

MORTALITY, MIGRATION, INCOME, AND AIR POLLUTION:

A COMPARATIVE STUDY<sup>\*†</sup>

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## ABSTRACT

The interrelationships among different demographic factors, specific causes of death, median family income, and estimated air pollution emissions were examined. Using the Medical Data Base (MEDABA) developed at Brookhaven National Laboratory, the entire population of the United States was cross-tabulated by income and emission levels of air pollutants. Path analysis was used to examine a number of patterns and relationships for each age, race, and sex group containing a minimum of 10,000 persons. Competitive and complementary effects were observed. These effects were frequently age dependent and occasionally sex related. This specialized data base, the application of path analysis, and the development of a dynamic population and mortality model, in combination, proved to be a useful tool for investigating the effects of energy related pollutants on the exposed population.

## 1. INTRODUCTION

The influence of environmental factors such as pollution and income on human mortality has been the concern of many researchers (Winkelstein et al., 1967; Lave and Seskin, 1973; Gregor, 1977). Recently, investigators have examined apparent contradictions and inconsistencies in the results of these health effect estimates and have raised some serious questions concerning the reliability and applicability of the numbers (Finch and Morris, 1977; Morgan, et al., 1977). Aside from these questions, this basic research approach falls short of its aim to elucidate the complex interactions involved in determining the health of contemporary society. The major fault with these studies is that there has been no attempt to explicitly account for both the positive and negative effects of energy production and the consumption process. This competitive effect on mortality as well as its influence on the other factors that affect health must be investigated together. It is our purpose to develop a system of analysis that: 1) is sensitive to detrimental as well as beneficial effects of energy production; 2) examines changes in all demographic variables and their component characteristics, and 3) takes into account the dynamic interrelationships between these variables with a specific emphasis on how this complex web of events affects the health of society.

The foundation for developing such an integrated design is based on numerous research results that describe relationships between quality of life indicators and changes in demographic variables. The most significant of these research efforts focus on some measure of economic wealth such as income and on environmental quality such as air pollution. Interestingly enough, while the effect of income on all the demographic change variables is frequently found in the literature, pollution is most frequently compared with mortality and rarely with fertility or migration. In addition, epidemiologic and demographic studies have shown that gross mortality rates are dependent upon the age, race, and sex structure of the population (Enterline, 1961; Verbugge, 1975). Since the population is never static and demographic factors such as migration and fertility are important in determining population size and composition for small geographic areas in the United States, inclusion of these population determinants into the analysis of health effects of energy production and consumption is mandated.

## 2. METHODOLOGY

The conceptual framework which we used for these investigations is based on the relationship between the independent variables of income and pollution, and several dependent variables such as mortality, natality, and net migration. Practical considerations require that a number of data concerns be explicitly addressed. We will first look at these types of problems for our independent variables.

As a surrogate of dose, the pollution variable could be either directly measured as concentration ( $\mu\text{g}/\text{m}^3$ ) or estimated as emissions (tons/sq.mi.). The incompleteness and inconsistency of the

concentration data for the total United States reduce its usefulness for a comprehensive and comparative study of health effects. While emissions data are only estimates of air quality and not actual measurement, they appear to be better and more suitable for studies of this type. The income variable presents an entirely different problem. Generally very good income statistics are available. The use of median family or per capita income as measures of improved life quality, which is of interest from an epidemiological point of view, however, is questionable but remains the best available information for this level of analysis. Another consideration that must be addressed is the existence of a significant correlation between the independent variables. We have found that the logarithm of emissions per square mile and the median family income in 1970 are correlated by a factor of 0.605. Statistically, this interrelationship adds a new dimension to the interpretation of causality because both direct and indirect associations exist. This factor is further compounded if the relationship between the independent variables is also nonlinear.

The dependent variables come from three different sources: U.S. Bureau of the Census, the National Center for Health Statistics, and a jointly sponsored study of migration from the Departments of Agriculture and Commerce. Enumeration of the population by the Census Bureau every decade provides the best available information and presents little problem for our analysis. The best available data for population mobility at the county level are those for net migration by age, race, and sex between 1960 and 1970. Since these data are the computed net change over time in the population that cannot be explained by natural increase, they do not represent gross migration, nor can they be used as such. Mortality data come from the aggregation of individual death certificates and census statistics. Problems of disease classification as single or multiple causes of death are the basic limitations of the mortality data. Secondary problems arise in determining residence at the time of death and in estimating the size and composition of the population.

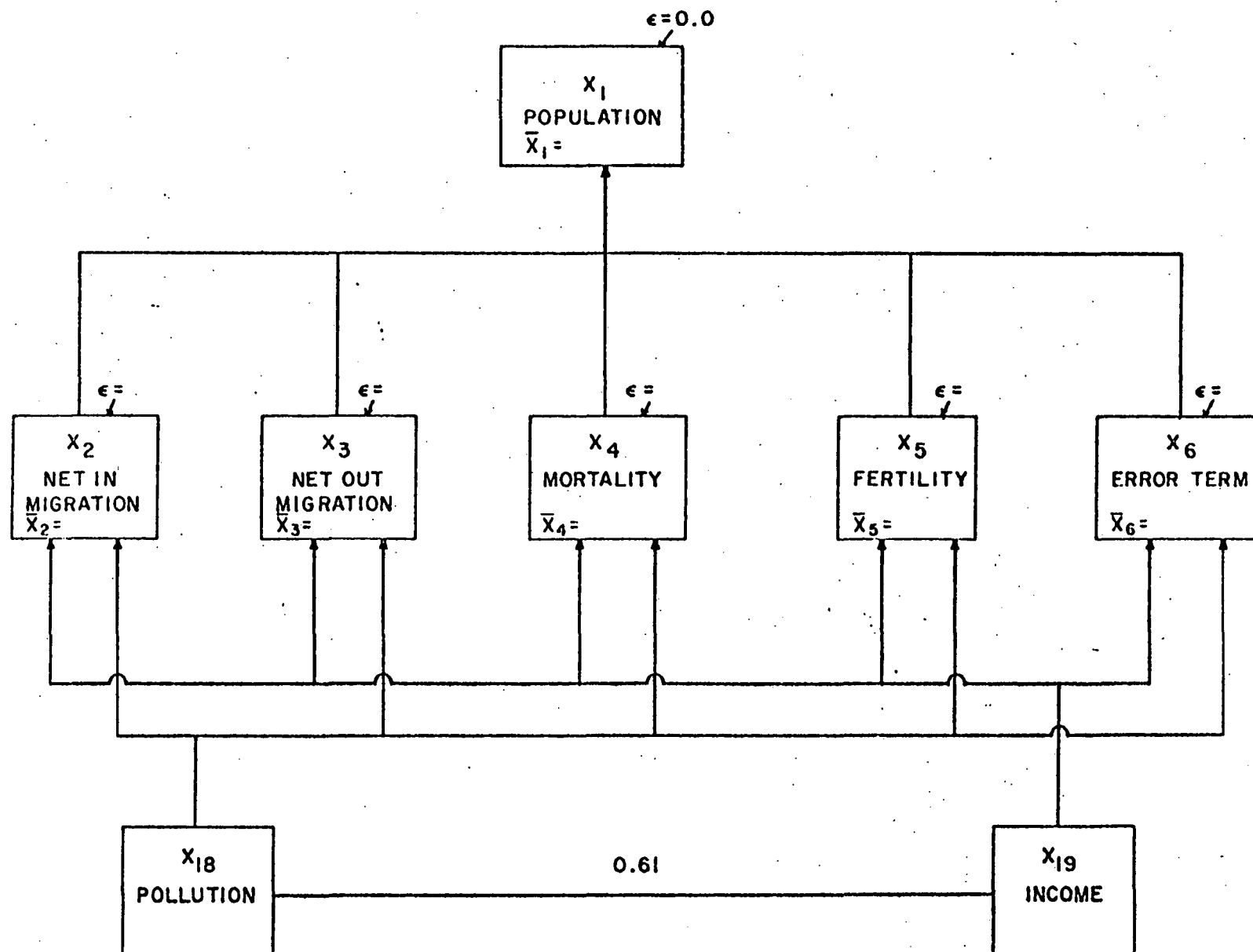
On the bases of these general considerations, we developed a more complete model, in two parts. A graphic representation of the first level of this model, Population Dynamic Model (PDM) is shown in Figure 1. In this model the current population at any point in time is explained by a causal structure which includes migration, mortality, and fertility. The theoretical framework describing this dynamic relationship is symbolically represented by the following equation:

$$P_{70} = P_{60} + F_{60-70} + IN_{60-70} - OUT_{60-70} - M_{60-70} + E \quad [1]$$

where

$P_{70}$  = total population in 1970,  
 $P_{60}$  = total population in 1960,  
 $F_{60-70}$  = total number of births between 1960 and 1970,  
 $IN_{60-70}$  = total in migration between 1960 and 1970,  
 $OUT_{60-70}$  = total out migration between 1960 and 1970,  
 $M_{60-70}$  = total mortality between 1960 and 1970,  
 $E$  = error factor.

FIGURE 1  
POPULATION DYNAMICS MODEL



$\epsilon = \text{COEFFICIENT OF ALIENATION} = \sqrt{1 - R^2}$   
 $\bar{X} = \text{MEAN RESPONSE}$

The relationship between mortality, income, and pollution is a complex interaction among different diseases, migration, and the population change that occurs when a cohort is exposed. The importance of a particular disease with respect to all causes of death in a population might be small even though a high correlation may be observed with environmental and social factors. If the objective is to identify the actual relationship between the combined effects of population and income on mortality, then it is essential that the analysis be performed in the context of a dynamic ecological/econometric model.

We have chosen to view total mortality as the sum of all cause-specific mortality, hoping to gain meaningful information about causal relationships by comparing the correlations of different disease-specific mortality rates with income and pollution. The fact that total mortality is expressed as a linear combination of specific diseases is based on the assumption that each disease is mutually exclusive of other causes of death. This system of data reduction (single cause of death) is not entirely satisfactory because it is unlikely that any death results from a single disease or accident. If the underlying cause of death is not clearly related to the contributory cause and also not related to the environmental conditions under study, then a vital link of information will be missing. Unfortunately, multiple cause of death data are not available.

The relationship of different diseases (i.e., infections and certain parasitic diseases) to varying income levels is well documented (Goldscheider, 1971). The relationship with pollution is not so clear. To examine possible associations between both income and pollution, and different causes of deaths, we developed a second level of our model, the Mortality Effects Model (MEM) which is graphically displayed in Figure 2. For the purpose of this study, influenza and pneumonia, cardiovascular diseases, malignant neoplasms, and all other causes of death will be the major delineating categories of mortality. In addition, the category malignant neoplasms will be broken down into those of the digestive system, urinary system, respiratory system, and all other neoplasms. Likewise, cardiovascular diseases will be divided into chronic ischemic heart disease, acute myocardial infarction, and all other cardiovascular diseases. For each of the relationships in our model, the following general equation is used:

$$\text{Rate}_{t,t+4} = b_0 + b_1 \text{ Income} + b_2 \log \text{ Pollution} , \quad [2]$$

where

$b_0$  = constant,

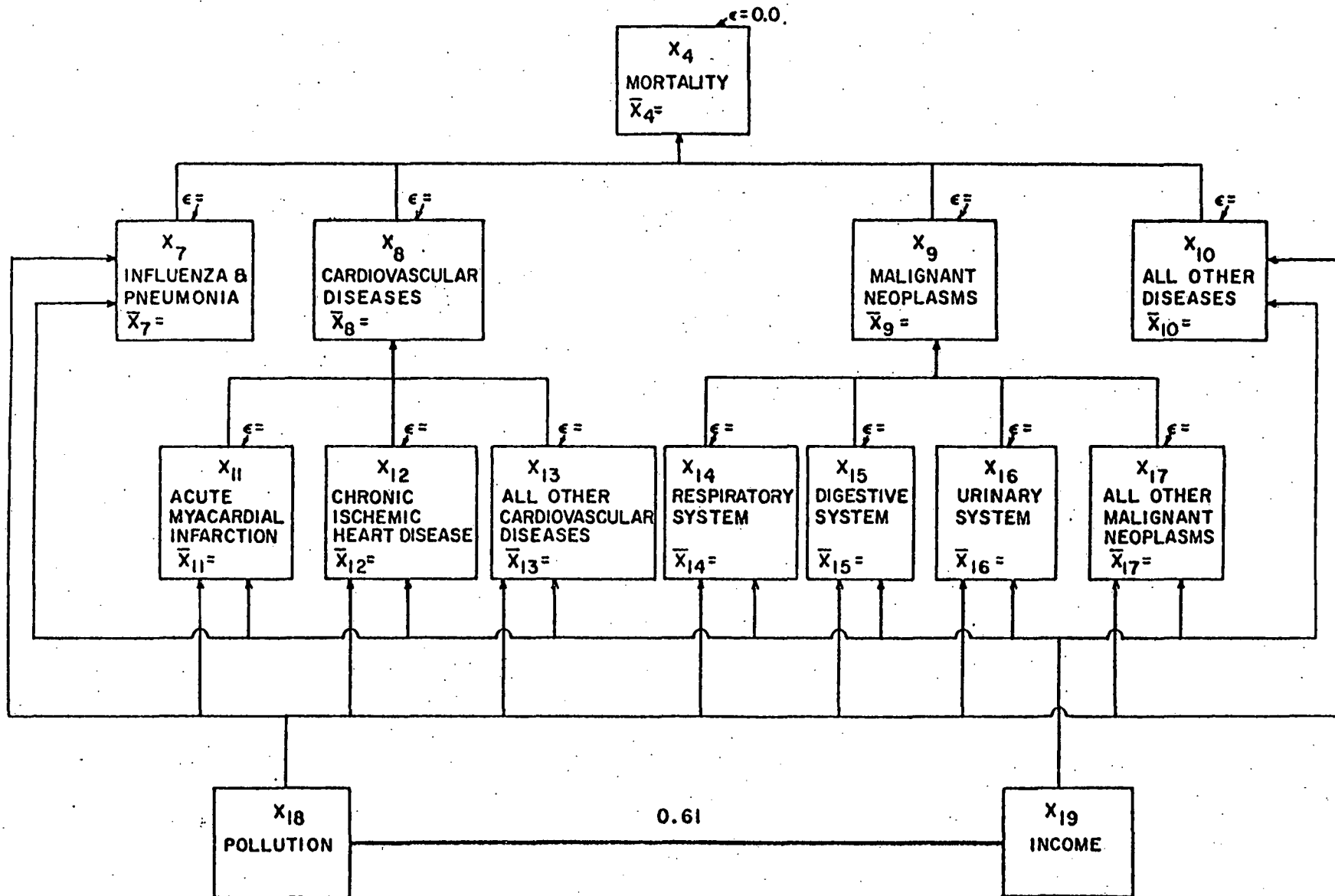
$b_1$  = regression coefficients for income,

$b_2$  = regression coefficients for pollution.

Since we are concerned with investigating a complex series of causal relationships, path analysis was chosen as the primary investigative technique. Using this modeling procedure as described in Duncan (1975), the strength and significance of each relationship



FIGURE 2  
MORTALITY EFFECTS MODEL (RATES/100,000)



$\epsilon$  = COEFFICIENT OF ALIENATION =  $\sqrt{1-R^2}$   
 $\bar{X}$  = MEAN RESPONSE

in the model can be examined by computing the respective standardized regression path coefficients  $P_{ji}$ , where  $i$  is the dependent variable and  $j$  is the variable whose direct effect on the dependent variable is being measured. The standardization method employed is

$$P_{ji} = B_{j,i} \frac{S_i}{S_j}, \quad [3]$$

where

$P_{ji}$  = beta or path coefficient of  $j$  on  $i$ ,  
 $B_{j,i}$  = regression coefficient of  $j$  on  $i$ ,  
 $S_i$  = standard deviation of the independent variable,  
 $S_j$  = standard deviation of the dependent variable.

Input to this model was generated by using the Medical Data Base developed at Brookhaven National Laboratory (Bozzo et al., 1977). All counties in the U.S. were aggregated into 192 groups based on 17 levels of income and 21 levels of pollution emissions/sq.mi. The income variable is represented by median family income data from the 1970 census. The pollution variable is represented by the decimal logarithm of the calculated sulfur emission ( $SO_x$ ) in tons/sq.mi. for 1970 (Drysdale and Calef, 1976). This logarithmic transformation of emission has a less skewed distribution than the raw estimates and is therefore preferred. The mortality variable is represented by age-, race-, sex-, and cause-specific mortality rates for 1969-71. The migration variable is represented by the net migration rates by age, race, and sex produced by Bowles and Beale and Lee. The fertility variable is represented by the average number of newborns for the period 1965-69 which was obtained from the same source as the migration data. We have decided to observe the effects of pollution and income by age cohorts because it is then possible to compute age-, race-, and sex-specific relationships that address the issue of cost in terms of life span reduction and not simply total unnecessary deaths.

### 3. ANALYSIS

For age standardized data, path coefficients describing the relationships between the dependent variables in the model with income and pollution were computed. Also path coefficients for both sexes were computed for five-year cohorts from 0 to 75+. We will first examine the interactions of traditional demographic change variables that describe our Population Dynamic Model (PDM) and then proceed to a more detailed investigation of health effects by examining the results of our Mortality Effects Model (MEM). In this presentation, only the "white" population will be examined. Future studies are planned to focus on other races and population subgroups as well as to attempt intergroup comparisons.

#### 3.1 AGE STANDARDIZED RESULTS FOR POPULATION DYNAMIC MODEL

The 1970 United States sex and race specific population was used to standardize each of the demographic variables in our Population Dynamics Model. A discussion of each of the population components follows.

### 3.1.1 MIGRATION

The two migration variables, net in and net out migration, represent the summed population gains and losses of each individual county within a pollution/income county aggregation. The net in migration coefficients are presented in Table 1 for both males and females.

TABLE 1

Path coefficients for net migration rates

	NET IN MIGRATION			NET OUT MIGRATION		
	Beta			Beta		
	R <sup>2</sup>	Income	Pollution	R <sup>2</sup>	Income	Pollution
White Male	0.24	0.46	-0.61	0.45	-0.75	0.76
		*	*		*	*
White Female	0.25	0.55	-0.58	0.42	-0.64	0.78
		*	*		*	*

\* Significant at 0.01 level.

The predictive power of the model for migration is low. Income and pollution are both significant variables. Income displays the expected positive sign for net in migration, indicating an attraction to higher income areas. Pollution, on the other hand, has a negative sign, showing an attraction to low pollution areas. For the net out migration variable, the explanatory power of the model is twice as great that for the net in migration data. The pollution variable has the largest explanatory power with the expected sign (positive). Income has less explanatory power and an opposite (negative) sign, as expected.

These results suggest a dual and somewhat surprising pattern: 1) income displays the expected effect for net in and net out migration with similar strength and an opposite sign; and 2) pollution has a positive effect on out migration which is twice as strong as the income effect. These results are consistent with the recent pattern of nonmetropolitan growth produced by net out migration from center cities (Beale, 1976). On the other hand, these results also suggest that a model which includes a term for the interaction between income and pollution, will give a more accurate prediction of out migration than will either variable alone.

### 3.1.2 FERTILITY AND MORTALITY

The variance and the beta coefficients for the correlation between income and pollution and the natural increase variables, mortality and fertility, are presented in Table 2.

TABLE 2

Path coefficients for vital rates

	R <sup>2</sup>	FERTILITY Beta		R <sup>2</sup>	MORTALITY Beta	
		Income	Pollution		Income	Pollution
White Male	0.09	-0.08   n.s.	-0.25   *	0.38	-0.74   *	0.63   *
White Female	0.17	-0.03   n.s.	-0.39   *	0.14	-0.29   *	0.47   *

\* Significant at 0.01 level.

Surprisingly, fertility was not found to be related to our income variable. Pollution, while showing little explanatory power, was negatively associated with fertility. These results may be interpreted in many ways. The data identify a geographical pattern for fertility distribution rather than a strong economic factor as a principal influence on birth rates. Mortality is the only dependent variable which shows a large and significant gender difference. The explanatory power of income and pollution on death rates is approximately 2.5 times greater for males than for females. This difference in variance is related to both independent variables. As suspected, income is negatively associated with mortality for both sexes, but much stronger for males. One possible explanation is the influence of occupation for low income males. We also found, as suspected, that pollution is positively associated with mortality rates, but we were surprised to find a 250 percent difference in the strength of this relationship between sexes. The influence of our independent variables on mortality shows a much more significant effect on males which can be specifically traced to income.

### 3.1.3 ERROR TERM

The last variable in our PDM model is the error term which represents the variation in the base population (1965) and also the true error inherent in the data. In Table 3 the results for the error term are presented.

TABLE 3

Path coefficients for the category  
error term

	R <sup>2</sup>	ERROR TERM Beta	
		Income	Pollution
White Male	0.18	0.02   n.s.	0.41   *
White Female	0.20	0.02   n.s.	0.44   *

\* Significant at 0.01 level.

The explanatory power of this variable is moderate. Pollution is the only significantly correlated variable and is important in determining the geographical distribution of the population. In other terms, pollution is more important than income in determining where people live.

### 3.2 AGE STANDARDIZED RESULTS FOR THE MORTALITY EFFECTS MODEL

To examine the combined effects of income and pollution on mortality, we turn now to our Mortality Effects Model. The statistically significant relationships between pollution and income on specific causes of death as described by path coefficients are summarized in the following sections.

#### 3.2.1 MALIGNANT NEOPLASMS

Income and pollution are good predictors for both sexes within the broad category malignant neoplasms (see Table 4).

TABLE 4

Path coefficients for the category  
malignant neoplasms

	R <sup>2</sup>	MALIGNANT NEOPLASMS Beta	
		Income	Pollution
White Male	0.61	0.17   *	0.67   *
White Female	0.55	0.31   *	0.51   *

\* Significant at 0.01 level.

Both of these independent variables have a positive effect on increasing cancer rates. We must call attention to the fact that the category malignant neoplasms is commonly accepted as the most rapidly increasing cause of death (Preston, 1976). Pollution is a better predictor than income for neoplasia death rates, with little observed difference between the sexes.

To examine this cause of death more closely, we have divided all neoplasm deaths into categories that are anatomically system specific (i.e., digestive, respiratory, urinary, and other). The relevant statistics are presented in Table 5.

TABLE 5  
Path coefficients for subcategories of  
malignant neoplasms

	DIGESTIVE			RESPIRATORY		
	Beta			Beta		
	R <sup>2</sup>	Income	Pollution	R <sup>2</sup>	Income	Pollution
White Male	0.69	0.22	0.68	0.30	0.02	0.56
		*	*		n.s.	*
White Female	0.55	0.20	0.61	0.38	0.52	0.14
		*	*		*	n.s.
	URINARY			OTHER NEOPLASIAS		
	Beta			Beta		
	R <sup>2</sup>	Income	Pollution	R <sup>2</sup>	Income	Pollution
White Male	0.47	0.49	0.29	0.19	0.09	0.37
		*	*		n.s.	*
White Female	0.01	0.04	0.06	0.48	0.28	0.49
		n.s.	n.s.		*	*

\* Significant at 0.01 level.

Surprisingly, the explanatory power of our model is strongest for digestive cancers and not for respiratory cancers as might have been expected. Here we observe pollution as the best predictor. We might hypothesize that this observation is linked to the recent increases in large bowel cancers which are common in industrial societies (Doll et al., 1970). The results for respiratory cancers

are unexpected and very difficult to explain. We observed that males are only pollution dependent while females are only income dependent. The case of males might be related to an additive risk of cigarette smoking and occupational exposure in conjunction with environmental pollution. The case of females being only significantly related to income may be explained by sociological and behavioral characteristics of women in higher income families. These explanations have not been fully explored and should be treated with caution.

For urinary cancers, our model is valid or shows differences in income and pollution only for males. The logical linkage of urinary cancers to occupational exposure is a plausible explanation for this phenomenon. We must remember that bladder cancers have been historically linked to certain chemical compounds which are found in different industries.

The other neoplasms category is by definition very difficult if not impossible to explain. Therefore, no attempt will be made to draw any conclusions concerning the influence of income and pollution on death rates of this "catch-all" category.

### 3.2.2 CARDIOVASCULAR DISEASES

For the cardiovascular disease category as a whole, we see in Table 6 that income and pollution have limited explanatory power.

TABLE 6

Path coefficients for the category  
cardiovascular disease

-----			
CARDIOVASCULAR			
Beta			
-----			
	R <sup>2</sup>	Income	Pollution
-----			
White Male	0.28	-0.50	0.65
		*	*
-----			
White Female	0.16	-0.27	0.50
		*	*
-----			

\* Significant at 0.01 level.

In contrast to malignant neoplasms, cardiovascular disease has multiple etiologies. This fact makes any analysis of this category, as a whole, illogical. We have therefore divided this broad and divergent grouping of disease deaths into the following categories. Relevant statistics are found in Table 7.

TABLE 7

Path coefficients for different subcategories of  
cardiovascular diseases

	CHRONIC ISCHEMIC HEART DISEASE			ACUTE MYOCARDIAL INFARCTION		
	Beta			Beta		
	R <sup>2</sup>	Income	Pollution	R <sup>2</sup>	Income	Pollution
White male	0.60	0.22	0.62	0.27	-0.60	0.15
		*	*		*	n.s.
White Female	0.48	0.08	0.64	0.15	-0.46	0.14
		n.s.	*		*	*

OTHER CARDIO-  
VASCULAR DISEASES  
Beta

	R <sup>2</sup>	Income	Pollution
White Male	0.38	-0.52	-0.14
		*	n.s.
White Female	0.30	-0.34	-0.27
		*	*

\* Significant at 0.01 level.

Income and pollution are very good predictors for chronic ischemic heart disease mortality rates, with pollution having higher explanatory power. There is physiological evidence to support this finding since a pulmonary involvement is an important factor in the outcome of this disease process. Explanatory power for the category other cardiovascular diseases is less than that for chronic ischemic heart disease. Here we find income to be the best predictor. Both income and pollution have negative coefficients which is logically explained from a medical standpoint by the infectious origins of rheumatic heart diseases. In the case of acute myocardial infarction, the explanatory power of the model is low. Income shows a negative and stronger association with this disease than does pollution, which is positive but weak. This factor may be linked with social mobility and its relationship to acute myocardial infarction (Syme, 1966).

### 3.2.3 INFLUENZA AND PNEUMONIA

In Table 8 we see that the category influenza and pneumonia is not predicted very well by our model even though for both income and pollution significant relationships with the "correct



or right sign are observed (e.g., negative or protective effect for income and positive or deleterious effect for pollution).

TABLE 8

Path coefficients for the category  
influenza and pneumonia

INFLUENZA & PNEUMONIA			
Beta			
	R <sup>2</sup>	Income	Pollution
White Male	0.25	-0.52 *	0.59 *
White Female	0.13	-0.42 *	0.37 *

\* Significant at 0.01 level.

#### 3.2.4 OTHER DISEASES

The other diseases category, Table 9, is very diffuse and not well defined, thus making interpretation of the results difficult.

TABLE 9

Path coefficients for the category  
other diseases

OTHER DISEASES			
Beta			
	R <sup>2</sup>	Income	Pollution
White Male	0.55	-0.81 *	0.14 n.s.
White Female	0.17	-0.47 *	0.08 n.s.

\* Significant at 0.01 level.

One very noticeable distinction is the observed difference in the behavior of our model for males and females. For both sexes, but particularly for males, income is the better predictor variable. This particular phenomenon has also been observed in the age specific analysis which will be discussed later in this paper in greater detail.

### 3.3 ANALYSIS OF AGE-STANDARDIZED RESULTS

In Figure 3. we have presented a graphical representation of all the significant path coefficients of our PDM model. We also have represented the influence of each component in determining the size and composition of the current population. As expected, the largest influence in determining current population is our error term or base population. Pollution, through this term, is the best predictor of current population size. This is not surprising since urban areas contained 73.5 percent of the total U.S. population in 1970 (U.S. Bureau of Census, 1975, p. 17). On the other hand, the pathways:

Pollution -> Mortality -> Current Population

and,

Pollution -> Out Migration -> Current Population

are important and seem to be related to the more recent nonmetropolitan growth patterns. With the exception of mortality, no significant differences can be observed between sexes.

This complex interaction between income, pollution and demographic variables opens an interesting discussion concerning different predictive population models. It seems that the actual changes in the trends of migration and mortality make monotonic projections inapplicable in most cases. We think that our model could be a more appropriate and versatile projection tool for small populations such as those at the county level.

The relative contribution of income and pollution on our primary disease categories on total mortality is presented in Table 10. Here, three unique and clearly defined disease death--influenza and pneumonia, cardiovascular diseases, and malignant neoplasms--are displayed with the composite category which we termed other diseases.

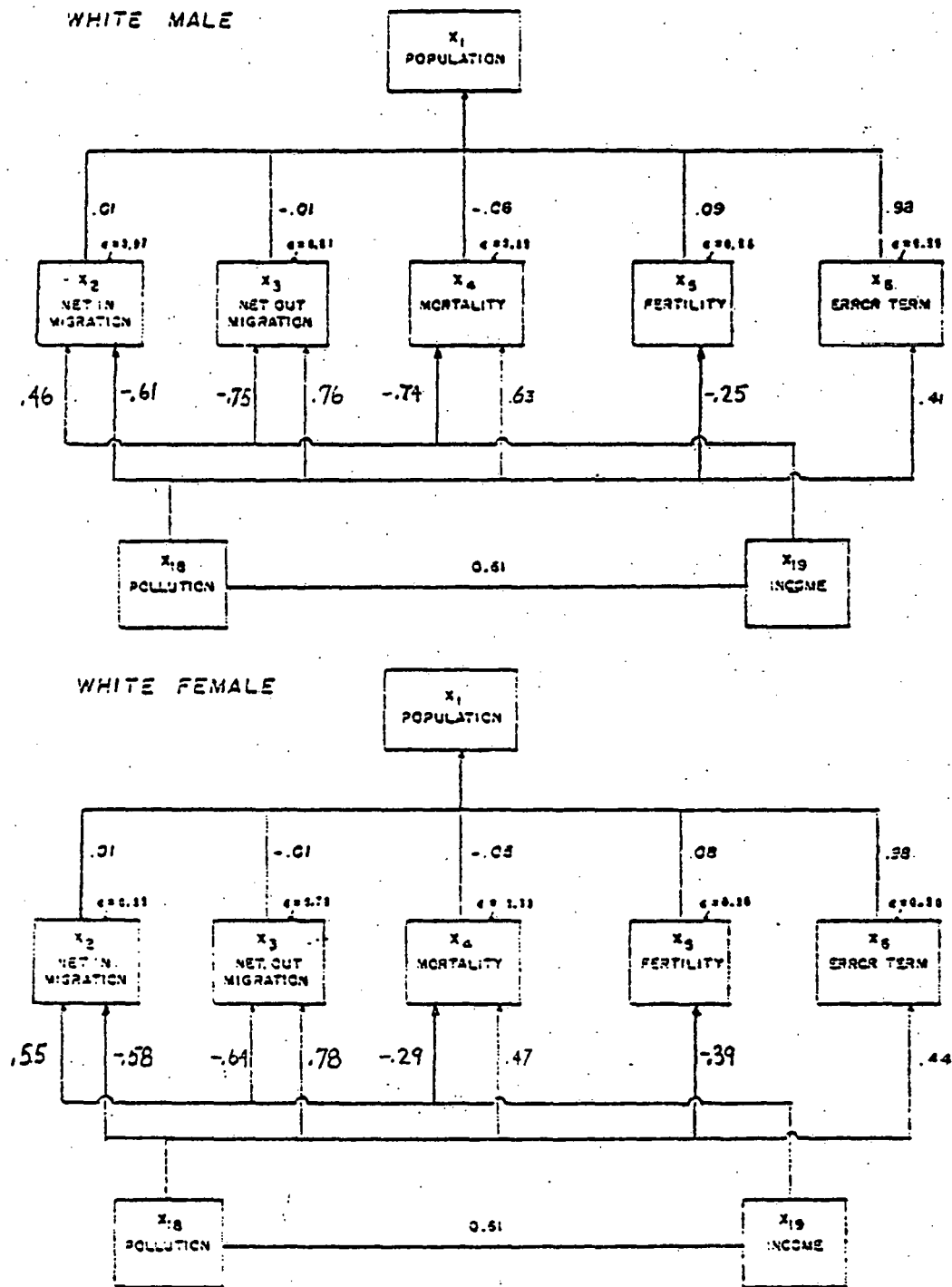
TABLE 10

The makeup of total mortality  
by disease categories

	Path Coefficients	
	White Male	White Female
Influenza and Pneumonia	0.08	0.07
Cardiovascular Diseases	0.56	0.65
Malignant Neoplasms	0.22	0.18
Other Diseases	0.56	0.31

FIGURE 3  
SIGNIFICANT BETA COEFFICIENTS,  
AGE STANDARDIZED POPULATION FOR  
WHITE MALES AND WHITE FEMALES,  
U.S. CIRCA 1970

POPULATION DYNAMICS MODEL



$r^2$  COEFFICIENT OF ALIENATION =  $\sqrt{1 - R^2}$

For white males the makeup of all deaths is more varied than for white females because the other disease category has a large influence on total mortality. This means that for males the relative influence of one disease or more probably the combined influence of a number of other causes of death is at least as significant a contributor to total mortality as cardiovascular diseases as a whole. For females, this situation does not exist--rather we see the relative contribution of other diseases to be only half that of cardiovascular diseases. We may hypothesize that improved environmental conditions, such as high income and low pollution, will tend to minimize certain causes of death, thereby producing a greater life expectancy and a more homogeneous mortality pattern. This pattern is typified by the case of white females. Further testing of this hypothesis is mandated.

The positive and negative effects of income on total mortality as manifested through several diseases are presented in Table 11.

TABLE 11

Influence of income on total mortality  
by different disease categories

	Computed Path Coefficients	
	White Male	White Female
Other Diseases	-0.46	-0.14
Acute Myocardial Infarction	-0.26	-0.12
Other Cardiovascular Diseases	-0.13	-0.08
Influenza and Pneumonia	-0.04	-0.03
Chronic Ischemic Heart Disease	0.12	n.s.
Neoplasms of the Digestive System	0.03	0.04
Neoplasms of the Urinary System	0.01	n.s.
Neoplasms of the Respiratory System	n.s.	0.02
All Other Neoplasms	n.s.	0.02

The negative or protective influence of income for both sexes is similar but stronger for white males. This effect is primarily manifested through other diseases, acute myocardial infarction, other cardiovascular diseases, and influenza and pneumonia. An observed deleterious effect of income is greater for white males than white females and is expressed through the chronic ischemic heart disease category. The remainder of this negative effect of income is expressed through the malignant neoplasms category.

The effects of pollution on total mortality through different diseases are presented in Table 12.

TABLE 12

Influence of pollution on total mortality  
by different disease categories

	Computed Path Coefficients	
	White Male	White Female
Other Cardiovascular Diseases	n.s.	-0.06
Chronic Ischemic Heart Disease	0.33	0.29
Neoplasms of the Digestive System	0.07	0.04
Neoplasms of the Respiratory System	0.05	n.s.
Influenza and Pneumonia	0.05	0.03
Other Malignant Neoplasms	0.02	0.04
Neoplasms of the Urinary System	0.01	n.s.
Acute Myocardial Infarction	n.s.	0.11
Other Diseases	n.s.	n.s.

With the exception of other cardiovascular diseases for white females, pollution increases mortality for both sexes through chronic ischemic heart disease, malignant neoplasms, influenza and pneumonia, and malignant neoplasms for all systems. Pollution has no significant effect on other diseases. In the cardiovascular group, the deleterious effect of pollution is most strongly observed through

chronic ischemic heart disease (defined according to ICD # 412, 8th revision). The influence of pollution in malignant neoplasms occurs mainly through the digestive system and the group other neoplasms. A positive effect of pollution on acute myocardial infarction was observed for white females but not for white males.

In summary, the age specific results of our mortality effects model are graphically displayed in Figure 4.

### 3.4 EFFECTS OF INCOME AND POLLUTION BY AGE

Our results for specific age cohorts show interesting differences in the relationship between income and pollution. A number of selected dependent variables are presented. In the following figures, the abscissa is scaled by the midpoints of each age cohort. The ordinate is the standardized regression coefficient for the displayed dependent variable on each of the independent variables. The shaded area is an approximation of the 1% confidence limit ( $\alpha > 0.01$ ).

#### 3.4.1 MIGRATION

Figure 5 graphically presents the influence of age on the relationships between income and pollution and the migration variables. For both sexes the relationship of income with net in migration is both strong and positive. Between the ages 15 to 25, and beyond age 55, income does not explain net in migration. The pollution variable closely resembles a mirror image of the income influence pattern with a slightly lower strength.

Net out migration, in contrast to net in migration, shows different behavioral patterns for each independent variable. Pollution, the stronger variable, shows a definite influence on increasing net out migration. The only exception occurs between the ages of 20 and 30 years, where the observed relationship is not significant. The influence of income in explaining net out migration is moderate, negative, and generally constant up to age 55 where the relationship becomes nonsignificant.

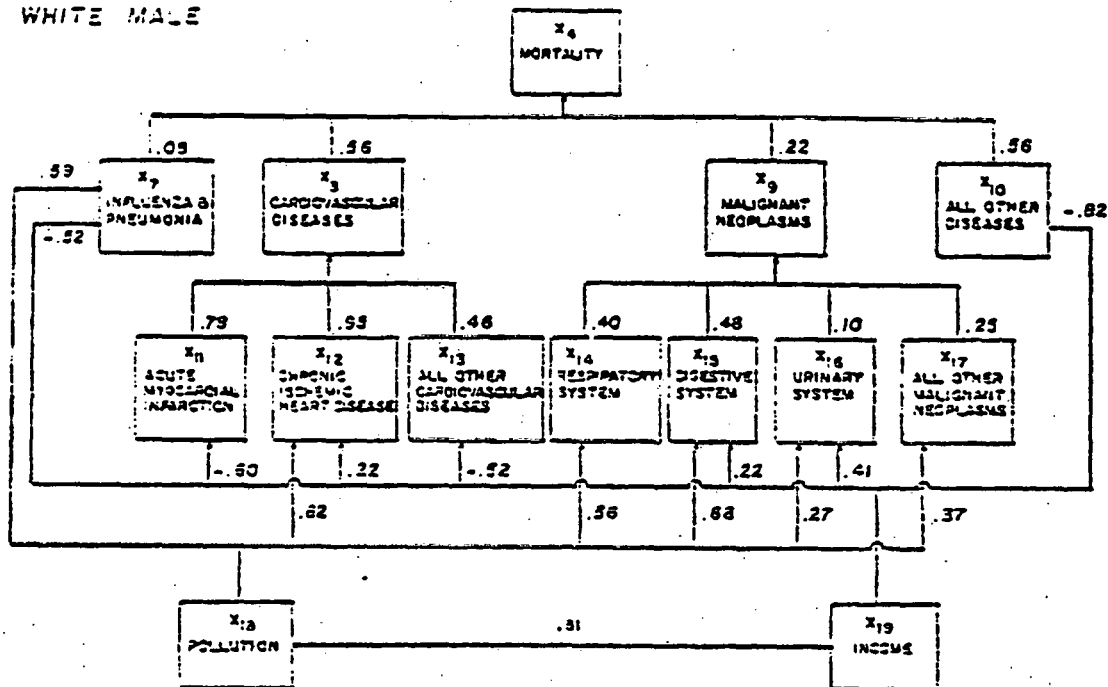
#### 3.4.2 MORTALITY

Figure 6 shows the effects of income and pollution on mortality for white males and white females at all ages. Beginning at age 30, the effect of pollution increases with age for both sexes. Pollution is significantly associated with mortality in the 0 to 4 age group for white females but not for white males. This finding suggests that pollution has a deleterious effect on infant mortality, but this area requires further research. Income shows an expected positive effect for both sexes. Surprisingly, the dependence of male mortality on income is much stronger than for females. Evidence of occupational exposure as a differential factor in determining increased mortality for lower income males is suggested by this general trend. The effect of income for older ages (60+) is not significant. This detrimental effect of income by sex may be better

FIGURE 4  
SIGNIFICANT BETA COEFFICIENTS,  
AGE STANDARDIZED POPULATION FOR  
WHITE MALES AND WHITE FEMALES,  
U.S. 1969-71

MORTALITY EFFECTS MODEL (RATES/100,000)

WHITE MALE



WHITE FEMALE

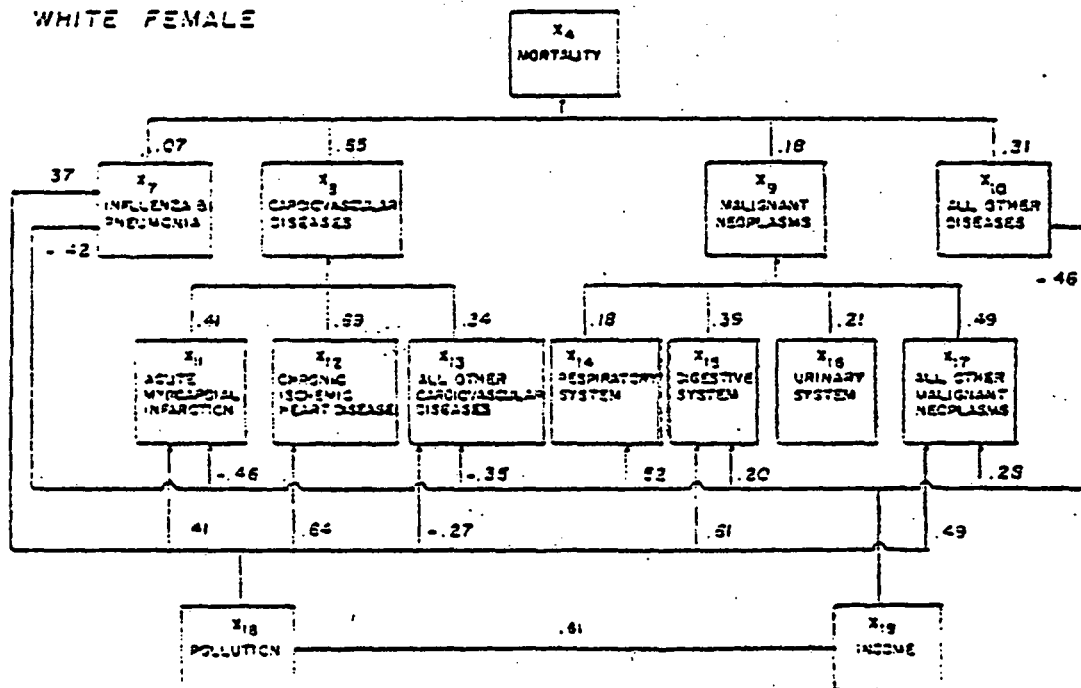
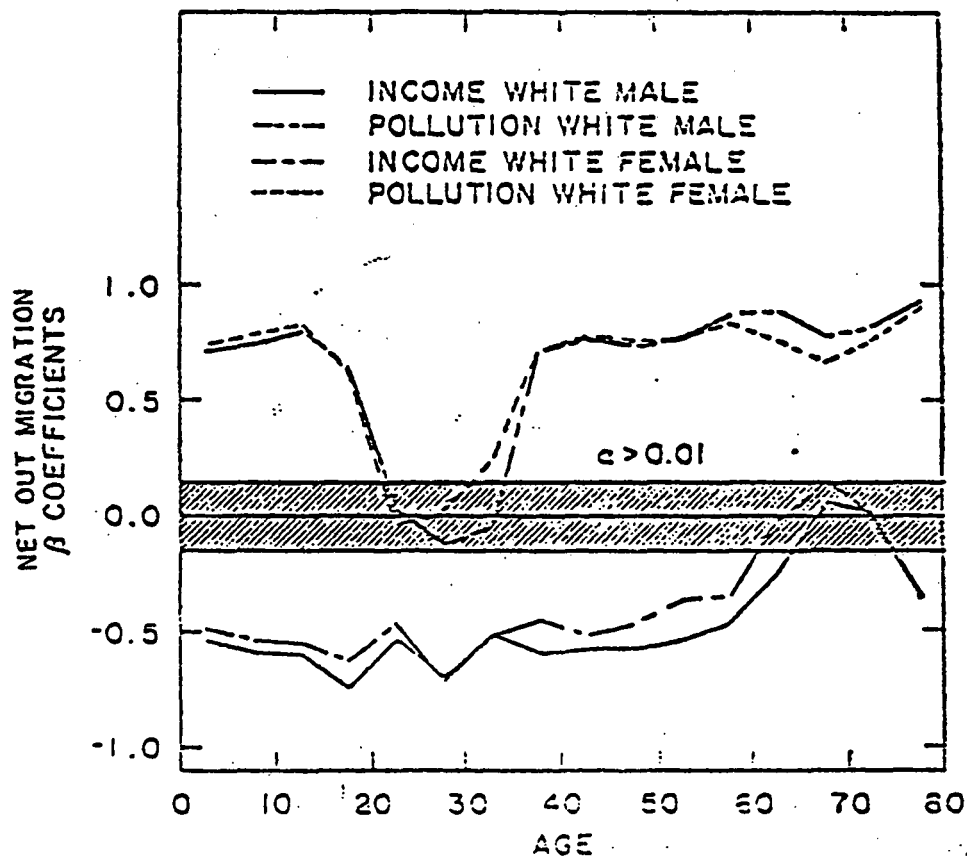
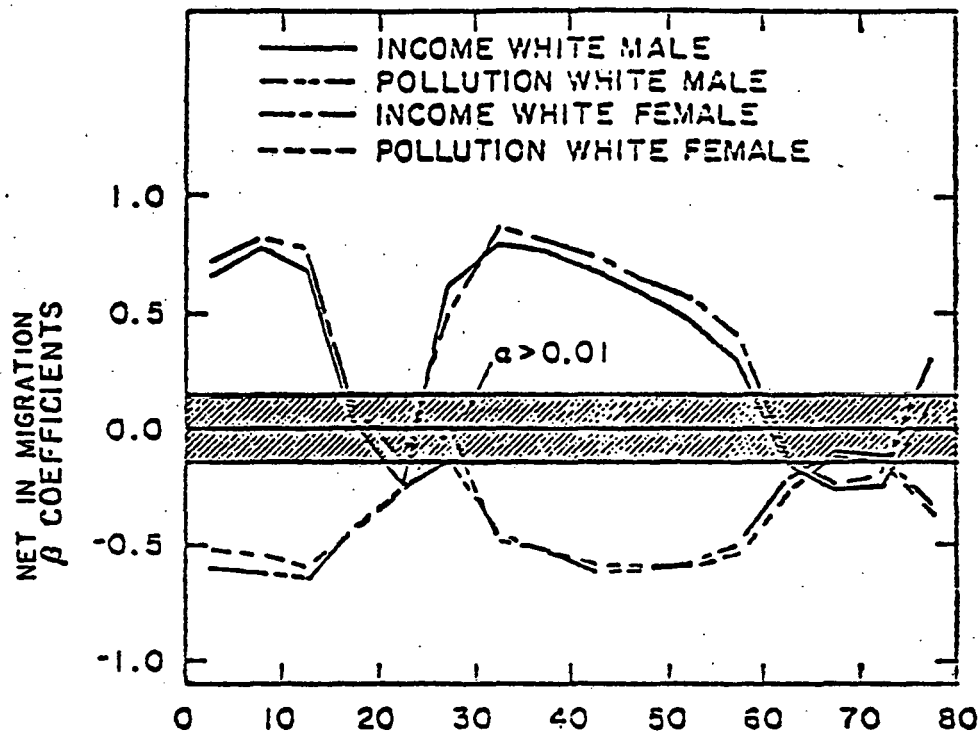


FIGURE 5

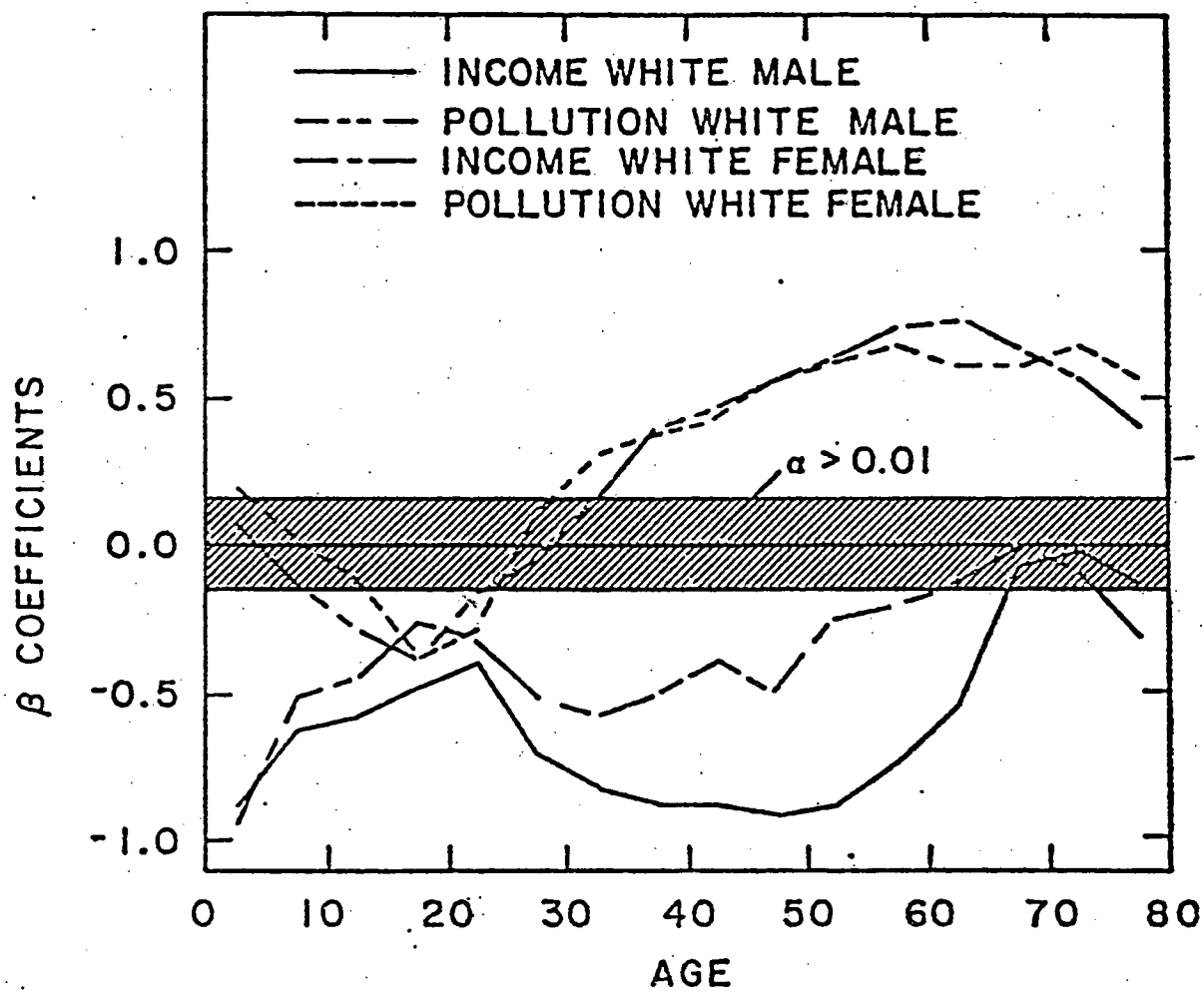
THE COMBINED EFFECT OF INCOME  
AND POLLUTION BY AGE FOR  
NET IN AND NET OUT MIGRATION



N.B. POINTS PLOTTED AT THE MID POINT OF EACH 5 YEAR AGE INTERVAL



FIGURE 6  
THE COMBINED EFFECTS OF  
INCOME AND POLLUTION BY  
AGE ON TOTAL MORTALITY



N.B. POINTS PLOTTED AT THE MID POINT OF EACH 5 YEAR AGE INTERVAL

understood by examining the combined effects of our independent variables on different disease deaths.

#### 3.4.3 INFLUENZA AND PNEUMONIA

Figure 7 presents the results for a respiratory infectious disease category. Influenza and pneumonia are, in general, negatively associated at a relatively constant level with income (the 0-9 age cohort is the only deviation from this trend). The suspicion that influenza and pneumonia are positively associated with pollution because of the respiratory component of the diseases is noted for older ages.

#### 3.4.4 CARDIOVASCULAR DISEASES

Examination of our results for cardiovascular diseases (Figure 8) shows a pattern that closely resembles the pattern for total mortality. The only striking difference is that cardiovascular diseases do not show the differentiating influence of income by sex.

Looking at two specific diseases within this group, we observe different patterns of correlation for our independent variables of income and pollution. Acute myocardial infarction (Figure 9) shows the strong negative dependence on income which is similar for both sexes. This pattern is significant past 20 years of age. Chronic ischemic heart disease, on the other hand, is more closely related to pollution. Similarly, this relationship is significant for both sexes past 20 years of age. The differences between these two cardiovascular disease subgroups may be related to differential diagnosis and medical care or contributory causes of death. This curious behavior of heart diseases has been described previously (Daw, 1954), and is suggested to be related to social class stratification. The implication of linking pollution to chronic ischemic heart disease is significant and warrants further research.

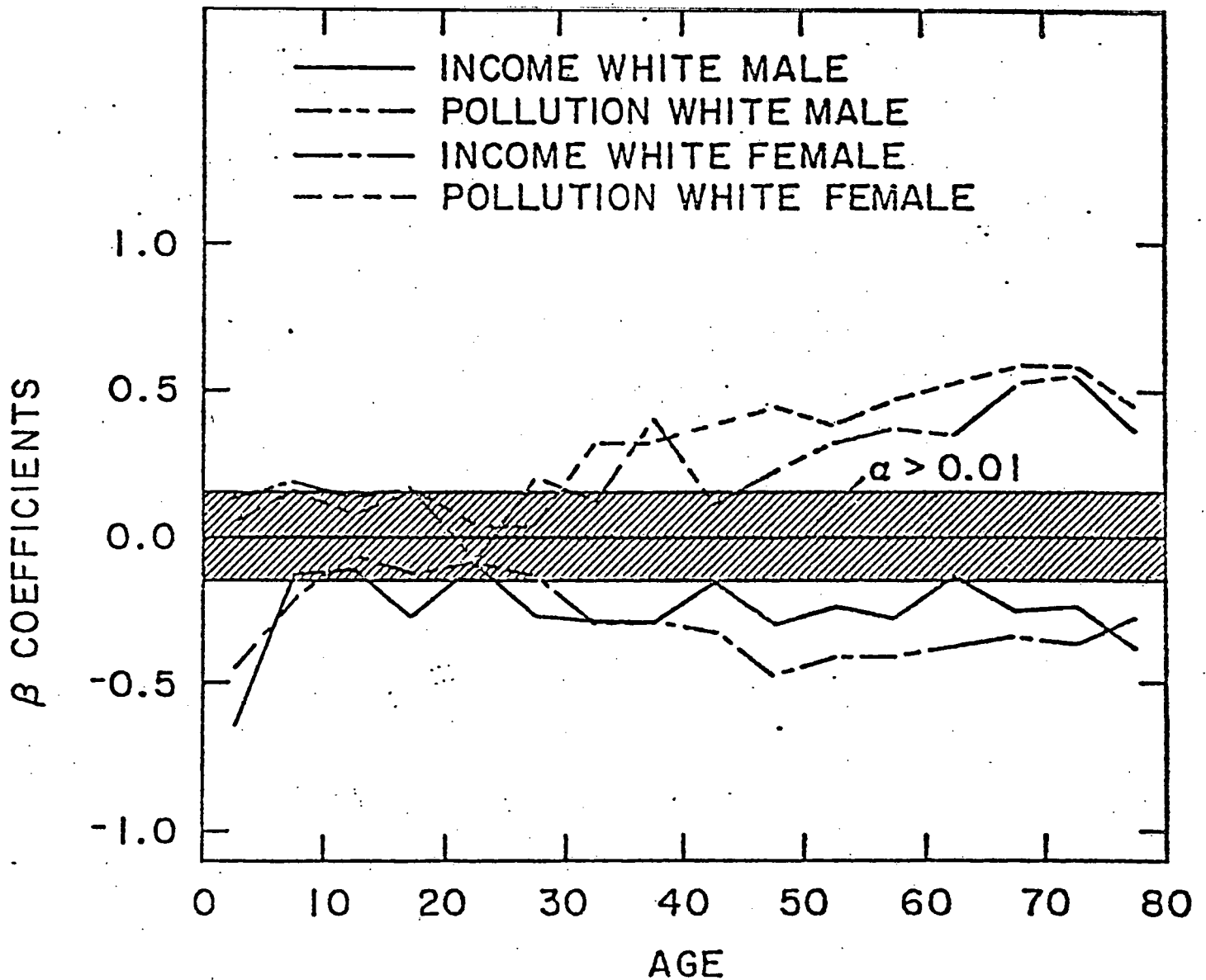
#### 3.4.5 MALIGNANT NEOPLASMS

The effect of income and pollution on malignant neoplasms is very complex (Figure 10). For both sexes, pollution appears as a positive influence on increasing cancer mortality past age 30. Income shows a positive effect for females past age 55. For males, there is a dual effect: a positive effect of income between ages 30 and 55 and a deleterious effect after age 65. Occupationally related diseases and greater industrial exposure of lower income white male groups might explain these findings. However, further study would be needed to confirm such conclusions. Malignant neoplasms are the only disease category we have examined that shows an income-dependent sex difference. Total mortality also shows this pattern.

#### 3.5 ANALYSIS OF THE EFFECTS OF INCOME AND POLLUTION BY AGE

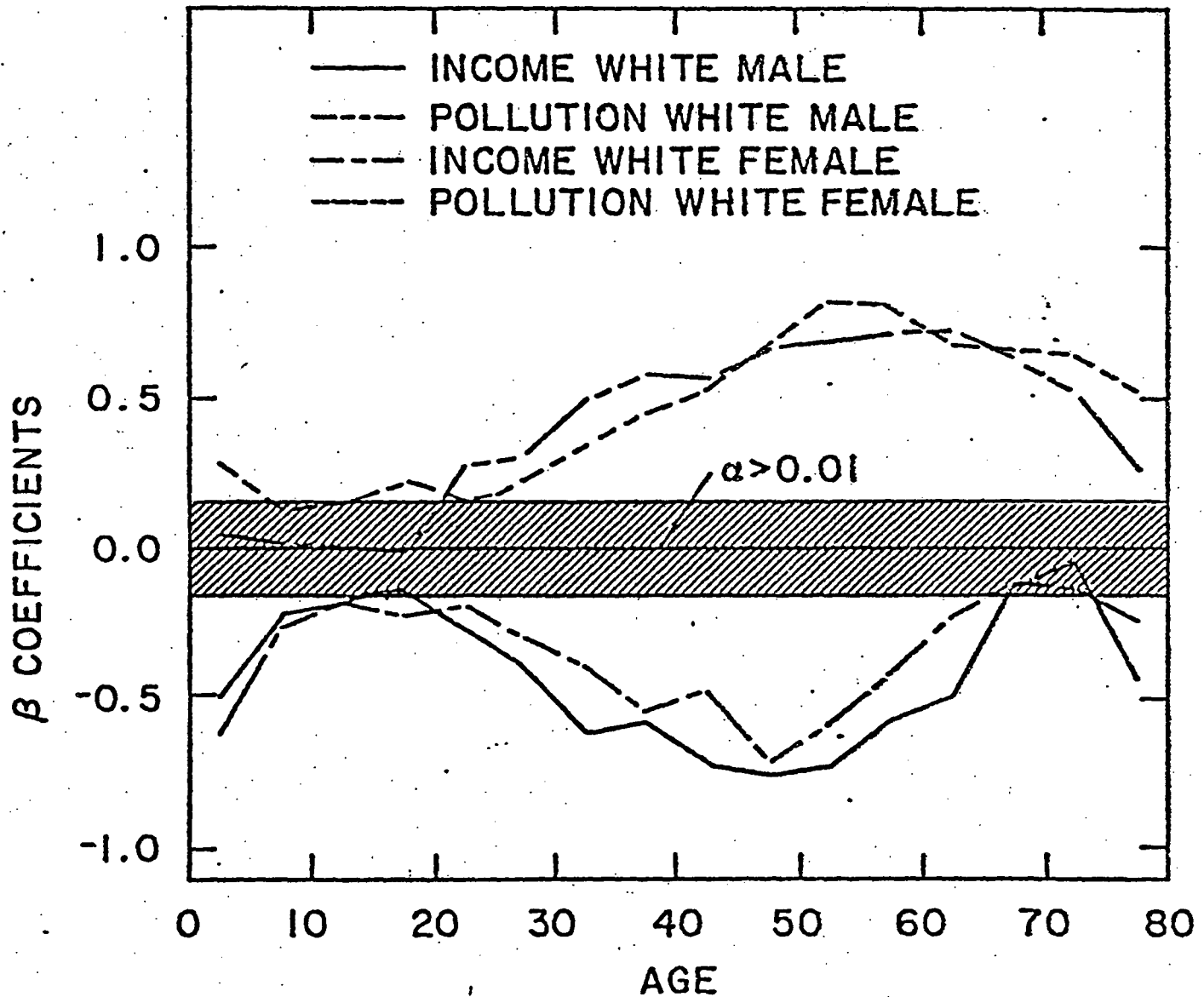
Comparison of the effects of income and pollution by age cohorts on the two migration variables identifies interesting similarities

FIGURE 7  
THE COMBINED EFFECTS OF  
INCOME AND POLLUTION BY  
AGE ON INFLUENZA AND  
PNEUMONIA



N.B. POINTS PLOTTED AT THE MID POINT OF EACH 5 YEAR AGE INTERVAL

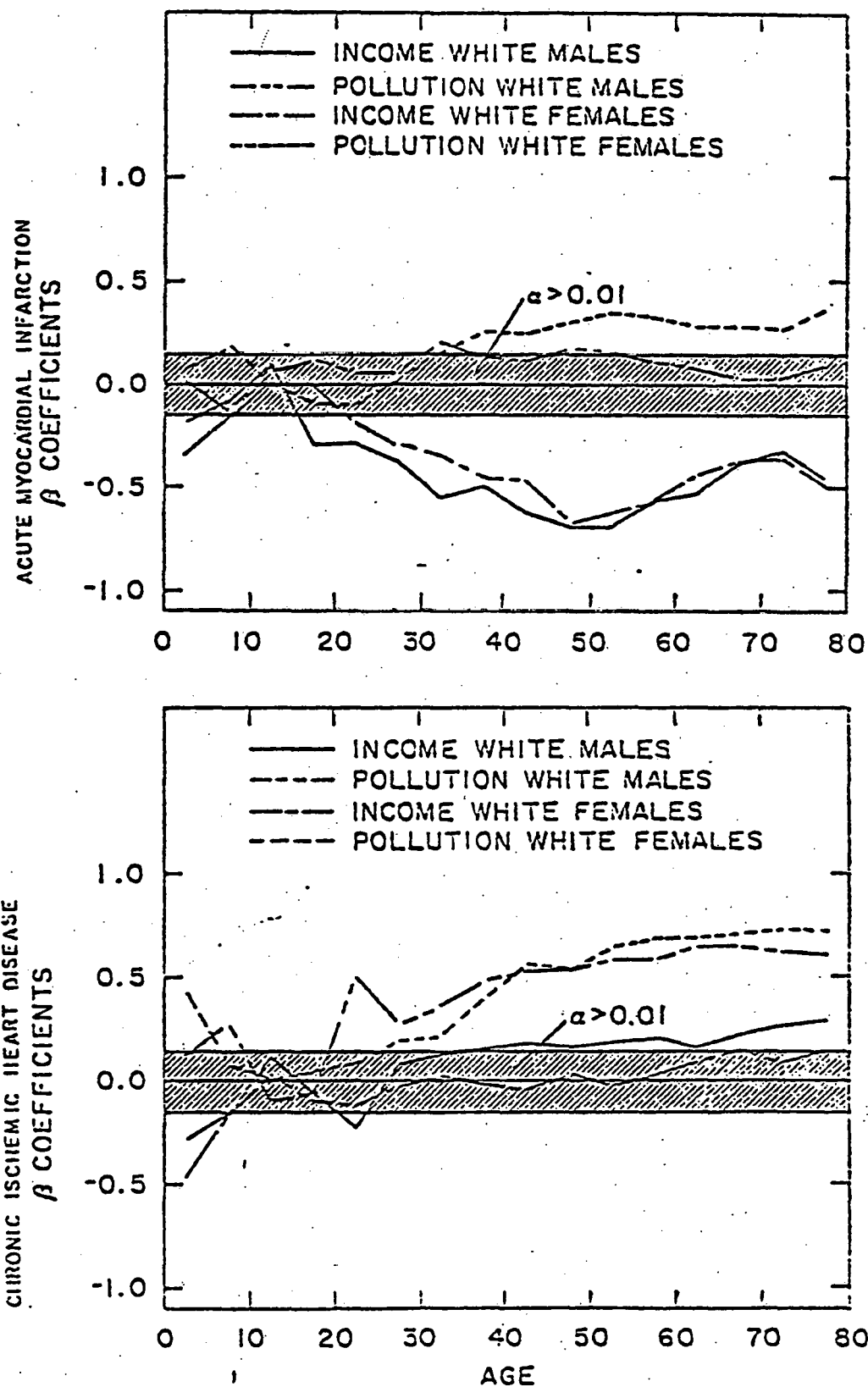
FIGURE 8  
THE COMBINED EFFECTS OF  
INCOME AND POLLUTION BY  
AGE ON CARDIOVASCULAR  
DISEASES



N.B. POINTS PLOTTED AT THE MID POINT OF EACH 5 YEAR AGE INTERVAL

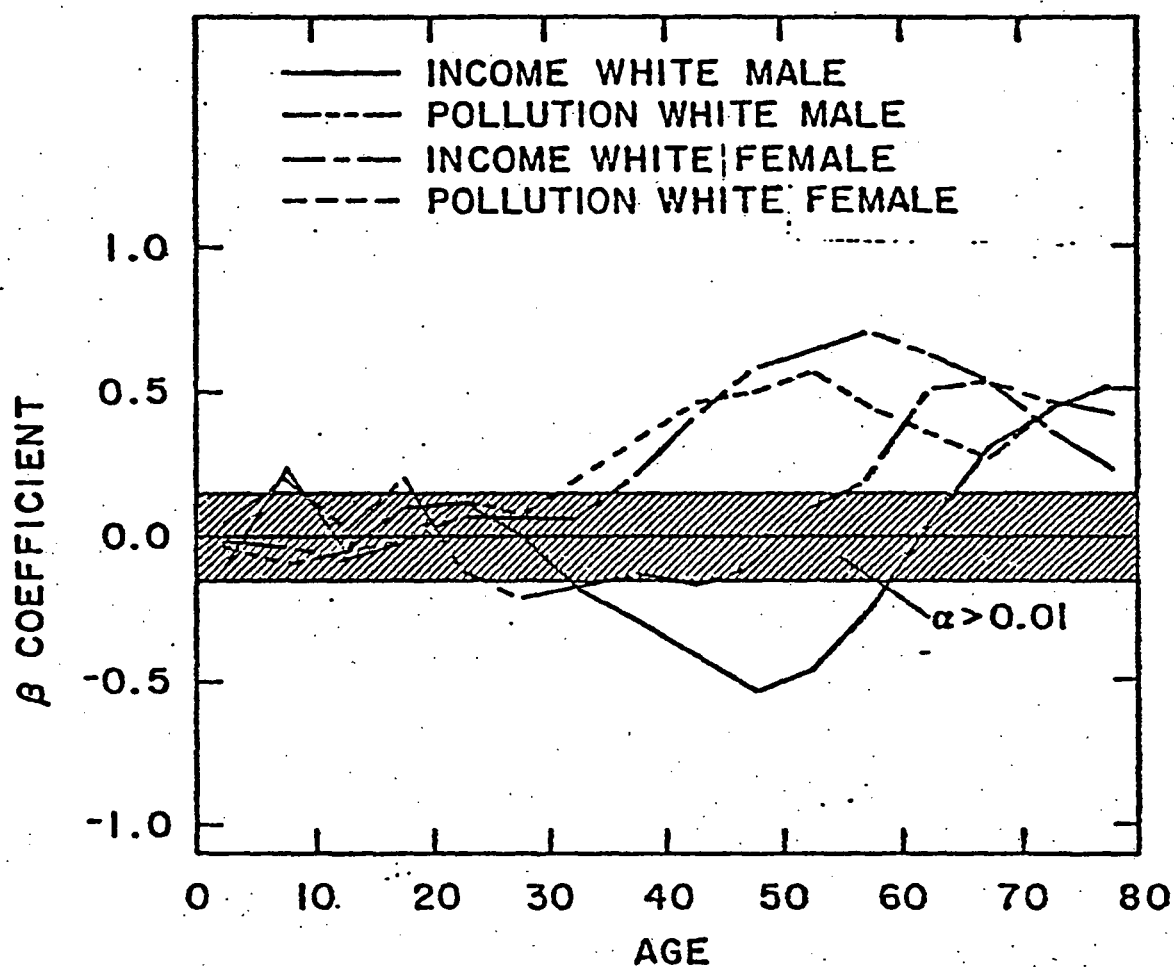
FIGURE 9

THE COMBINED EFFECTS OF  
INCOME AND POLLUTION BY  
AGE ON ACUTE MYOCARDIAL  
INFARCTION AND CHRONIC  
ISCHEMIC HEART DISEASE



N.B. POINTS PLOTTED AT THE MID POINT OF EACH 5 YEAR AGE INTERVAL

FIGURE 10  
THE COMBINED EFFECTS OF  
INCOME AND POLLUTION BY  
AGE ON MALIGNANT NEOPLASMS



N.B. POINTS PLOTTED AT THE MID POINT OF EACH 5 YEAR AGE INTERVAL

and differences. Up to age 55, the effect of income on net in migration parallels quite closely the effects of pollution on net out migration. Beyond this age, pollution maintains its influence on net out migration while income loses its significance in explaining net in migration. This general pattern supports the notion that retirement, as an intervening variable, reduces the importance of income as an attraction while pollution continues to motivate individuals to move out of an area. Upon examining the effect of pollution in retarding net in migration and of income in encouraging net out migration, we see once again a very similar pattern of approximately the same level of strength. Only between the ages of 15 and 30 is any difference noted. Here pollution loses significance while income continues to be a good explanatory variable. The high mobility of this age group (see Figure 11) and its attraction to high income or employment areas is consistent with this observation.

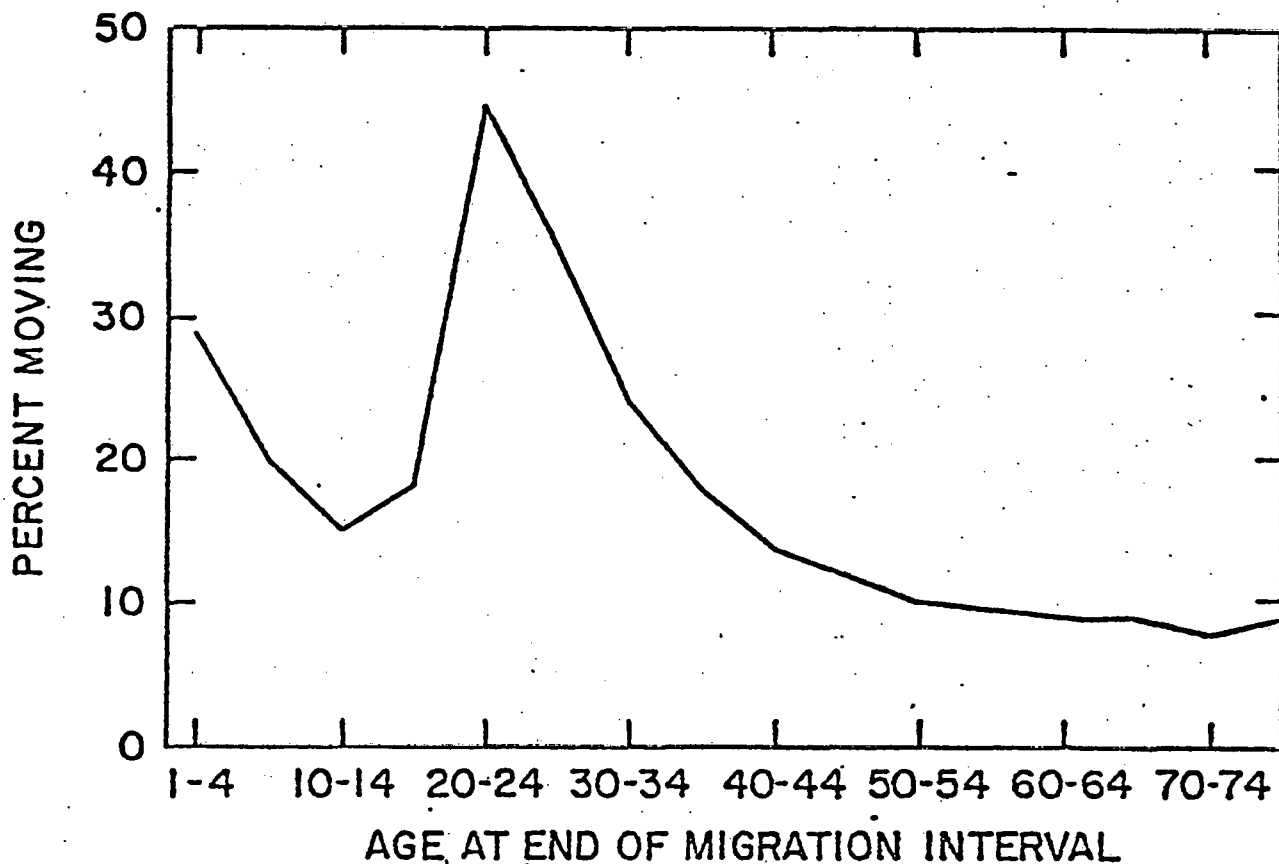
Pollution has shown a consistent positive effect on mortality which is significant after age 30. Cardiovascular diseases and malignant neoplasms show this same basic pattern. Within the cardiovascular group, chronic ischemic heart disease is related only to pollution. The income variable shows a protective effect but also displays an unexpected sex difference. The positive effect of income can be specifically traced to cardiovascular diseases and the other disease category. The sex difference can be traced to malignant neoplasms and the other disease category.

#### 4. CONCLUSIONS

The model used in this study has been successful in showing relationships between population size, migration flows, mortality rates, and the related variables of median family income and emission of air pollutants per square mile. The strong correlation between income and pollution found in our study ( $R = 0.605$ ) requires that any interpretation of demographic change and health effects cannot be based on one of these variables alone. Pollution measurements are meaningless in the absence of economic data. Furthermore, the observation that income and pollution are linearly correlated over only a certain range suggests the importance of urban/rural differentiation. Urban areas show a pollution-to-income ratio which is different from the rural suburban gradient. This apparent nonlinearity of the pollution-income relationship for urban areas warrants further investigation.

In determining population size, income and pollution act not only through mortality but also through migration and the previous cohort as base population. The migration variable is clearly a more influential determinant of population size (and population composition) than is mortality. The strong interrelationships between population size, the previous cohort, income, and pollution suggest that bidirectional effects are present. Not only is population size determined by income and pollution, but conversely, income and pollution are determined by population size through changes in the density of the population. Density, as a complex entity logically appears to be the primary influence in social, economic, and environmental modeling.

FIGURE 11  
PERCENT OF POPULATION  
RESIDENTIALLY MOBILE IN ONE  
YEAR IN THE UNITED STATES BY  
AGE: CIRCA 1970\*



\* ADAPTED FROM LONG, LH, AND CG BOERTLEIN  
THE GEOGRAPHIC MOBILITY OF AMERICANS: AN INTER-  
NATIONAL COMPARISON: CURRENT POPULATION REPORTS.  
SPECIAL STUDIES SERIES P-23. NO. 64 U.S. BUREAU  
OF CENSUS, WASHINGTON, DC.



As expected, we found a strong age dependence in our study. Since there was little or no explanatory power of our model between the ages of 15 and 30, neither income nor pollution can be regarded as major determinants of population size or other PDM variables.

The observed influence of income and pollution on total mortality proved to be nonspecific. The contributory effect of income on infectious diseases such as influenza and pneumonia seems significantly associated with young and older ages. Other influences of income are observed in our broadly defined group, other diseases. A positive effect of income on malignant neoplasm is evident at very old ages; however, no logical explanation for this phenomenon can be advanced at this point. The contribution of income to acute myocardial infarction is negative, and is supported by findings from other researchers.

The pollution effect can be traced at two different levels: (1) a very weak effect in increasing mortality at younger ages, and (2) a nonspecific effect on increased cardiovascular disease and malignant neoplasm. For the 0-4 age group the effect of pollution is highly income dependent (i.e., evident only in high-income levels exposed to high pollution). On the other hand, an effect is not detectable in low-income high-pollution levels. The pollution effect on chronic diseases can be traced to chronic ischemic heart disease (IDCA-412). Because this specific cause of death shows a strong association with pollution levels in conjunction with its rising incidence as a major cause of death, the need for more extensive research in this area is clearly mandated. Specifically, research on the definition of this disease, analyzing multiple causes of death, and standardization of diagnosis and reporting is critically needed in this area. Our findings show that an association of pollution with malignant neoplasms is present, that it is age dependent and non-site specific.

By close examination of the interaction of two significant variables-- median family income and computed county level emissions of air pollution-- a better understanding of the effects of energy production and consumption on contemporary American society has been initiated. Without making ecologically fallacious inferences concerning cause and effect, we can conservatively conclude that both complementary and competitive energy-related influences are significantly associated with fluctuations in demographic and vital rates of the U.S. population. With a specific interest in health, these associations are observed to vary differentially by age and cause of death. Finally, while this specific investigative strategy has only been partially explored by this analysis, its utility as a basic research tool and as a guide for the formulation of health, environmental, and energy policy is clearly evident.

## BIBLIOGRAPHY

- [1] C. L. Beale, The Revival of Population Growth in Nonmetropolitan America, Economic Research Service, U.S. Department of Agriculture, ERS-605, 1976.
- [2] G. K. Bowles, C. L. Beale, and S. L. Lee, Net Migration of the Population, 1960-1970, by Age, Sex, and Color, Parts 1-5, University of Georgia, Athens, 1975.
- [3] S. R. Bozzo, F. Galdos, K. M. Novak, and L. D. Hamilton, Medical Data Base: A Tool for Studying the Relationship of Energy-Related Pollutants to Ill Health, BNL 50840, 1978.
- [4] E. A. Cherniavsky, Topical Report: Brookhaven Energy System Optimization Model, Informal Report BNL 19569, 1974.
- [5] R. H. Daw, Some Statistical Aspects of Mortality from Degenerative Heart Disease, Institute of Actuaries Journal, 8(1), 69-100 (1954).
- [6] R. Doll, C. Muir and J. Waterhouse (editors). Cancer Incidence in Five Continents, Vol. II, Switzerland: International Union Against Cancer, 1970.
- [7] F. R. Drysdale and C. E. Calef, The Energetics of the United States of America: An Atlas, BNL 50501, 1976.
- [8] O. D. Duncan, Introduction to Structural Equation Models, Academic Press, New York, 1975.
- [9] O. D. Duncan, Path analysis: sociological examples, Am. J. Sociol. 72, 1-16 (1966).
- [10] R. A. Easterlen, On the relation of economic factors to the recent fertility decline, Presented at Population Assoc. Am. Annu. Meet., Chicago, April 1965.
- [11] P. E. Enterline, Causes of death responsible for recent increases in sex mortality differentials in the United States, Milbank Mem. Fund Q. 39, 312-28 (1961).
- [12] S. J. Finch, and S. C. Morris, "Consistency of reported effects of air pollution on mortality" BNL 21808-r2, Biomedical and Environmental Assessment Division, Brookhaven National Laboratory, Upton, New York, 1977.
- [13] R. Freeman, "The sociology of human fertility: A trend report and bibliography", Current Sociology V: X-XI, No. 2, 1961-62.
- [14] C. Goldscheider, Population, Modernization and Social Structure, Little, Brown, Boston 1971.

- [15] J. J. Gregor, Intra-urban Mortality and Air Quality: An Economic Analysis of the Costs of Pollution Induced Mortality, Pennsylvania State University, 1977.
- [16] L. D. Hamilton, "Health effects of air pollution" BNL 20743. Conference on Computer Support of Environmental Science and Analyses, United States Energy Research and Development Administration, Albuquerque, New Mexico: July 9-11, 1975.
- [17] ISSA. Workshop report: International symposium on sulfurs in the atmosphere, Subrovnik, Yugoslavia, 1977 (to be published in Atmospheric Environment, January 1978.
- [18] L. B. Lave and E. P. Seskin, Personal communication to S. J. Finch, November 9, 1976.
- [19] L. B. Lave, and E. P. Seskin, An analysis of the association between U.S. mortality and air pollution, J. Am. Statistical Assoc. 68, 284-90 (1973).
- [20] C. C. Li, Path Analysis: A Primer, The Boxwood Press, Pacific Grove, 1975.
- [21] L. H. Long, and C. G. Boertlein, "The geographical mobility of Americans: An international comparison", Current population Reports. Special Studies, Series P-23, No. 64, Washington, D.C.: Government Printing Office (Census).
- [22] M. G. Morgan, S. C. Morris, A. K. Meier, and D. L. Shenk, "A probabilistic methodology for estimating air pollution health effects from coal-fired power plants", accepted for publication in Energy Systems and Policy (in press), 1977.
- [23] S. H. Preston, Mortality Patterns in National Populations, Academic Press, Inc., New York, 1976.
- [24] R. Ropetto, "The interaction of fertility and size distribution of income", cited in N. Birdsall, "Analytical approaches to the relationship of population growth and development", Population and Development Review, March and June 1977.
- [25] W. Rich, Smaller families through social and economic progress, Washington, D.C., Overseas Development Council, 1973.
- [26] W. A. Sevian, The Energy System Network Simulator (ESNS): General Description and Sample Analysis, BNL 50492, 1975.
- [27] S. L. Syme, N. O. Borhani, and R. W. Buechley, "Cultural mobility and coronary heart disease in an urban area", American Journal of Epidemiology, (82)2, 1966.
- [28] R. L. Tanner and W. H. Marlow, "Size discrimination and chemical composition of ambient air borne sulfate particles by diffusion sampling", (in press) Atmospheric Environment, 1977.

- [29] U.S. Bureau of Census, Statistical Abstract of the United States, 1975 (96th edition), Washington, D.C.: Government Printing Office, 1975.
- [30] U.S. National Center for Health Statistics, Vital Statistics, Washington, D.C.: Government Printing Office, 1963-74.
- [31] U.S. Bureau of Census, U.S. census of the population 1960 and 1970, vol. I, Current Population Reports, page 25, No. 545, 1961.
- [32] L. M. Verbugge, "Sex differences in illness and death in the United States", The Johns Hopkins University Center for Metropolitan Planning and Research, Baltimore, 1975.
- [33] R. E. Weiss, et. al., "Sulfate aerosol: Its geographical extent in the Midwestern and Southern United States", Science, Vol 195, No. 4282, page 979, 1977.
- [34] W. Winkelstein, S. Kantor, E. W. Davis, C. S. Maneri, and W. E. Mosher, The relationship of air pollution and economic status to total mortality and selected respiratory system mortality in men. I. Suspended Particulates, Arch. Environ. Health 14, 162-72 (1967).
- [35] R. Zentgraf, S. R. Bozzo, and F. Galdos, Correlation Between Emissions and Concentration Data for Total Suspended Particulates, SO<sub>2</sub> and SO<sub>4</sub>, BNL Informal Report, in press, 1978.