21*	0.14	0.14	0.17*	0.11	0.14	0.08	0.08	0.17*	0.19*	0.16	0.12
1 vs 4,6	0.04	0.35*	0.51*	0.53*	0.32*	0.27*	0.22*	0.35*	0.26*	0.19*	0.23*	0.13	0.12	0.16*	0.20*	0.16*	0.10
1 vs 4,7	0.06	0.16*	0.41*	0.39*	0.19*	0.21*	0.14	0.12	0.20*	0.15*	0.15*	0.08	0.08	0.16*	0.19*	0.21*	0.10
1 vs 5,6	0.03	0.36*	0.52*	0.52*	0.37*	0.26*	0.21*	0.31*	0.29*	0.26*	0.19*	0.15*	0.12	0.13	0.16*	0.08	0.03
1 vs 5,7	0.06	0.23*	0.45*	0.34*	0.18*	0.21*	0.14	0.08	0.21*	0.19*	0.11	0.08	0.07	0.14	0.16*	0.15*	0.03
1 vs 6,7	0.05	0.38*	0.48*	0.35*	0.15*	0.11	0.10	0.23*	0.18*	0.15*	0.11	0.05	0.09	0.04	0.02	0.10	0.01
1 vs 4,5,6	0.04	0.37*	0.53*	0.54*	0.38*	0.28*	0.25*	0.36*	0.30*	0.27*	0.23*	0.17	0.13	0.18	0.20*	0.17	0.13
1 vs 4,5,7	0.06	0.24*	0.46*	0.40*	0.21*	0.22*	0.16	0.16	0.22*	0.19*	0.15	0.10	0.08	0.18	0.19*	0.22*	0.14
1 vs 4,6,7	0.07	0.39*	0.55*	0.53*	0.33*	0.27*	0.24*	0.35*	0.28*	0.23*	0.24*	0.14	0.12	0.17	0.20*	0.21*	0.11
1 vs 5,6,7	0.06	0.40*	0.56*	0.53*	0.38*	0.28*	0.24*	0.31*	0.31*	0.29*	0.21*	0.16	0.12	0.15	0.17	0.16	0.03
1 vs 4,5,6,7	0.07	0.41*	0.56*	0.54*	0.38*	0.29*	0.27*	0.36*	0.31*	0.30*	0.24*	0.19	0.13	0.19	0.20	0.22*	0.15

Table 1: Values of Multiple Coherence R^2 Relating Total Mortality to Lagged Regression Models Involving Black Smoke (4), SO_2 (5), Temperature (6), and Relative Humidity (7) for Pooled 14-Year London Data (*indicates significance at α = .01 level).

Model	Frequency (Cycles/Day)																
	0.00	0.03	0.06	0.09	0.13	0.16	0.19	0.22	0.25	0.28	0.31	0.34	0.38	0.41	0.44	0.47	0.50
2 vs 4	0.03	0.10	0.15*	0.22*	0.13*	0.10	0.06	0.03	0.03	0.06	0.04	0.07	0.05	0.12*	0.13*	0.10	0.04
2 vs 5	0.02	0.16*	0.20*	0.19*	0.20*	0.13*	0.04	0.00	0.05	0.11*	0.04	0.11*	0.03	0.10	0.09	0.05	0.00
2 vs 6	0.06	0.41*	0.28*	0.21*	0.07	0.10	0.01	0.13*	0.01	0.02	0.01	0.00	0.07	0.03	0.02	0.00	0.00
2 vs 7	0.02	0.08	0.09	0.06	0.00	0.00	0.03	0.01	0.07	0.03	0.04	0.02	0.01	0.03	0.02	0.04	0.00
2 vs 4,5	0.03	0.18*	0.23*	0.22*	0.20*	0.13	0.06	0.12	0.06	0.12	0.05	0.11	0.07	0.17*	0.13	0.11	0.07
2 vs 4,6	0.07	0.42*	0.31*	0.30*	0.18*	0.15*	0.13	0.20	0.03	0.07	0.08	0.07	0.08	0.15*	0.17*	0.11	0.05
2 vs 4,7	0.04	0.20*	0.21*	0.25*	0.14	0.10	0.07	0.03	0.10	0.08	0.06	0.08	0.05	0.13	0.14	0.13	0.05
2 vs 5,6	0.06	0.42*	0.32*	0.29*	0.26*	0.18*	0.10	0.16*	0.06	0.11	0.09	0.14	0.08	0.13	0.12	0.06	0.01
2 vs 5,7	0.03	0.26*	0.26*	0.20*	0.20*	0.13	0.05	0.01	0.12	0.13	0.06	0.12	0.04	0.12	0.11	0.09	0.01
2 vs 6,7	0.09	0.45*	0.33*	0.23*	0.07	0.11	0.05	0.13	0.07	0.04	0.05	0.02	0.08	0.07	0.04	0.04	0.01
2 vs 4,5,6	0.08	0.43*	0.33*	0.32*	0.27*	0.18	0.13	0.24*	0.06	0.13	0.10	0.14	0.13	0.18	0.18	0.12	0.07
2 vs 4,5,7	0.04	0.28*	0.27*	0.25*	0.20*	0.14	0.07	0.13	0.13	0.14	0.07	0.13	0.08	0.18	0.15	0.14	0.09
2 vs 4,6,7	0.10	0.47*	0.34*	0.31*	0.18	0.16	0.14	0.20	0.11	0.08	0.10	0.09	0.10	0.16	0.18	0.14	0.06
2 vs 5,6,7	0.09	0.47*	0.35*	0.29*	0.26*	0.18	0.12	0.16	0.13	0.13	0.11	0.15	0.09	0.15	0.14	0.09	0.01
2 vs 4,5,6,7	0.11	0.48*	0.36*	0.33*	0.27*	0.19	0.14	0.24*	0.14	0.14	0.11	0.16	0.13	0.20	0.19	0.14	0.09

Table 2: Values of Multiple Coherence R^2 Relating Cardiovascular Mortality to Lagged Regression Models Involving Black Smoke (4), SO_2 (5), Temperature (6), and Relative Humidity (7) for Pooled 14-Year London Data (*indicates significance at α = .01 level).

Model	Frequency (Cycles/Day)																
	0.00	0.03	0.06	0.09	0.13	0.16	0.19	0.22	0.25	0.28	0.31	0.34	0.38	0.41	0.44	0.47	0.50
3 vs 4	0.01	0.05	0.34*	0.32*	0.08	0.08	0.03	0.07	0.06	0.03	0.04	0.03	0.02	0.01	0.02	0.02	0.01
3 vs 5	0.01	0.05	0.31*	0.29*	0.09	0.10	0.01	0.06	0.05	0.04	0.02	0.01	0.01	0.01	0.03	0.01	0.02
3 vs 6	0.01	0.07	0.24*	0.26*	0.03	0.04	0.01	0.07	0.09	0.15*	0.01	0.00	0.03	0.00	0.02	0.09	0.00
3 vs 7	0.01	0.04	0.19*	0.11*	0.00	0.04	0.04	0.08	0.04	0.03	0.01	0.01	0.05	0.04	0.02	0.00	0.00
3 vs 4,5	0.02	0.06	0.38*	0.33*	0.10	0.10	0.03	0.07	0.07	0.04	0.05	0.05	0.02	0.04	0.03	0.03	0.02
3 vs 4,6	0.02	0.11	0.46*	0.44*	0.10	0.12	0.04	0.16*	0.11	0.16*	0.08	0.04	0.04	0.01	0.04	0.11	0.01
3 vs 4,7	0.02	0.07	0.41*	0.34*	0.08	0.12	0.05	0.12	0.09	0.05	0.04	0.04	0.06	0.04	0.04	0.02	0.01
3 vs 5,6	0.01	0.11	0.42*	0.44*	0.11	0.14	0.03	0.16*	0.12	0.16*	0.05	0.03	0.04	0.01	0.04	0.10	0.02
3 vs 5,7	0.02	0.07	0.40*	0.31*	0.09	0.15*	0.04	0.12	0.08	0.05	0.03	0.03	0.06	0.05	0.04	0.01	0.02
3 vs 6,7	0.02	0.11	0.37*	0.30*	0.03	0.08	0.05	0.13	0.12	0.15*	0.02	0.01	0.06	0.04	0.04	0.09	0.00
3 vs 4,5,6	0.02	0.11	0.46*	0.44*	0.12	0.14	0.05	0.17	0.13	0.16	0.08	0.07	0.05	0.04	0.04	0.11	0.02
3 vs 4,5,7	0.02	0.09	0.44*	0.35*	0.10	0.15	0.06	0.13	0.10	0.06	0.05	0.06	0.06	0.07	0.05	0.03	0.02
3 vs 4,6,7	0.03	0.13	0.49*	0.44*	0.10	0.16	0.07	0.20*	0.13	0.16	0.08	0.05	0.08	0.04	0.05	0.11	0.01
3 vs 5,6,7	0.02	0.13	0.46*	0.44*	0.11	0.19	0.06	0.21*	0.14	0.17	0.06	0.03	0.08	0.05	0.05	0.10	0.02
3 vs 4,5,6,7	0.03	0.13	0.49*	0.45*	0.12	0.19	0.07	0.21	0.15	0.17	0.08	0.07	0.08	0.07	0.06	0.11	0.02

Table 3: Values of Multiple Coherence R^2 Relating Respiratory Mortality to Lagged Regression Models Involving Black Smoke (4), SO_2 (5), Temperature (6), and Relative Humidity (7) for Pooled 14-Year London Data (*indicates significance at α = .01 level).

Model	Residual Power Frequency (Cycles/Day) .06	.09	.13	Null Hypothesis F-Test	F-Value Frequency (Cycles/Day) .06	.09	.13
1 vs 4	508	643	385				
1 vs 5	480	694	387				
1 vs 6	479	708	399				
1 vs 4,6	368	475	317	1 vs 4	15.59*	14.50*	8.79*
1 vs 5,6	357	485	292	1 vs 5	14.12*	17.67*	13.33*
1 vs 4,6,7	338	473	312	1 vs 4,6	3.55	.17	.64
1 vs 5,6,7	327	482	290	1 vs 5,6	3.67	.25	.28
1 vs 4,5,6,7	326	468	285	1 vs 4,6	2.57	.30	2.25
2 vs 6	117	189	137				
2 vs 4,6	112	168	121	2 vs 6	1.80	5.125*	5.42*
2 vs 5,6	110	170	109	2 vs 6	2.61	4.582	10.53*
2 vs 6,7	108	185	137	2 vs 6	3.42	.89	.00
2 vs 4,6,7	106	165	121	2 vs 4,6	2.26	.73	.00
2 vs 5,6,7	105	169	109	2 vs 5,6	1.90	.24	.00
2 vs 4,5,6,7	104	160	108	2 vs 4,6	1.54	1.00	2.41
3 vs 4	81	89	69				
3 vs 5	85	93	69				
3 vs 4,6	67	73	68	3 vs 4	8.57*	8.99*	.60
3 vs 5,6	71	74	67	3 vs 5	8.08*	10.53*	1.22
3 vs 4,6,7	63	73	68	3 vs 4,6	2.53	.00	.00
3 vs 5,6,7	66	73	67	3 vs 5,6	3.03	.55	.00
3 vs 4,5,6,7	62	72	67	3 vs 4,6	1.61	.28	.30

Table 4: Residual Analysis of Power for Models Involving Total Mortality (1), Cardiovascular Mortality (2), and Respiratory Mortality (3) as Outputs and Black Smoke (5), Temperature (6), and Relative Humidity (7) as Inputs (*indicates significance at α = .01 level).

have been averaged over all years, the coherence is a persistent phenomenon which does not depend on the overall base levels of any of the series.

5. Describing the Joint Contributions of Temperature and Pollution: The Partial Regression (Impulse Response) Functions

The thrust of the previous section was to establish that pollution (either ℓn(BSM) (4) or $\ell n(SO_2)$ (5)) and temperature (6) are jointly contributing to detrended filtered mortality through some general linearly filtered regression relation of the form (3.1), (3.2). We may rewrite this result as

$$y_j(t) = \sum_{u=-\infty}^{\infty} x_{j1}(t-u)\beta_1(u) + \sum_{u=-\infty}^{\infty} x_{j2}(t-u)\beta_2(u) + e_j(t) \qquad (5.1)$$

where

$y_j(t)$ = mortality for the t^{th} day of the j^{th} year
$x_{j1}(t)$ = ℓn(pollution) for the t^{th} day of the j^{th} year
$x_{j2}(t)$ = temperature on the t^{th} day of the j^{th} year (10 times temperature in degrees C)

The partial regression or impulse response functions $\beta_1(t)$ and $\beta_2(t)$ determine how the effects of pollution and temperature are passed along to mortality. One would anticipate that the relation should be causal, i.e., $\beta_k(t)$ should be zero for $t < 0$, so that the interpretation can be made in terms of mortality depending on the present and past values of pollution and temperature.

Again, the computations for $\beta_k(t)$ involve calculations which can be performed using the pooled spectral matrix; details can be found in Appendix A or in Wahba (1969), Shumway (1970), or Brillinger (1976).

Figure 7 shows the estimated regression (impulse response) functions relating $\ell n(SO_2)$ and temperature to overall mortality. The partial regression functions given in the lower half are reasonably stable with large coefficients in the positive direction. The values for the coefficients are given in Table A1 in Appendix A, and we note that, neglecting small statistically insignificant coefficients, the lagged regression relation seems to have the approximate form

$$y_j^T(t) = 22.30x_1(t) + .21x_2(t) - .15x_2(t-2) \qquad (5.2)$$

where $y_j^T(t)$ denotes the predicted total mortality. The interpretation is that the predicted mortality excess (it has been detrended and filtered) is instantaneously related to $\ell n(SO_2)$ and to a lesser extent with temperature. However, the lagged effects of temperature in past days tend to produce negative contributions, i.e., as temperature goes down, mortality increases and vice versa. The maximum effect occurs at $t = 2$, which implies that the negative contribution to overall mortality leads by about two days. The results using ℓn(BSM) instead of $\ell n(SO_2)$ are practically identical to those obtained for $\ell n(SO_2)$.

The corresponding partial regression functions for cardiovascular mortality, shown in Figure 8 and Table B3, are similar except that the

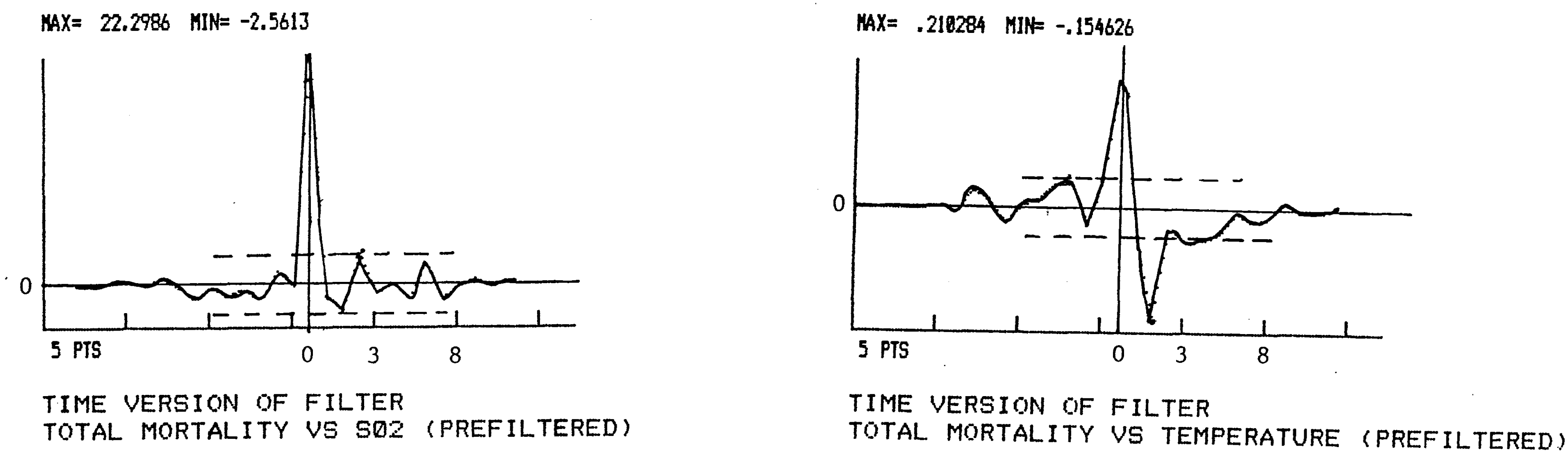
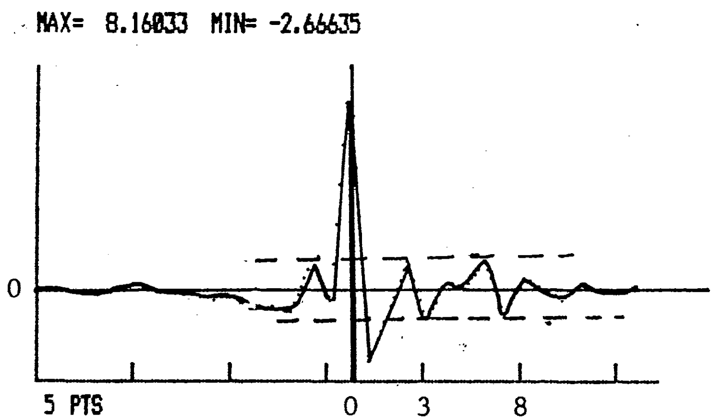
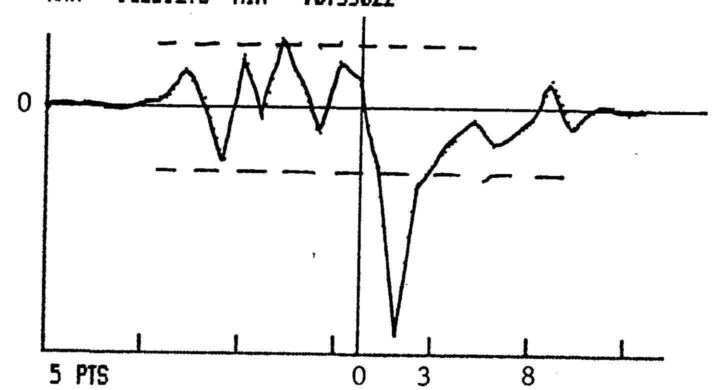


FIGURE 7. Partial Regression Functions (Lagged) Relating Total Mortality to Transformed SO_2 and Temperature for All 14 London Winters. Upper and lower dotted lines approximate 95% confidence limits when estimated filter coefficient is zero. (See Table B2.)



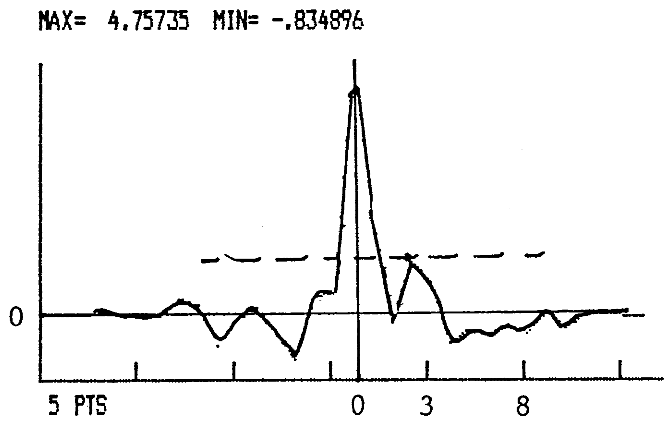
TIME VERSION OF FILTER
CARDIOVASCULAR MORTALITY VS SO2 (PREFILTERED)



TIME VERSION OF FILTER

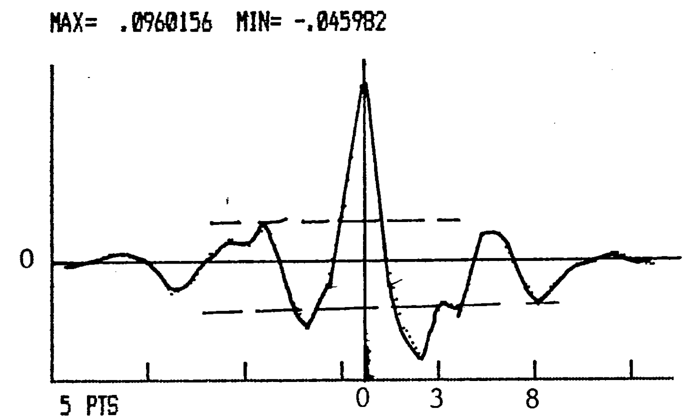
CARDIOVASCULAR MORTALITY VS TEMPERATURE (PREFILTERED)

FIGURE 8. Partial Regression Functions (Lagged) Relating Cardiovascular Mortality to Transformed SO_2 and Temperature for All 14 London Winters. Upper and lower dotted lines approximate 95% confidence limits when estimated filter coefficient is zero. (See Table A3.)



TIME VERSION OF FILTER

RESPIRATORY MORTALITY VS SO_2 (PREFILTERED)



TIME VERSION OF FILTER

RESPIRATORY MORTALITY VS TEMPERATURE (PREFILTERED)

FIGURE 9. Partial Regression Functions (Lagged) Relating Respiratory Mortality to Transformed SO_2 and Temperature for All 14 London Winters. Upper and lower dotted lines approximate 95% confidence intervals when estimated filter coefficient is zero. (See Figure B4.)

positive temperature contribution at lag zero disappears, and we obtain a strictly negative contribution due to temperature. This implies a slightly different prediction model for cardiovascular mortality which is of the form

$$y_j^C(t) = 8.16x_1(t) - .075x_2(t - 2) \tag{5.3}$$

The respiratory mortality regression functions, shown in Figure 9 and Table A4, are similar to those given for overall mortality except that there is some action over the non-causal ($t < 0$) part of the pollution filters. One may arrive at the approximate prediction model

$$y_j^R(t) = 4.76x_1(t) + .096x_2(t) - .046x_2(t - 2) \tag{5.4}$$

In any case, the partial regression functions as a whole indicate strongly that the pollution effect occurs at lag zero and that there are significant negative temperature effects occurring with the maximum occurring at a lag of two days.

Equations (5.2), (5.3), and (5.4) suggest that total and respiratory mortality are associated with two-day temperature differentials; whereas the cardiovascular mortality depends negatively on the temperature observed two days in the past.

6. Conclusions and Recommendations

The overall picture which begins to emerge from this initial analysis of the short-term effects of pollution and weather factors on different types of mortality in London can be summarized as follows:

1. The best model for associating daily mortality with the environmental factors here involves using temperature in combination with the logarithms of either the black smoke or sulfur dioxide pollutant levels.

2. The two pollutants predict mortality equally well and appear to be acting identically in all respects. The nonlinear relation holds consistently over all years and, hence, at all levels. No threshold effect was evident.

3. Relative humidity does not appear to be an important contributing factor.

4. The mechanism by which the factors are influencing mortality has pollution acting strongly and instantaneously. Temperature acts negatively at a lag of two days in the case of cardiovascular mortality; whereas the two-day temperature differential exerts a positive effect on respiratory and total mortality.

5. The strongest coherence occurs at frequencies corresponding to periods of seven to 21 days. This implies that the pollution and temperature episodes must persist in order to have a discernible effect on mortality. Very short pollution episodes or temperature swings of less than seven days do not appear to have much effect.

The above conclusions were reached on the basis of the limited London data base and should not be extrapolated to other sets of data collected in the United States or, more specifically, in California. The general nature of the relations which are indicated suggests that it may be very important to apply the techniques to data in California. For example, the fact that mortality and pollution were associated in years with low and moderate pollution levels may be peculiar to the London set, or it may be more generally applicable. It will be important, in future efforts, to determine whether similar kinds of associations can be isolated in the California data.

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APPENDIX A

Summary of Equations Used in Frequency Domain Regression

Consider the lagged multiple regression model relating p jointly stationary input series $x_{j1}(t),\ldots,x_{jp}(t)$ to the output series $y_j(t)$ in the j^{th} year where data are available over $j=1,\ldots,N$ years for $t=0,1,\ldots,T-1$ time points. We assume that the conditional mean of the output series is of the form

$$\mu_j(t) = \sum_{k=1}^{p} \sum_{u=-\infty}^{\infty} x_{jk}(t-u)\beta_k(u) \tag{A1}$$

where $\beta_1(t),\ldots,\beta_p(t)$ are regression functions to be estimated. The stationary conditional covariance function is of the form

$$\text{cov}(y_j(s), y_k(t)) = \delta_{jk} R(s-t) \tag{A2}$$

where $\delta_{jk}=1$ for $j=k$ and is 0 otherwise.

The underlying model can be expressed in the frequency domain by transforming via the discrete Fourier transform (DFT); that is, let

$$Y_j(\ell) = T^{-1/2} \sum_{t=0}^{T-1} y_j(t) e^{-2\pi i \nu_\ell t} \tag{A3}$$

for $\nu_\ell = \ell/T$ and $\ell=0,1,\ldots,T/2$. Then, for a subset of L frequencies where the conditional spectrum is approximately constant and under suitable restrictions on $x_{jk}(t)$, $\beta_j(t)$ (cf. Brillinger (1975), (1979)), we may rewrite (A1) in terms of an approximation expressed in terms of the DFTs, say

$$M_j(\ell) = \sum_{k=1}^{q} X_{jk}(\ell) B_k \tag{A4}$$

with the conditional covariance assumed to be given by $f(\nu)$, the Fourier transform of (A2), for all frequencies in the band. In matrix notation, (A4) becomes

$$\underline{M}(\ell) = X(\ell)\underline{B} \tag{A5}$$

where $\underline{M}(\ell) = \left(M_1(\ell),\ldots,M_N(\ell)\right)'$, $X(\ell) = \{X_{jk}(\ell), j=1,\ldots,N,\ k=1,\ldots,p\}$, and $\underline{B} = (B_1, B_2,\ldots,B_p)'$. This exhibits the model in the standard linear regression format, so that the estimator

$$\hat{\underline{B}}(\nu) = \Big(\sum_{\ell} X^*(\ell)X(\ell)\Big)^{-1}\sum_{\ell} X^*(\ell)\underline{Y}(\ell) \tag{A6}$$

is natural for the regression vector at frequency ν. An approximation to the time domain estimator is given by the inverse DFT ($\nu_m = m/M$)

$$\hat{\beta}_j(t) \doteq \frac{1}{M}\sum_{m=0}^{M-1} \hat{B}_j(\nu_m)e^{2\pi i\nu_m t}, \tag{A7}$$

and its variance is approximated by (cf. Wahba (1969), Shumway (1970))

$$\mathrm{var}\Big(\hat{\beta}_j^M(t)\Big) \doteq \frac{1}{M^2}\sum_{m=0}^{M-1} \hat{f}(\nu_m)s^{jj}(\nu_m) \tag{A8}$$

where $s^{jj}(\nu_m)$ is the j^{th} diagonal element of inverse of the spectral matrix of the inputs, say

$$S(\nu) = X^*(\nu)X(\nu). \tag{A9}$$

The mean square power

$$\hat{f}(\nu) = (LN)^{-1}\sum_{\ell} \| \underline{Y}(\ell) - X(\ell)\hat{\underline{B}} \|^2 \tag{A10}$$

is the estimated conditional error spectrum, $\Big(\|\underline{a}\|^2 = \underline{a}^*\underline{a}\Big)$.

We can measure the strength of the linear relation at each frequency ν using the multiple coherence

$$R^2(\nu) = \frac{\underline{s}_{xy}^*(\nu)S^{-1}(\nu)\underline{s}_{xy}(\nu)}{s_y^2(\nu)} \tag{A11}$$

where

$$\underline{s}_{xy}(\nu) = \sum_{\ell} X^*(\ell)\underline{Y}(\ell) \tag{A12}$$

and

$$s_y^2(\nu) = \sum_{\ell} \|Y(\ell)\|^2 \tag{A13}$$

are proportional to the pooled sample spectra and cross spectra between the inputs and the outputs. The test that the conditional mean is zero (no regression) can be based on the F-statistic

$$F_{2p,n-2p} = \frac{R^2(\nu)}{(1-R^2(\nu))}\;\frac{(n-2p)}{2p} \tag{A14}$$

where $n = 2NL$ in this case. If $F_{\alpha;2p,n-2p}$ is the upper α level significance point, we may reject the no regression hypothesis when

$$R^2(\nu) > \frac{kF_{\alpha;2p,n-2p}}{1+kF_{\alpha;2p,n-2p}} \tag{A15}$$

with

$$k = \frac{2p}{n-2p} \,. \tag{A16}$$

One may also compare two competing linear models by noting that if $\hat{f}_1(\nu)$ and $\hat{f}_2(\nu)$ are the estimated error spectra of the form (A10) resulting under hypotheses H_1 and H_2 with the regression coefficients defined by H_1 constituting a proper subset of the p_2 coefficients defined under H_2. The F-statistic appropriate for this case is of the form

$$F_{2(p_2-p_1),n-2p_2} = \frac{(\hat{f}_1(\nu)-\hat{f}_2(\nu))}{\hat{f}_2(\nu)} \frac{(n-2p_2)}{2(p_2-p_1)} \,. \tag{A17}$$

The above equations were computed by pooling the year-by-year spectral matrices obtained from BMD-03T (Dixon (1977)) over years. The results were then input to BMD-04T which computes (A6), (A9), (A10), and (A11) at each frequency. Equation (A7) was evaluated by computing the DFT of the frequency response $\underline{B}(\nu)$ using a fast Fourier transform algorithm.

APPENDIX B

Additional Tables Mentioned in Text

Frequency (Cycles/Day)		Series						
		1	2	3	4	5	6	7
.00	0	160.7909	14.52	46.29	0.0182	0.0111	71.9304	3.4821
.03	1	329.7257	67.07	68.46	0.0955	0.0613	480.5129	15.1434
.06	2	747.82	161.85	122.64	0.5924	0.2902	1353.67	65.33
.09	3	1016.73	239.78	130.59	0.7768	0.4603	2329.36	83.57
.13	4	464.42	147.18	75.49	0.4787	0.2950	1151.82	63.91
.16	5	528.21	161.19	63.92	0.5096	0.2965	952.15	60.23
.19	6	380.50	126.92	62.13	0.2979	0.1713	761.04	74.93
.22	7	444.17	117.97	74.39	0.2446	0.1312	600.98	87.24
.25	8	382.11	72.37	87.87	0.2152	0.1166	500.45	79.61
.28	9	505.72	119.63	65.68	0.2106	0.1273	532.36	75.43
.31	10	393.70	102.66	62.25	0.1664	0.0915	381.54	70.49
.34	11	351.24	100.66	63.73	0.1350	0.0736	369.99	79.26
.38	12	348.20	128.97	65.10	0.1273	0.0635	344.79	67.74
.41	13	363.26	156.27	50.05	0.1150	0.0546	279.95	57.55
.44	14	420.51	131.38	56.55	0.1621	0.0799	276.65	42.18
.47	15	369.06	160.78	69.77	0.0926	0.0551	296.23	52.44
.50	16	384.83	110.66	48.03	0.0620	0.0456	275.36	43.66

Table B1: Power Spectra of Prefiltered London Data--Total Mortality (1), Cardiovascular Mortality (2), Respiratory Mortality (3), Black Smoke (4), SO_2 (5), Temperature (6), and Relative Humidity (7).

INPUT LENGTH= 32 OUTPUT LENGTH= 32

FILTER COEFF

K	COEFF
1	-1.3583E-03
2	.0294172
3	-.0700377
4	-.0581218
5	.204837
6	.231599
7	-.038908
8	.593063
9	-.299427
10	-1.44497
11	-.326181
12	-1.15405
13	-.572982
14	-1.47537
15	1.2719
16	-.0769192
0-- 17 --	22.2986
18	-1.24824
19	-2.5613
20	3.00622
21	-.899371
22	.164415
23	-1.42616
24	2.06768
25	-1.39718
26	-.283807
27	.460213
28	-.135586
29	.291687
30	.0165984
31	.040385
32	8.82939E-03

INPUT LENGTH= 32 OUTPUT LENGTH= 32

FILTER COEFF

K	COEFF
1	-4.65617E-05
2	1.38798E-04
3	1.17164E-03
4	-3.67097E-05
5	2.26537E-04
6	4.61388E-03
7	-4.04938E-03
8	.0256173
9	.0125792
10	-.0207848
11	5.64887E-03
12	.0118377
13	.0345718
14	.0369206
15	-.0218786
16	.0457965
0--17--	.210284
18	-.0450681
19	-.154626
20	-.0301956
21	-.0501447
22	-.046024
23	-.0366833
24	-4.08345E-03
25	-.0195358
26	-.0121703
27	9.87381E-03
28	-4.62872E-03
29	-5.22996E-03
30	2.7563E-03
31	-1.00061E-03
32	2.09763E-04

Table B2: Partial Regression Functions Relating Total Mortality to SO_2 and Temperature (see Figure 7).

INPUT LENGTH= 32 OUTPUT LENGTH= 32
FILTER COEFF

K	COEFF
1	-1.87502E-03
2	.0171671
3	-.0385517
4	-.137567
5	.0422558
6	.270779
7	-.0607811
8	-.0881424
9	-.172537
10	-.325013
11	-.292396
12	-.717985
13	-.620582
14	-.801128
15	1.13285
16	-.411467
0--17--	8.16033
18	-2.66635
19	-.963702
20	1.05646
21	-1.13018
22	.262212
23	.0919638
24	1.1302
25	-.958793
26	.41795
27	.0822173
28	-.329725
29	.212652
30	-4.0707E-03
31	-.0401015
32	.0222291

INPUT LENGTH= 32 OUTPUT LENGTH= 32
FILTER COEFF

K	COEFF
1	1.45748E-06
2	-5.14892E-05
3	1.28175E-03
4	-7.91954E-04
5	-5.66487E-04
6	2.23176E-03
7	3.0294E-03
8	.0126233
9	2.83829E-03
10	-.0169352
11	.017796
12	-3.4276E-03
13	.0231278
14	7.96446E-03
15	-8.19643E-03
16	.0152335
0--17--	9.91875E-03
18	-.0198162
19	-.0755022
20	-.0258922
21	-.0171661
22	-9.64089E-03
23	-3.54213E-03
24	-.0117801
25	-8.51488E-03
26	-3.52707E-03
27	.0101193
28	-5.88095E-03
29	-6.31517E-04
30	1.76477E-03
31	-1.23796E-03
32	2.93144E-05

Table B3: Partial Regression Functions Relating Cardiovascular Mortality to SO_2 and Temperature (see Figure 8).

INPUT LENGTH= 32 OUTPUT LENGTH= 32

FILTER COEFF

K	COEFF
1	-4.84128E-04
2	-3.37409E-04
3	4.71508E-03
4	.0727388
5	-9.32217E-03
6	9.27878E-03
7	-.0184066
8	.289103
9	.163919
10	-.542163
11	-.0662001
12	.111934
13	-.339129
14	-.834896
15	.348543
16	.404412
0--17--	4.75735
18	1.8606
19	-.103793
20	.990905
21	.625469
22	-.514234
23	-.271813
24	-.372542
25	-.21456
26	-.344422
27	.0515372
28	-.231247
29	2.287E-04
30	.0340911
31	-1.43664E-03
32	-4.96182E-03

INPUT LENGTH= 32 OUTPUT LENGTH= 32

FILTER COEFF

K	COEFF
1	-9.79698E-05
2	-3.11327E-04
3	4.73802E-04
4	5.17618E-03
5	3.47422E-03
6	4.76346E-04
7	-.014571
8	-9.30418E-03
9	3.89406E-03
10	.0114
11	9.05875E-03
12	.0191972
13	-.0113996
14	-.03129
15	-.0110839
16	.0283768
0--17--	.0960156
18	7.55571E-03
19	-.030374
20	-.045982
21	-.018628
22	-.0222468
23	.0133849
24	.0141025
25	-5.52109E-03
26	-.0193967
27	-8.74296E-03
28	-9.34187E-04
29	1.07776E-03
30	5.08059E-03
31	6.01741E-04
32	-4.24155E-04

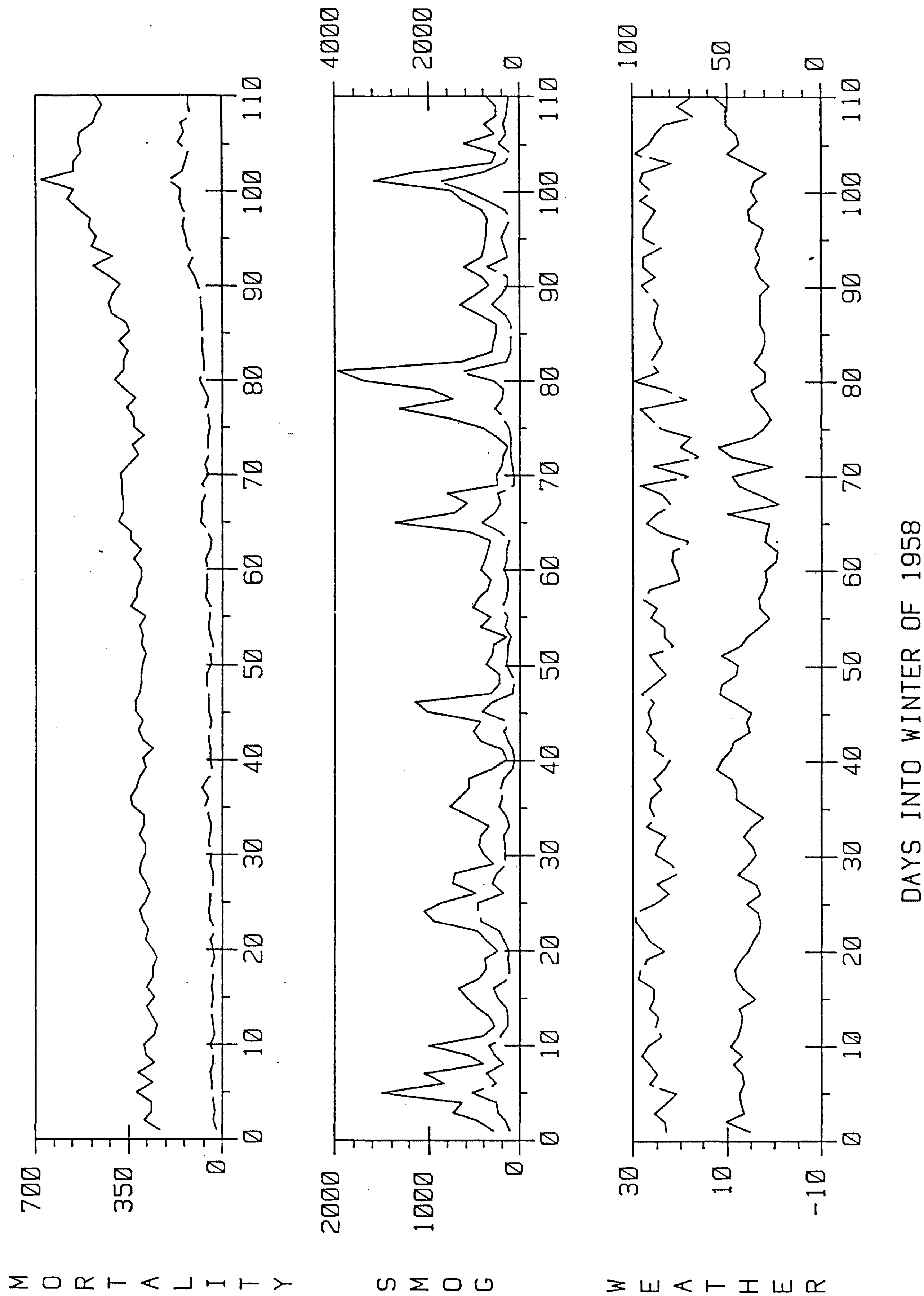
Table B4: Partial Regression Functions Relating Respiratory Mortality to Transformed SO_2 and Temperature (see Figure 9).

APPENDIX C

Basic London Data Used in Analysis (1958-1971)

Key

Mortality	1.	Total (Upper)
	2.	Cardiovascular (Middle)
	3.	Respiratory (Lower)
Pollution	4.	Black Smoke (Upper, Scale on Left)
	5.	Sulfur Dioxide (Lower, Scale on Right)
Weather	6.	Temperature (Lower, Scale on Left)
	7.	Relative Humidity (Upper, Scale on Right)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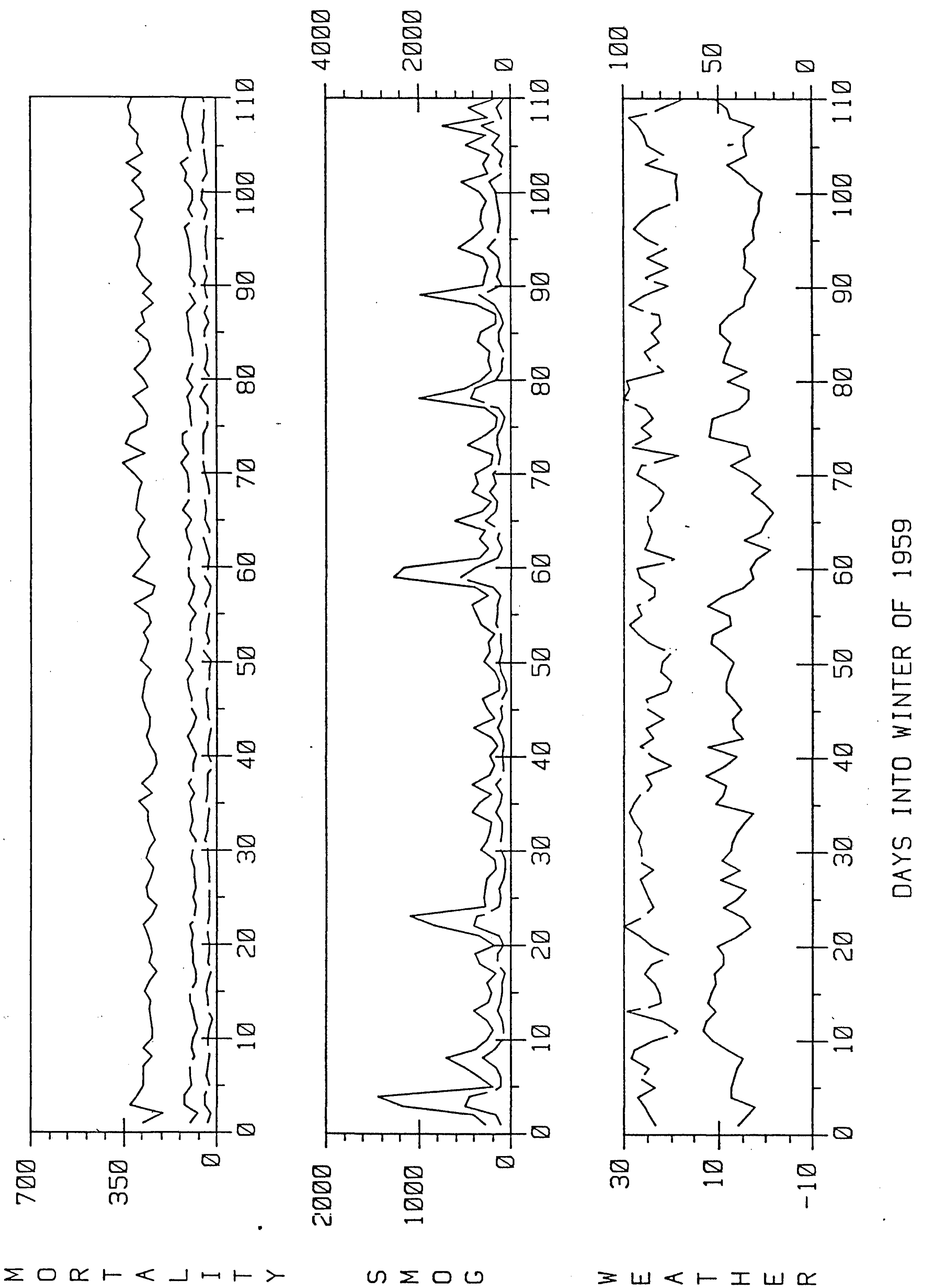
350
0
SMOG
4000
2000
0
2000
1000
0
WEATHER
100
50
0
30
10
-10
0
10
20
30
40
50
60
70
80
90
100
110
DAYS INTO WINTER OF 1958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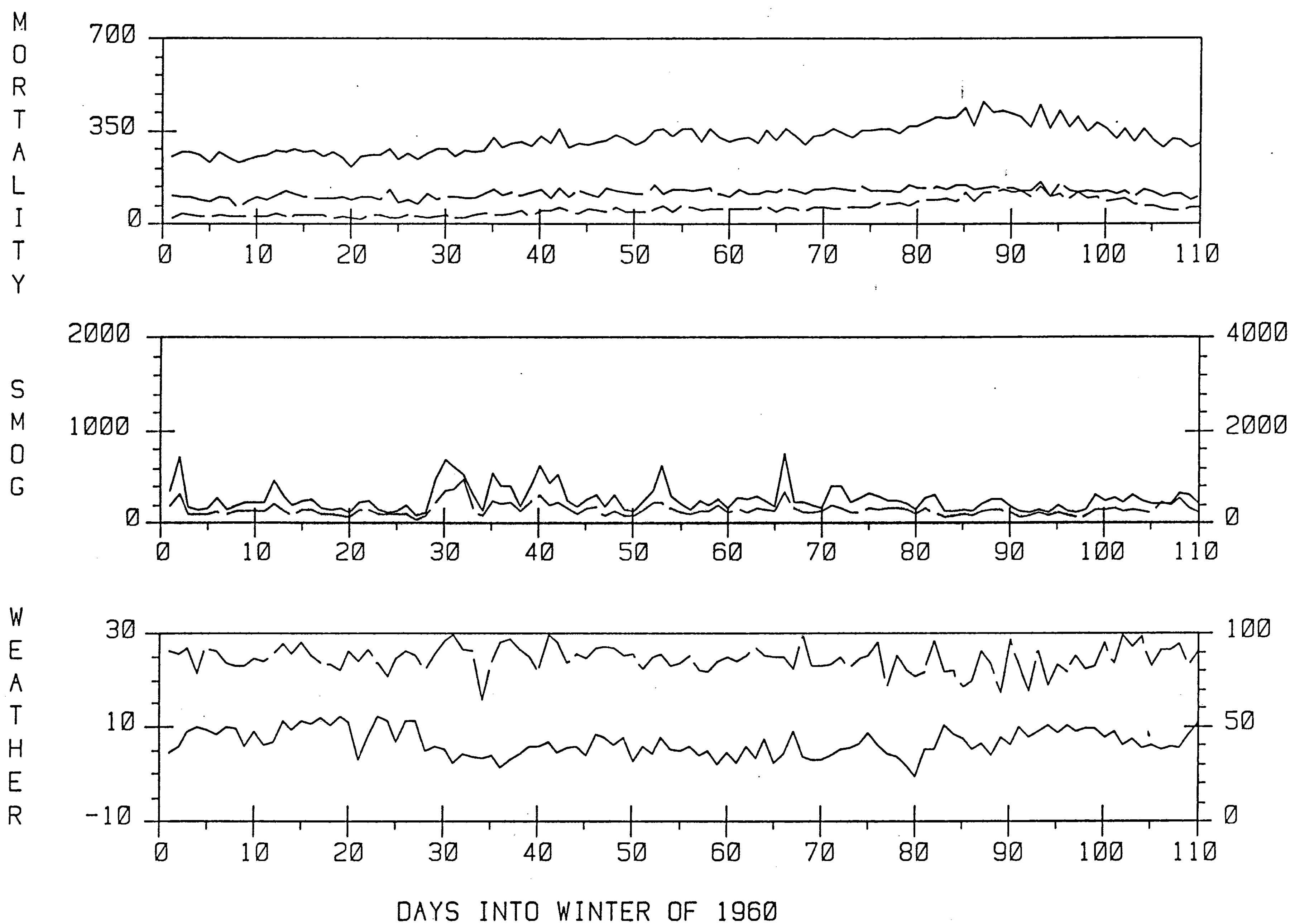
0
SMOG
2000
1000
0
4000
2000
0
WEATHER
30
10
-10
100
50
0
0
10
20
30
40
50
60
70
80
90
100
110
DAYS INTO WINTER OF 1959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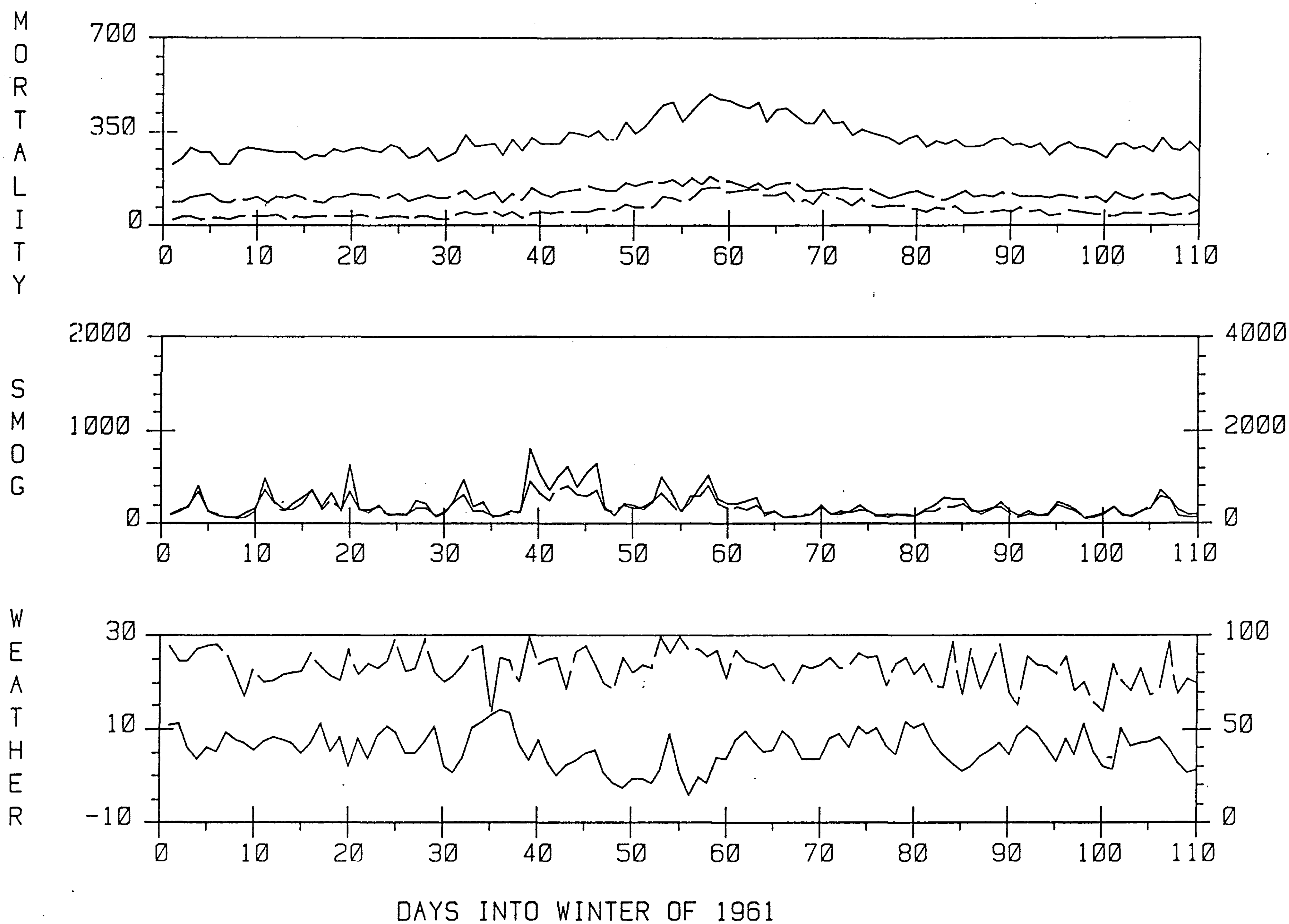
700
350
0
0 10 20 30 40 50 60 70 80 90 100 110
SMOG
2000
1000
0
4000
2000
0
0 10 20 30 40 50 60 70 80 90 100 110
WEATHER
30
10
-10
100
50
0
0 10 20 30 40 50 60 70 80 90 100 110
DAYS INTO WINTER OF 1960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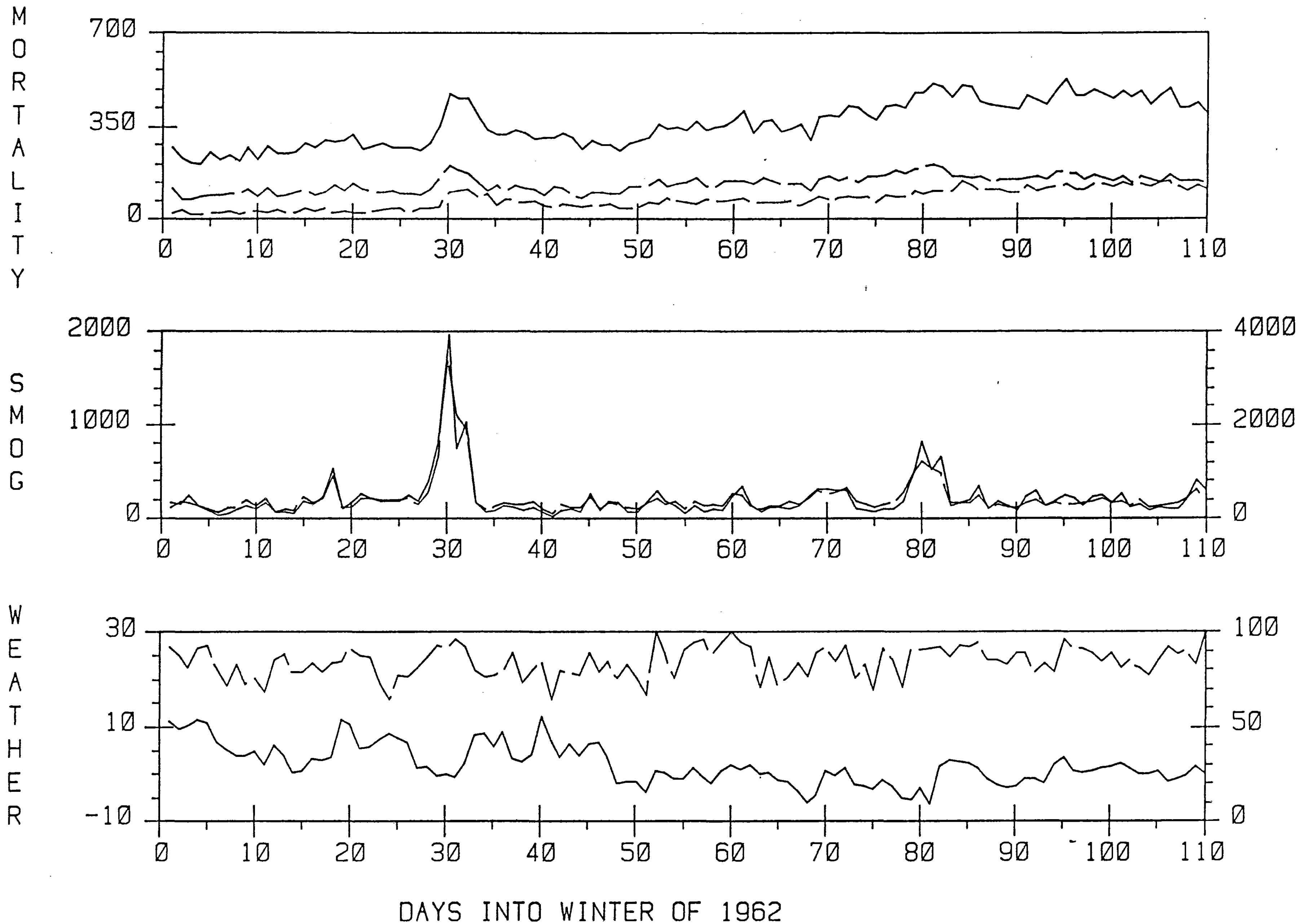
350
0
SMOG
2000
1000
0
4000
2000
0
WEATHER
30
10
-10
100
50
0
0 10 20 30 40 50 60 70 80 90 100 110
DAYS INTO WINTER OF 1961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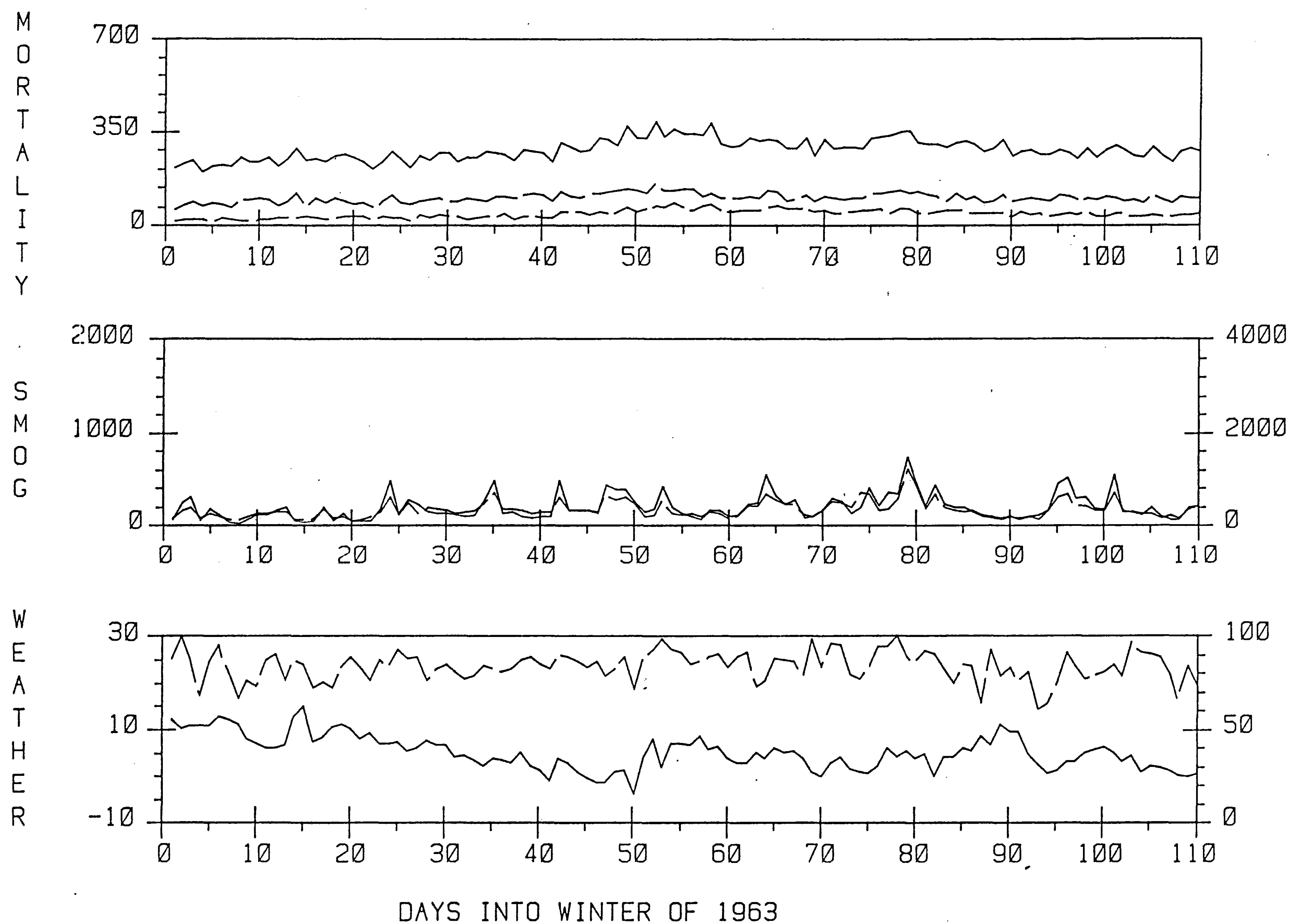
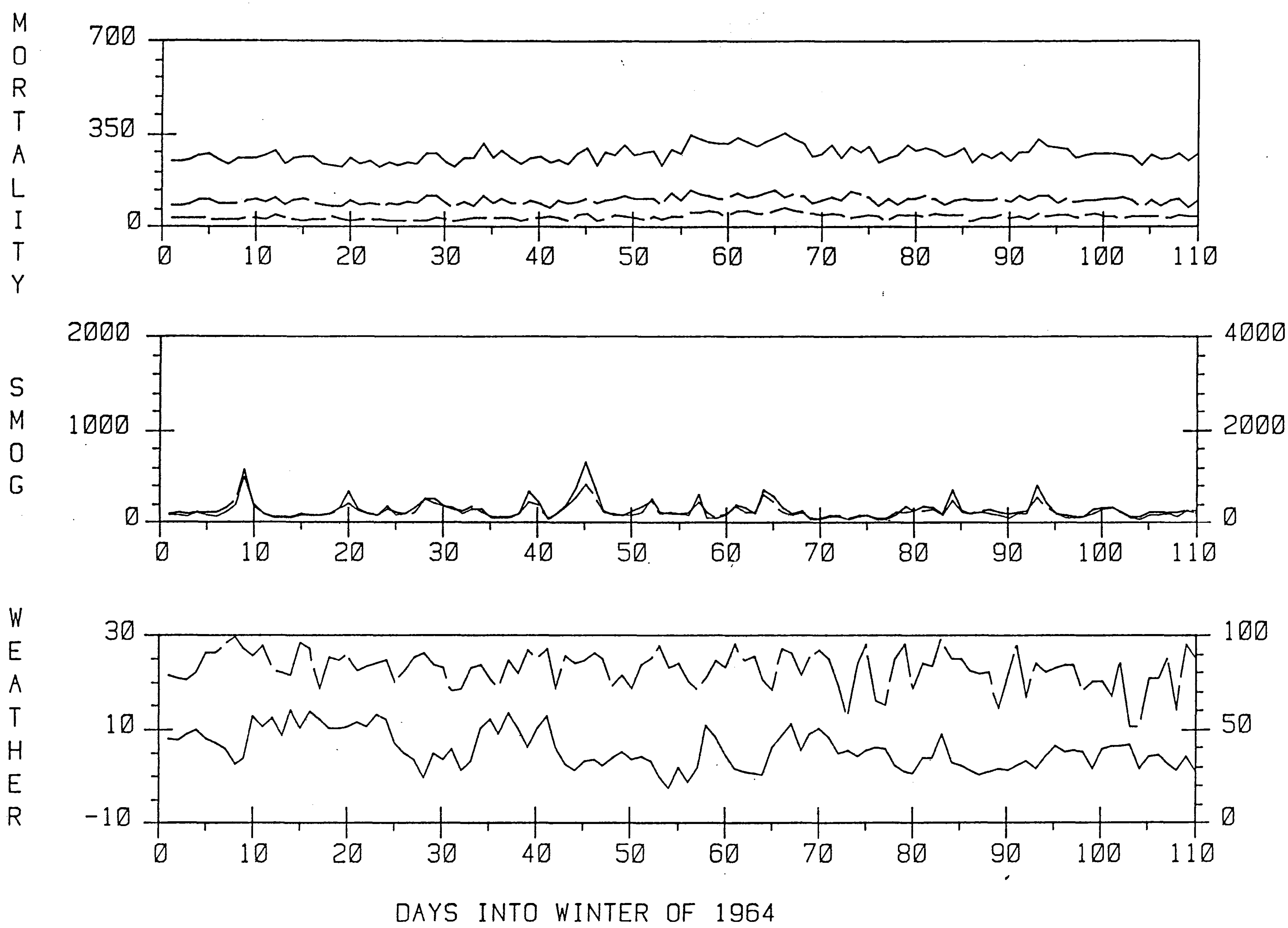
SMOG
2000
1000
0
4000
2000
0
WEATHER
30
10
-10
100
50
0
0 10 20 30 40 50 60 70 80 90 100 110
DAYS INTO WINTER OF 1962



MORTALITY
700
350
0
SMOG
2000
1000
0
4000
2000
0
WEATHER
30
10
-10
100
50
0
0 10 20 30 40 50 60 70 80 90 100 110
DAYS INTO WINTER OF 1963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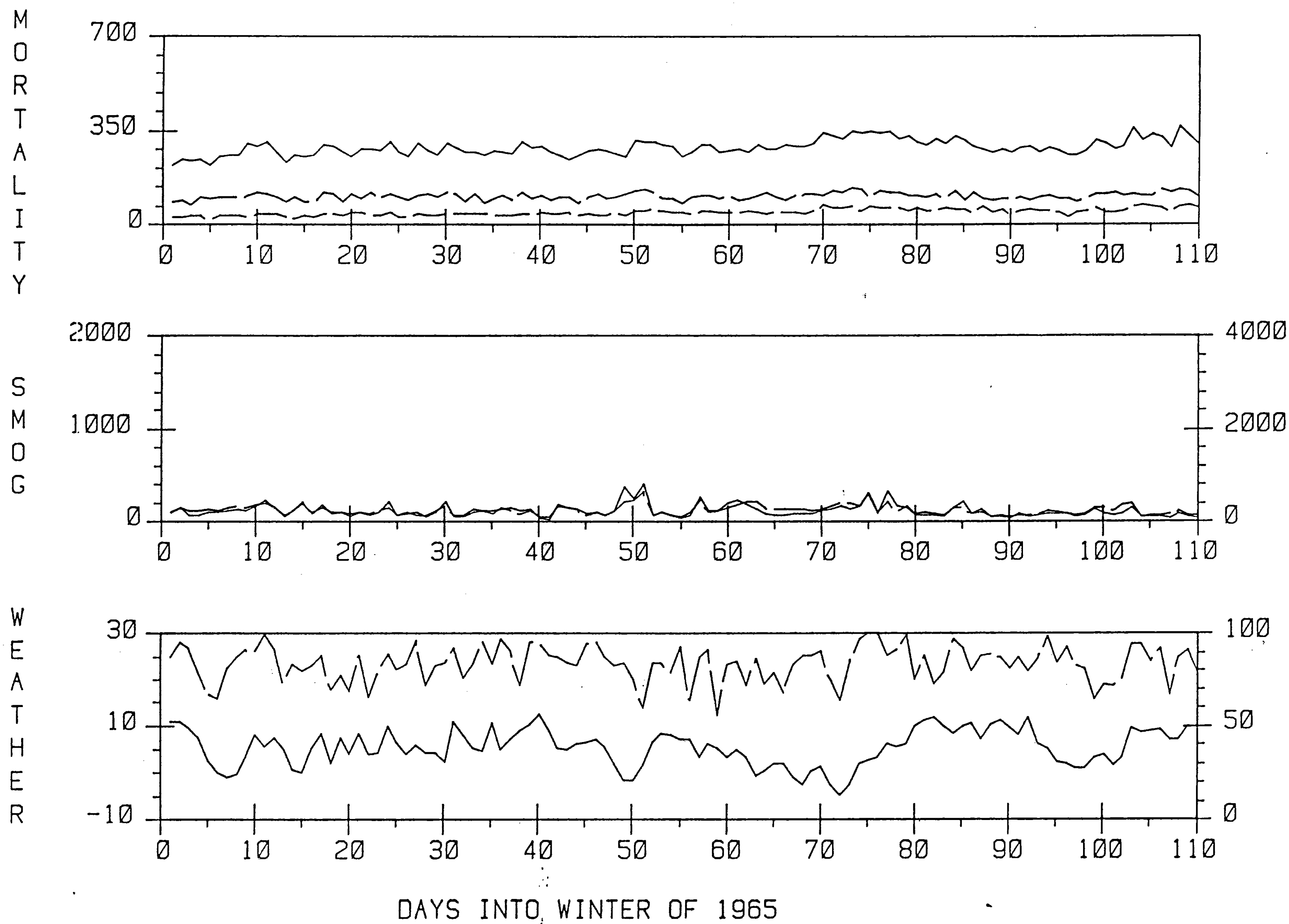
SMOG
2000
1000
0
4000
2000
0
WEATHER
30
10
-10
100
50
0
0 10 20 30 40 50 60 70 80 90 100 110
DAYS INTO WINTER OF 1964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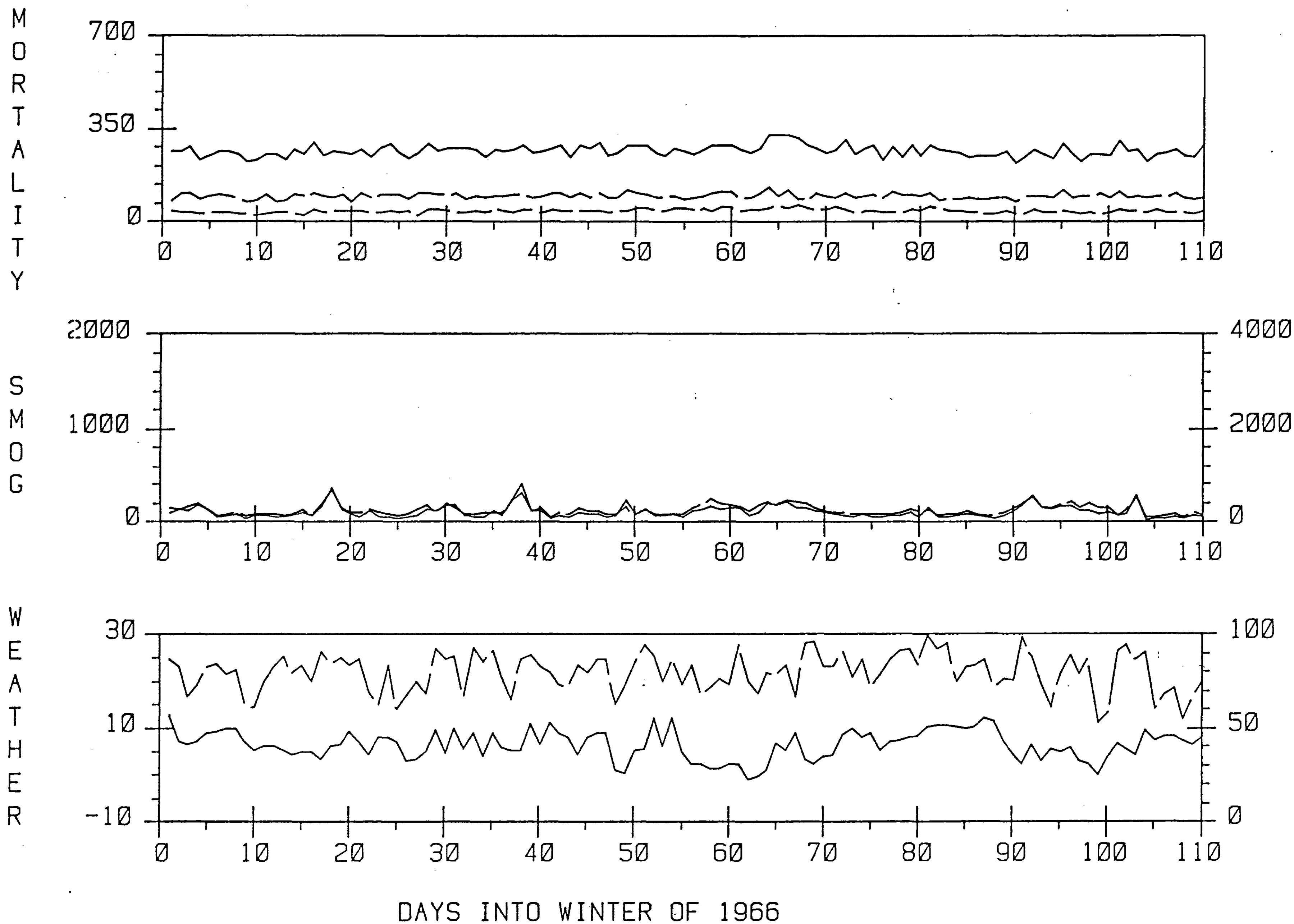
700
350
0
SMOG
2000
1000
0
4000
2000
0
WEATHER
30
10
-10
100
50
0
0 10 20 30 40 50 60 70 80 90 100 110
DAYS INTO WINTER OF 1965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SMOG
2000
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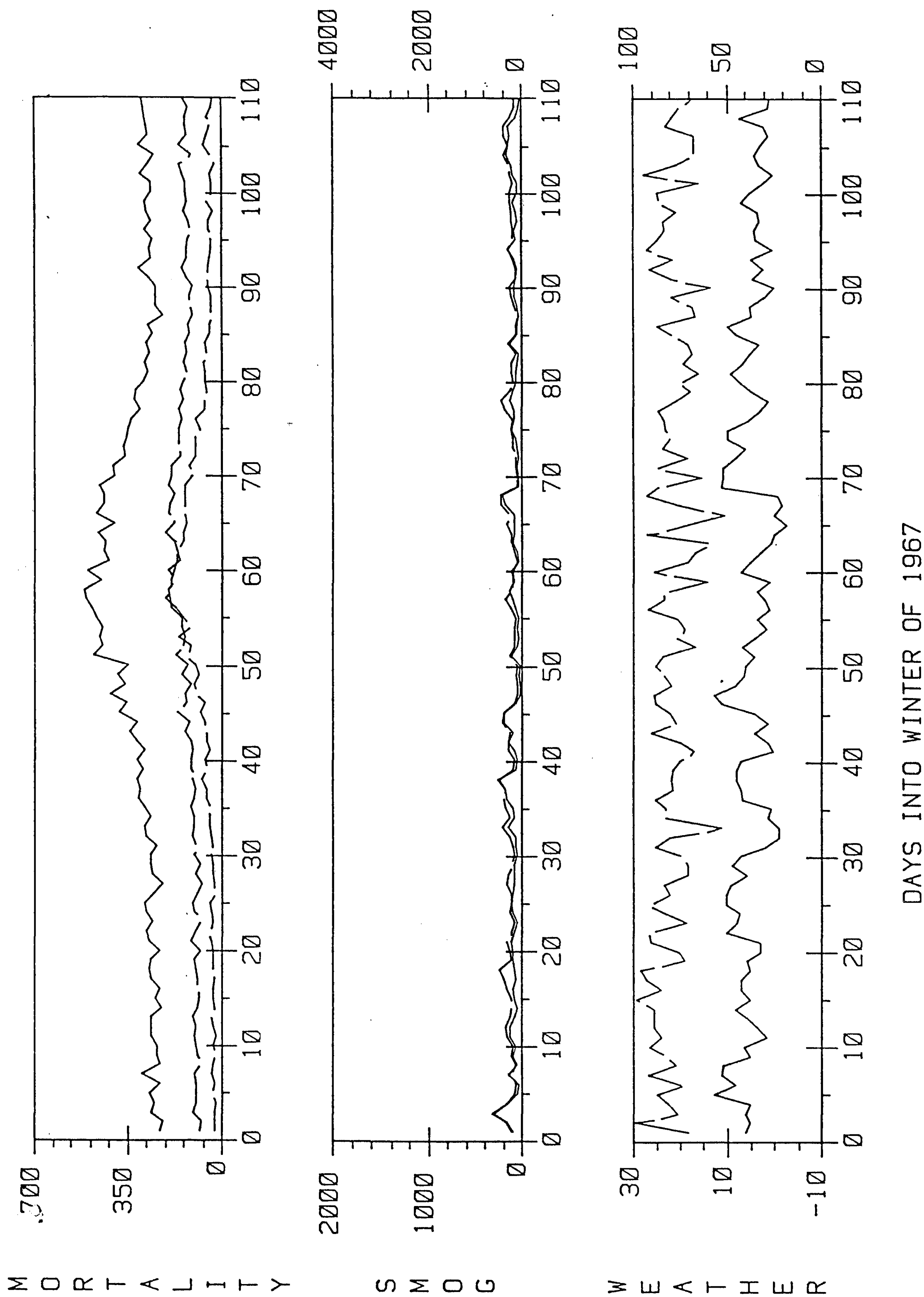
0
4000
2000
0
WEATHER
30
10
-10
100
50
0
0 10 20 30 40 50 60 70 80 90 100 110
DAYS INTO WINTER OF 1966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4000
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0
WEATHER
30
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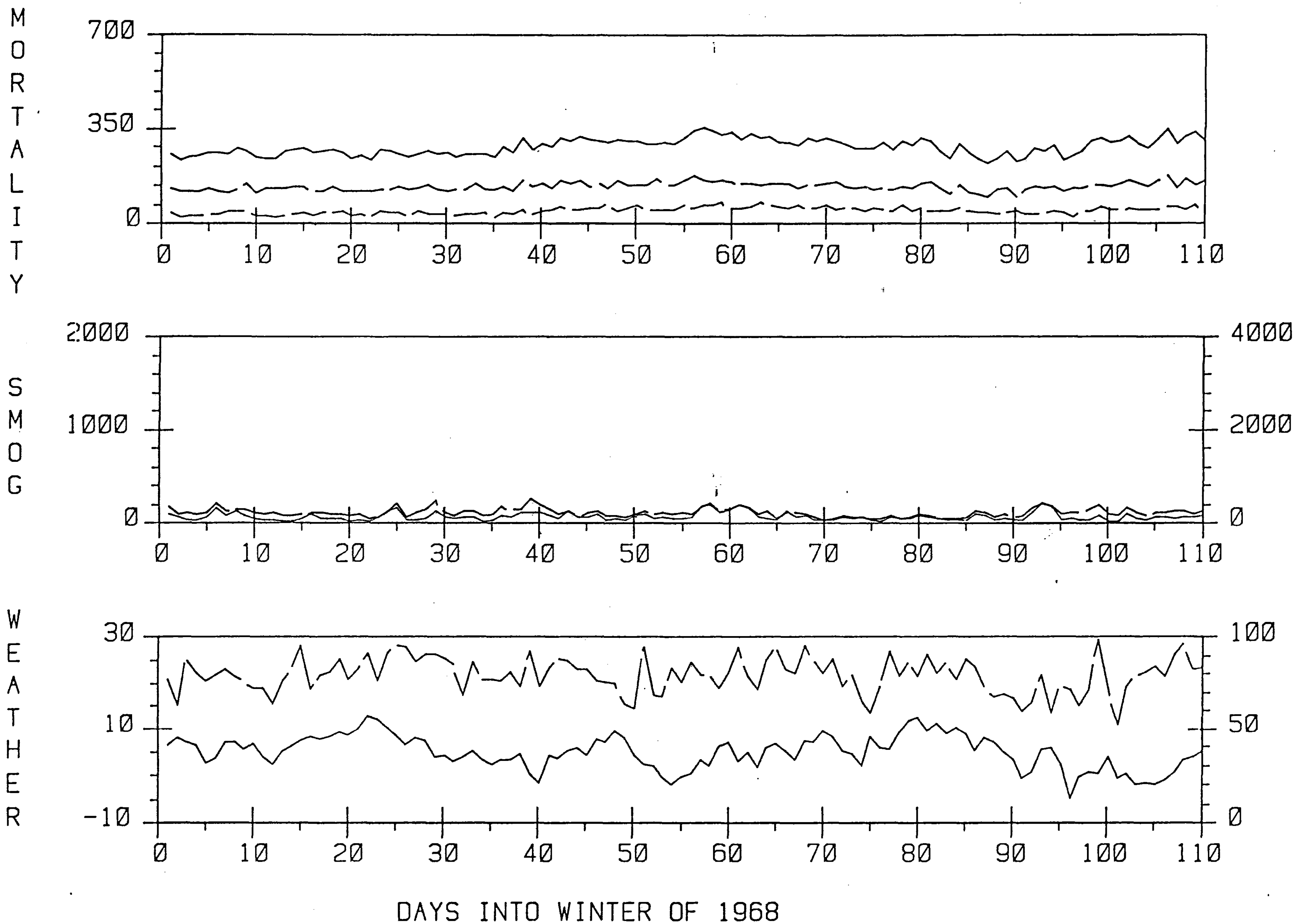
50
0
0
10
20
30
40
50
60
70
80
90
100
110
DAYS INTO WINTER OF 1967


MORTALITY
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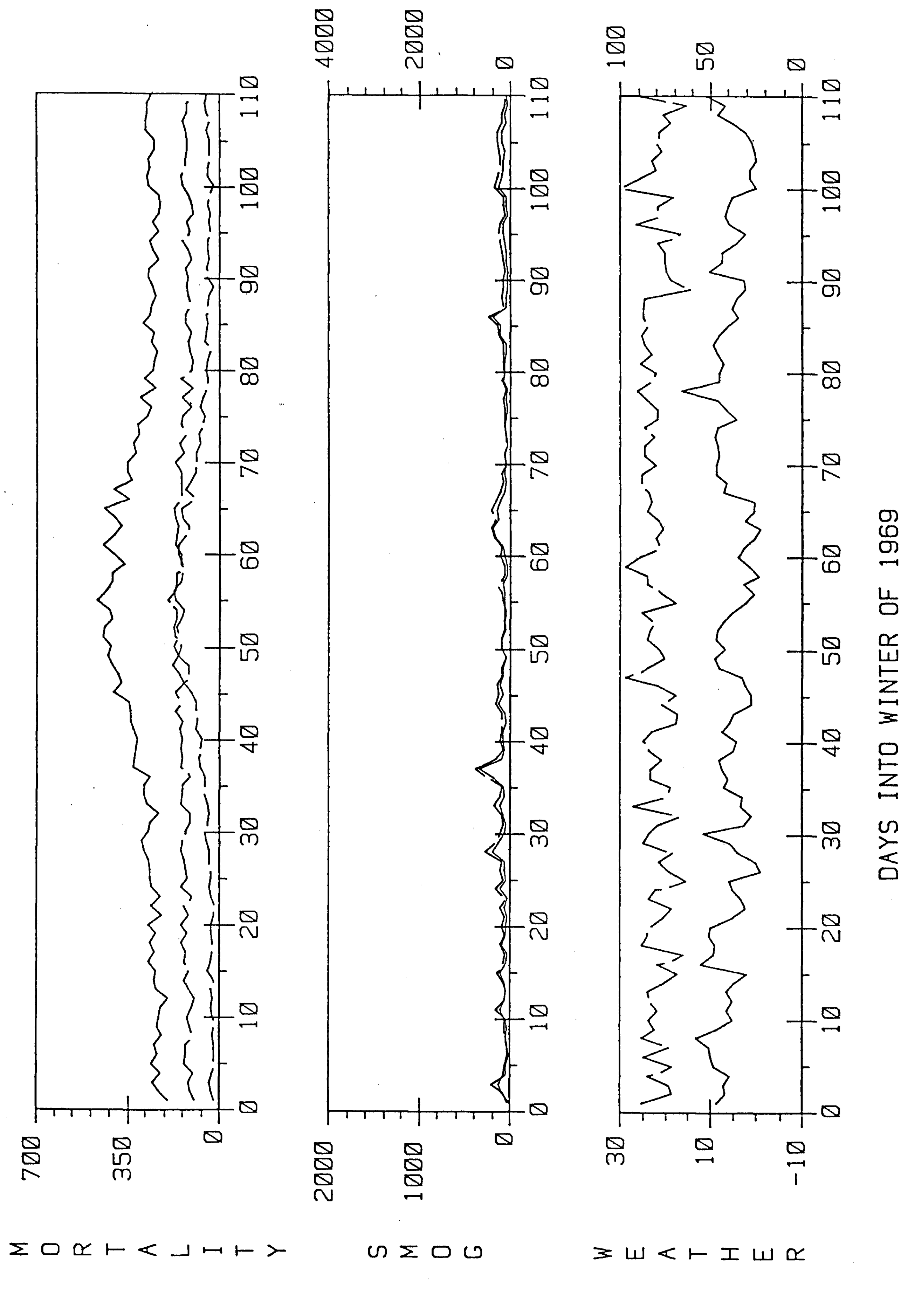
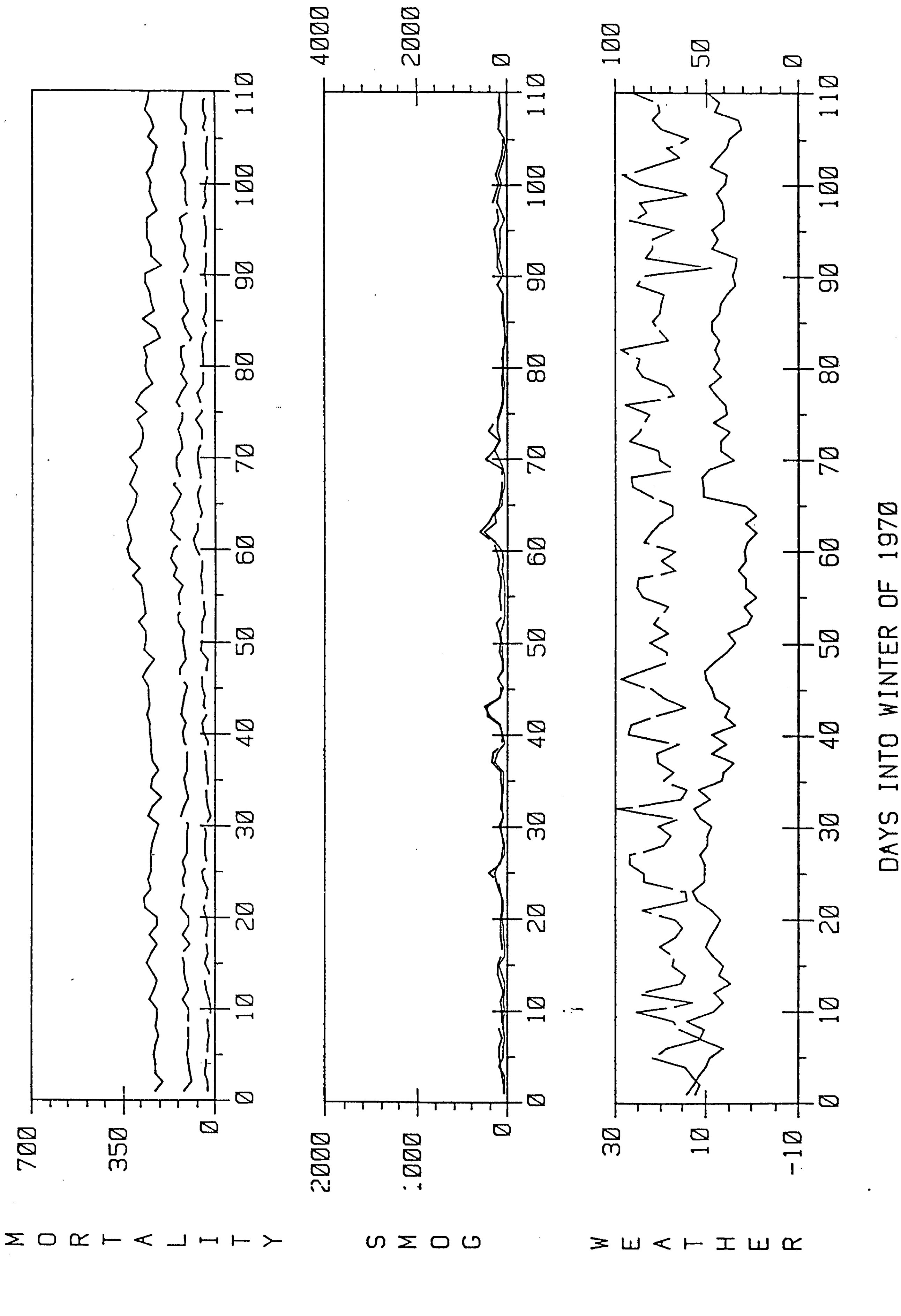
SMOG
2000
1000
0
4000
2000
0
0 10 20 30 40 50 60 70 80 90 100 110
WEATHER
30
10
-10
100
50
0
0 10 20 30 40 50 60 70 80 90 100 110
DAYS INTO WINTER OF 1968


MORTALITY
700
350
0
SMOG
2000
1000
0
4000
2000
0
WEATHER
30
10
-10
100
50
0
0
10
20
30
40
50
60
70
80
90
100
110
DAYS INTO WINTER OF 1969


MORTALITY
SMOG
WEATHER
DAYS INTO WINTER OF 1970
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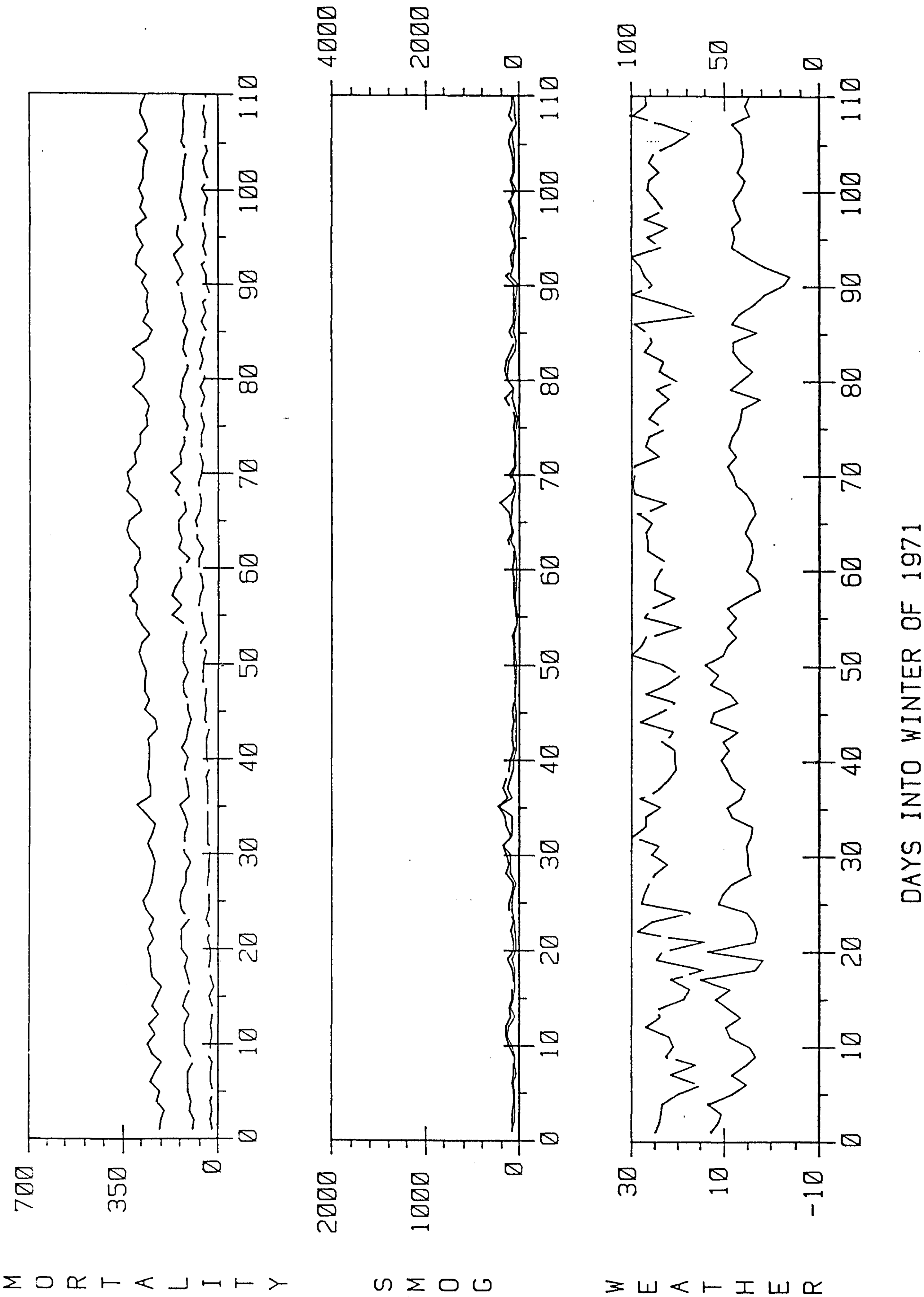
10
20
30
40
50
60
70
80
90
100
110
700
350
2000
1000
4000
30
-10


MORTALITY
700
350
0
SMOG
2000
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0
WEATHER
30
10
-10
100
50
0
0
10
20
30
40
50
60
70
80
90
100
110
DAYS INTO WINTER OF 1971