An Ecological Study of Demographic and Industrial Influences on Cancer Mortality Rates in Texas[1]

John K. Thomas
Department of Rural Sociology, Texas A&M University, College Station, TX 77843-2125

Lawrence B. Noel, Jr.
Department of Veterinary Anatomy and Public Health, Texas A&M University, College Station, TX 77843-4458

Joseph S. Kodamanchaly
Department of Agricultural Economics and Rural Sociology, The Pennsylvania State University University Park, PA 16802

Abstract

Four ordinary least squares regression models were run for age-, race-, and sex-adjusted cancer mortality rates, standardized by the direct method. Digestive cancer, genital cancer, lymphatic and hematopoietic cancer, and urinary cancer rates were based on the average number of cancer related deaths for the period 1986 to 1994 and the 1990 size of population subgroups in 254 Texas counties. The four cancer rates were highly intercorrelated indicating that particular counties had high rates for many of the four cancer groups. Black proportion of population and urban county status had statistically significant influences on high cancer mortality rates in all of models. Median family income was inversely related to cancer mortality rates in all of the models, except that of urinary cancer. Contrary to expectations, Hispanic proportion of county population, level of manufacturing employment, accumulated pounds of toxic chemical wastes, and number of insecticide-treated acres had unimportant influences on cancer death rates. Foreign-born proportion of county population was associated with only digestive cancer mortality. Future research at the individual level in high death-rate counties is needed to better identify causal factors, and to improve variable measurement and model specification.

Keywords: *cancer mortality rates, agricultural pesticides, industrial factors, Texas*

Introduction

The ubiquity of pesticides and other toxic chemicals in our environment has caused much public concern about their effects on human health and safety (Szasz 1994; Andelman and Underhill 1987; Gordis 1988; Gots 1993). Since farm use of pesticides peaked around 850 million pounds in 1980, they have varied between 658 million pounds to 786 million pounds (US Environmental Protection Agency 1997). Very few pesticides have been tested for their toxicological effects on people, yet at least 70 among the almost 2,800 pesticide products that are registered for agricultural use have chemical agents which are known to cause cancer, birth defects, or neurological problems (Bullard and Wright 1993). In addition to these toxins, an estimated 1.4 billion tons of regulated hazardous chemical wastes are produced yearly by manufacturing companies in the United States, and 430 million tons of unregulated industrial wastes containing heavy metal and organic compounds are annually released into the environment (Gerrard 1994).[2]

Because few chemicals in the national waste stream have well understood effects on humans, ecological and individual-level studies on the epidemiological effects of human exposure to chemical waste toxins have increased during the past two decades. Although many of these studies unveiled connections between toxic chemical exposure and the incidence of cancer (Blot and Fraumeni 1976; Goldman 1991; Gottlieb, Shear, and Seale 1982; Gould 1986; Lave 1972; L.J. Phillips 1992; Stockwell, Sorenson, Eckert, and Carreras 1993; Zimmerman 1995), their findings were uniquely limited because of their level of analysis. Ecological studies generally have a national or regional scope that does not provide any information about the patterns of exposure and mortality at the county level for particular states where there is high prevalence of cancer incidence and large volumes of carcinogens in chemical wastes. Individual-level studies, in the form of case-record and cohort designed research, are based on detailed health and medical history data of people selected from local or community geographical units. These studies,

especially the studies reviewed by Marsh and Caplan (1987), controlled confounding factors, such as tobacco, alcohol and drug consumption, and pregnancy, that ecological studies were unable to measure (Hogue and Brewster, 1988). Individual-level studies produce, however, findings that are location specific and can not be generalized to larger geographical areas and populations.

In this study, an exploratory ecological design was employed to identify demographic, economic, and toxic chemical factors that might influence cancer mortality rates in Texas. Reasons for conducting an ecological study were the following: it was less costly than other epidemiological designs, it used secondary data which are readily available at the county level of analysis, it did not require *a priori* knowledge of an area's morbidity and mortality patterns, and it provided a logical first step to identify specific counties with high incidences of cancer mortality in order to conduct future detailed individual-level studies (Morgenstern 1995, Szklo 1988).[3] The study sought to answer three questions: (1) Do socio-economic characteristics of a county influence site-specific (i.e., in reference to the human body) rates of carcinogenic mortality? (2) Does the degree of county urbanness influence site-specific carcinogenic death rates? and (3) Do factors such as the application of agricultural pesticides and the volume of toxic chemical waste emissions influence site-specific rates of cancer mortality? The first two questions address characteristics (e.g. ethnic composition, median family income, etc.) that epidemiologists and others have often used individually or in combination as confounders of mortality rates (Morgenstern 1995). The third question regards industrial influences that researchers are giving increased scrutiny.

Toxic Chemicals in Texas Counties

Comprehensive data on pesticides and toxic chemicals in the environment have become available only recently. Regulated by the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), agricultural pesticides include the following: herbicides, insecticides, fungicides, nematocides, and chemicals targeted to more specific pest eradication, such as araricides (spiders). Other classifications of chemicals include defoliants, desiccants, disinfectants, plant and insect growth regulators, and fumigants (Moses et al. 1993). Herbicides and insecticides are by far the most applied pesticides by volume.

The total volume of conventional pesticide usage (agricultural and non-agricultural) declined nationally from a high of 1,487 million pounds in 1979 to approximately 1,200 million pounds in 1995 (Aspelin 1997). Much of the decline resulted from increased government regulation and higher product costs. Within this overall downward trend, herbicide usage increased nationally from 41 percent to 46 percent of the total volume of pesticides applied. Insecticide usage declined, however, from 17 percent to 11 percent of the total national volume. The agricultural share of pesticide usage has been disproportionately high. It rose from 74 percent in 1979 to 79 percent in 1995. The combined herbicide and insecticide proportion of agricultural usage has varied in a small range from 62 percent (or 680 million pounds) in 1979 to 59 percent (or 552 million pounds) in 1995 (Aspelin 1997).[4]

Indiscriminate killing of beneficial insects aside, the application of pesticides causes several problems that exacerbate human exposure potential. Application technologies involving open-system mixing, hand-held sprayers, field flagmen, and open-cockpit planes and tractors increase risks of splash (skin and eye contact) and inhalation exposure (Thomas and Ladewig 1993). Studies show also that 85 to 90 percent of applied pesticides is dispersed off-target to air, soil and water through drift, runoff, volatilization, and off-gassing (Moses et al. 1993). For example, the EPA reported in 1988 that 46 pesticides were detected in the groundwater of 26 states; 18 of these pesticides were at levels that exceeded health advisory levels (Kellogg, Maizel and Goss 1992). Texas ranked fifth nationally in the number of acres (6.4 million) which had a high risk of ground water contamination by pesticides (Kellogg, Maizel and Goss 1992). Because of high toxicity levels, biopersistence, and nonpoint pollution problems, the EPA has banned chlorinated hydrocarbons such as DDT, aldrin, dieldrin, and heptachlor, and decertified other chemicals such as arsenic acid (a desiccant) for agricultural use. Moses and his associates (1993) provide an exhaustive overview of research that addresses the health effects in particular populations exposed to pesticides. Most of the epidemiological research has focused, however, on farm owners/operators and ignored farm workers.

One of the most successful Congressional efforts to improve the identification and monitoring of industrial toxins in the national waste stream involved the passage of the 1986 Emergency Planning and Community Right-to-Know Act (EPCRA). It authorized the annual collection and provision to the public information about the presence and release of hazardous and toxic chemical wastes and mandated development of the Toxic Release Inventory (TRI). Since 1988, the first reliable year of TRI data collected by the Environmental Protection Agency (EPA), Texas has ranked annually either first or second in the total volume of toxic chemicals released by manufacturers in standard industrial codes 20 to 39 who participated in the TRI (US Environmental Protection Agency 1993, 1996).[5]

Researchers have recently used the TRI to indicate sources and locations of potential exposure risk (Perlin, Setzer, Creason and Sexton 1995; Pollock and Vittas 1995; Ringquist 1997; Thomas, Kodamanchaly and Harveson

1998). TRI facilities (approximately 23,000 nationally) are much more numerous than other sources of toxic wastes, such as the 3,900 storage, treatment, and disposal facilities (STDF) monitored under the Resource Conservation and Recovery Act , and the 1,200 Superfund sites identified by the Comprehen-sive Environmental Response, Compensation and Liability Act (Gerrard 1994).

Texas' approximate 1,200 TRI facilities reported 408 million pounds of toxic chemical releases, about 11 percent of the 1990 national volume.[6] Petrochemical companies in the state accounted for most (57%) of these releases. Of the 250 toxic chemicals listed in the TRI prior to 1995, 121 chemicals were known carcinogens, based on criteria set forth in the Occupational Safety and Health Administration's Hazard Communication Standards (US Environmental Protection Agency 1996). The total volume of all TRI chemical wastes declined in Texas from 310 million pounds in 1988 to 250 million pounds in 1994; carcinogenic releases declined from 44 million pounds to 24.6 million pounds in this period (Thomas and Harveson 1997). Fifty carcinogenic chemicals were released in Texas to air, injected into wells, placed in landfills, and emitted to water. In 1990, the greatest released volume (> 3 million pounds) of carcinogens included benzene, formaldehyde, lead, acrylonitrile, tetrachloroethylene, and 1,3-butadiene. TRI facilities are located predominately in metropolitan areas and in Texas Gulf Coast counties. These facilities have historically employed a large number of chemical and petroleum workers and have produced annually the largest volume carcinogenic releases (Thomas and Harveson 1997).

Empirical Evidence and Research Hypotheses

The percentage of cancer related deaths in Texas increased from 7 percent in 1935 to 23 percent in 1993, making cancer is the second leading cause of death (Texas Cancer Registry 1996). However, little is known about the relationship between cancer mortality and exposure to pesticides and toxic chemicals in the state (Napton and Day 1992; Thomas, Kodamanchaly and Harveson 1998). Much of this lack of knowledge is due to common problems of measurement inaccuracies that make exposure to toxic chemicals difficult to estimate, to coverage inconsistencies in secondary data (Anderton 1996; Zimmerman 1994), and to a comprehensive measurement of factors that differentially affect human susceptibility (Sexton et al. 1993).

Based upon empirical evidence reported in past studies, eight research hypotheses were tested for each of four site-specific groups of cancer: digestive, genital, lymphatic/ hematopoietic and urinary cancers. These general groups facilitated the aggregation of incidences of cancer mortality to sufficiently calculate stable rates at the county level. Although these groups have been employed elsewhere (Stokes and Brace 1988), a drawback to their use is that individual cancers within each group have different etiologies and characteristics. Respiratory cancer was not included among the four groups because of unmeasured effects of tobacco-smoking behavior on lung cancer incidence.

(H_1) *Black percentage of population positively influences rates of cancer*. At most ages, Blacks experience greater mortality rates than Whites for most causes of death (Goldman 1991 Hummer 1996; Sorlie, Backlund and Keller 1995). More than half of this racial difference in mortality is due to cardiovascular disease and cancer (Rogers 1992). Nationally, Black males experience higher rates of lung, esophagus, and prostate cancer than White males (Rios, Poje and Detels 1993; Rogers 1992). Among women, Blacks older than 15 years of age are 2.6 times more likely than Whites to die from cervical cancer (Centers for Disease Control 1990). Socioeconomic factors explain most of these differences in death rates according to Hummer (1996). Blacks with lower socio-economic status are less likely than whites to have health insurance, to receive preventative and follow-up treatment services, to be less knowledgeable about the signs of cancer and potential treatment, and to use tobacco. However, other research indicates that after statistical controls are applied for demographic factors, family size, and income, gaps in cancer mortality rates narrow between the two races (Rogers 1992). In Texas, Black males had higher cancer mortality rates in 1992 than Anglo and Hispanic males; the highest rates for all male racial groups were for prostate, lung, and colon cancers (Texas Cancer Registry 1998). Black females had rates second to those rates of Anglo females; the highest rates for all women were for breast, lung and colon cancers (Texas Cancer Registry 1998).

(H_2) *Hispanic percentage of population positively influences rates of cancer*. Hispanics comprised 21 percent and 26 percent of the Texas population in 1980 and 1990, respectively. However, 39 of 254 Texas counties had more than 40 percent Hispanic population in 1980 and this number increased to 44 counties in 1990 (Murdock and Hoque 1992). Many of these counties, which are located in the western and southwestern areas, have large concentrations of Hispanics who are employed in agricultural and manufacturing jobs along the Rio Grande River. Federal laws and programs do not protect these workers against exposure to toxic chemicals (Goldman 1991; Rios, Poje and Detels1993).

(H_3) *Foreign-born percentage of population positively influences rates of cancer*. Texas ranked third in the number of foreign-born population (1.2 million), who entered the United States from 1980 to 1990 (Murdock and Hoque 1992). Limited available evidence indicates that foreign-born eth-

nicity is associated with specific cancers. In their national study of cancer mortality rates for White males in the period 1975 to 1980, Stokes and Brace (1988) found that foreign-born ethnicity produced the largest, most consistent, and positive influences on digestive, genital, urinary cancers. Foreign-born ethnicity was negatively related, however, to respiratory cancer mortality rates and had no influence on lymphatic cancer.

(H_4) *Median family income negatively influences rates of cancer*. Level of income is related to cancer mortality through differences in individuals' life course experiences. Such experiences include consumer nutrition, financial inability to change residences contiguous to hazardous sources (Mohai and Bryant 1992), to afford adequate health insurance, and to pursue timely medical treatment (Sorlie, Backlund and Keller 1995). Thus, low-income families have life styles and situations, mixed with limited resources, that potentially expose them to greater carcinogens and other environmental hazards (Bullard and Wright 1993). Scant empirical evidence indicates, however, that median family income is positively associated with only respiratory cancer mortality and is negatively related to genital cancer mortality (Stokes and Brace 1988). Other research shows it has positive influences on the rate for all cancers and on the breast cancer death rate (Goldman 1991).

(H_5) *Urban county status positively influences rates of cancer.* Although research has shown that urban and rural cancer trends are converging, urban rates still exceed rural rates, particularly for white females who live in central cities (Greenberg 1984). Densely populated areas with high concentrations of manufacturing facilities and employment produce more hazardous wastes than areas with fewer facilities, thereby increasing exposure risk (Pollock and Vittras 1995; Ringquist 1997), illness, and mortality (Glickman and Hersh 1995; Nieves and Nieves 1992; Stockwell, Sorenson, Eckert and Carreras 1993). In a New Jersey study of 194 municipalities, Najem and his associates (1985) observed a pattern of cancer mortality concentrated in cities located in the highly industrialized, densely populated northeastern part of the state.

(H_6) *Level of manufacturing employment positively influences rates of cancer*. Risks of exposure to carcinogens in the workplace are documented often (U.S. Environmental Protection Agency 1996). Some estimates of exposure risk vary from approximately 5 percent to over 25 percent of all cancer deaths (Swanson, Schwartz and Burrows 1988). For example, in the mid-1970s, the National Cancer Institute (NCI) prepared maps that revealed clusters of high incidences of cancer in the highly industrialized Northeast, the Southeast, and Gulf Coast regions (Mason 1975). In a follow-up study to the NCI report, Blot and Fraumeni (1976) found significantly high rates of lung cancer mortality in counties with paper, petrochemical, and transportation industries. Additionally, Pickle and Gottlieb (1980) found that the likelihood of pancreatic mortality increased about two-fold for workers employed by refining and paper manufacturing industries in Louisiana. In Hoover and Fraumeni's (1975) geographical analysis of US cancer mortality from 1950 to 1969, they reported excess rates for bladder, lung, liver, and certain other cancers among residents in counties where chemical industries were most concentrated. More recently, Austin and Schnatter (1983) found a significant excess number of deaths due to brain cancer among White males over 55 years of age who were employees of a Texas chemical plant.

(H_7) *Accumulated pounds of TRI carcinogens positively influences rates of cancer*. By their very definition, chemical wastes reported to the TRI are hazardous to human health (US Environmental Protection Agency 1996). Toxic chemicals in these wastes bio-accumulate, bio-magnify, and biopersist in varying degrees in the environment. In addition to cancers, they can cause genetic and chromosomal mutations, developmental, acute and chronic toxicities, and neurotoxicity (Geschwind et al. 1992; Stockwell, Sorenson, Eckert and Carreras 1993). Najem and others (1995) found a consistent and significant positive association between 8 of 12 cancers studied and the presence of disposal sites for toxic chemical waste.

(H_8) *The number of pesticide treated acres positively influences rates of cancer*. Many farms are exempted under the law because of their operational size. Consequently, an estimated high of 2.5 million employed farm workers are excluded from most occupational safety and health regulations and other protective labors laws (e.g., Fair Labor Standards Act, workers' compensation and unemployment benefits, and social security benefits) (Goldman 1991; Rios, Poje and Detels 1993; US General Accounting Office 1992). This exclusion is estimated to have produced an annual death rate among farm workers that is five times greater than the national rate for all occupations combined (University of California Agricultural Health and Safety Center and the Western Consortium for Public Health 1992).

Ample evidence indicates that rates of leukemia, myeloma, stomach, pancreas, and prostate cancers are greater among farm workers who are exposed to pesticides than among the rest of the population (Goldman 1991; Moses et al. 1993). Some scientists found that herbicide use was positively related to the risk of non-Hodgkin's lymphoma among farmers (Hoar et al. 1986) and to genital, lymphatic, digestive, and respiratory cancer mortality among rural males (Stokes and Brace 1988). They reported further that insecticide use positively influenced respiratory cancer mortality rates among rural males (Stokes and Brace 1988).

Methods

Regressor Variables

Data for this study were compiled from several sources. Four demographic variables were obtained from the US Bureau of the Census (1973, 1983, and 1993). The *average Black percentage* and *average Hispanic percentage* of a county population were calculated each by adding the respective percentages of total county population for the 1970, 1980 and 1990 census periods and dividing each of the totals by three. *Average foreign-born percentage* of the county population was the mean proportion of a total county population that was born of foreign background during each of the three census periods. This segment of the population was assumed to be primarily Hispanics who immigrated from Mexico, although Asian segments have increased since 1980 (Murdock and Hogue 1992). *Median family income* was obtained for each census year, summed, and averaged.[7]

To control for urban influence on cancer mortality, a measure of *urbanness* was constructed using the nine rural-urban influence codes prepared by Ghelfi and Parker (1995). For simplicity, counties were recoded as: (1) 1990 location in large metropolitan area with one million population or more, or location in small metropolitan area with fewer than one million population; (2) adjacency to a large metropolitan area with a city of 10,000 or more, adjacency to a large metropolitan area without a city of at least 10,000, adjacency to a small metropolitan area with a city of 10,000 or more, or adjacency to a small metropolitan area without a city of at least 10,000; (3) not adjacent to a metropolitan area and with a city of 10,000 or more population, or not adjacent to a metropolitan area and with a city of 2,500 to 9,999 population; and (4) not adjacent to a metropolitan area and with no city or a city with a population less than 2,500.

Potential exposure to carcinogens in the environment was crudely indicated by three industrial measures: level of manufacturing employment, volume of toxic chemical wastes released by manufacturing industries, and average numbers of agricultural acres treated with two types of pesticides. None of these measures assessed, however, actual exposure (i.e., amount, duration, vector, etc.) to chemical carcinogens or the health-related responses to exposure.

Because TRI data were not available before 1987, data on *manufacturing employment* were obtained from the US Bureau of the Census for the years 1970, 1975, 1980, 1985, and 1990. Level of manufacturing employment was measured as the five-period average of the number of persons employed in manufacturing industries with standard industrial codes (SICs) of 20 to 39, the same industries required to report to the TRI.

The *volume of carcinogenic chemical releases* was measured in dry pounds and standardized to a common list of 122 chemicals to maintain comparability for the TRI reporting years of 1988 to 1994, and to measure the potential accumulation of these known carcinogenic wastes. A limitation of this measure is that carcinogenic toxicities of waste chemicals are not all equal and they do not cause the same cancers (Stockwell, Sorenson, Eckert and Carreras 1993). Moreover, these chemicals represented less than one-half of the list of chemicals monitored by the EPA prior to 1995 to be hazardous in other ways.

Agricultural acreage data were obtained for four time periods (1974, 1978, 1982, and 1987) from the Census of Agriculture (US Bureau of the Census 1976, 1980, 1984, and 1992).[8] *Insecticide treated acres* was the averaged number of acres to which herbicides were applied during the four agricultural census reporting periods. *Insecticide treated acres* were measured similarly. Use of these pesticide measures had some limitations. Pesticides and other agricultural chemicals such as fertilizers are often applied to the same acres. Consequently, acreage measures can be collinear and non-independent of one another. Data on the applied pounds of particular agricultural pesticides, though preferred, were unavailable at the county level. Furthermore, pesticides vary by toxicity and longevity in the environment. Number of treated acres is a crude measure at best of the environmental presence and hazard posed by these chemicals.

Response Variables

Carcinogenic mortality rates of four site-specific causes of death were computed for each county in 1990. Mortality data were provided for the years 1986 to 1994 by the National Center of Health Statistics (NCHS).[9] Carcinogenic sites in humans were taken from the ninth edition of the International Classification of Diseases (ICDs) and combined according to the site groups identified by Greenberg (1984) and Stokes and Brace (1988). The four site groups appear in Table 1. Other site-specific cancers had too few numbers of deaths reported, particularly for rural counties, by NCHS to be useful in the analysis. Mortality rates based on small numbers of death are unreliable given the large standard errors they produce (Gots 1993; Greenberg 1984).

Rates were based on per 100,000 population. The standardization procedure was conducted in several steps. First, the numbers of death for the years of 1986 to 1994 were totaled and averaged for each county to stabilize severe fluctuations that might have occurred in the numbers of death from year to year (Morgenstern 1995; Shryock and Siegel 1976). Second, the expected number of age-, race-, and sex-adjusted deaths was calculated by carcinogenic cause of

Table 1. Major Groups of Cancer in an Ecological Study of Cancer Mortality in Texas

Site Types of Cancer	ICD Codes*
Digestive	esophagus (150); stomach (151); small intestine, including duodenum (152); colon (153); hepatic and splenic flexurers and traverse colon (153); rectum, rectosigmoid junction and anus (154); liver and intrahepatic bile ducts (155); gallbladder and extrahepatic bile ducts (156); pancreas (157); and peritoneum, retroperitoneum, and other ill-defined sites within the digestive organs and peritoneum (158-159)
Genital	cervix uteri (180); other parts of the uterus (179, 181, 182); ovary and other uterin adnexa (183); other and unspecified female genital organs (184); prostate (185); testis (186); and penis and other male genital organs
Lymphatic and Hematopoietic	Hodgkin's disease (201); lymphosarcoma and reticulosarcoma (200); other malignant neoplasms of lymphoid and histiocytic tissue (202); leukemia and aleukemia (204-208); multiple myeloma and immunoproliferative neoplasms (203)
Urinary	bladder (188); kidney and other unspecified urinary organs (189)

*Source: Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death, Ninth Edition, World Health Organization, Geneva, Switzerland, 1977.

death (Shryock and Siegel 1976). Sizes of county subgroup populations that were reported in the 1990 US Census were used to adjust the rates. Age categories were defined as 0-14 years, 15-64 years, and 65 and older. Racial/ethnic categories were Anglo, Black, and Hispanic (Murdock and Hoque 1992). Other ethnic groups (i.e., Asians, American Indians, and other groups), which comprised 12.8 percent of the Texas population in 1990, were not included in the analysis (US Bureau of the Census 1993). Sex categories were male and female. Finally, the adjusted death rates were standardized using the direct method and the sizes of age, race, and sex subgroup populations in Texas (Shryock and Siegel 1976). Adjusted site-specific cancer mortality rates are reported in Figures 1 through 4.

Analytical Procedures

Bivariate correlation procedures were conducted initially, followed by ordinary least squares (OLS) regression analyses. The statistical significance of each bivariate correlation coefficient was determined for the hypothesis *rho* = 0. Due to the parametric nature of the data, ordinary least squares regression analyses (OLS) were conducted to determine if any of the socio-economic and industrial characteristics of counties significantly influenced the variation in each of the four site-specific cancer mortality rates (SAS Institute, Inc. 1990). Values for the variance inflation factor (VIF > 10) and tolerance (TOL => 0) were computed to determine the occurrence of multicollinearity among the regressor variables (Hamilton 1992). The herbicide and insecticide variables

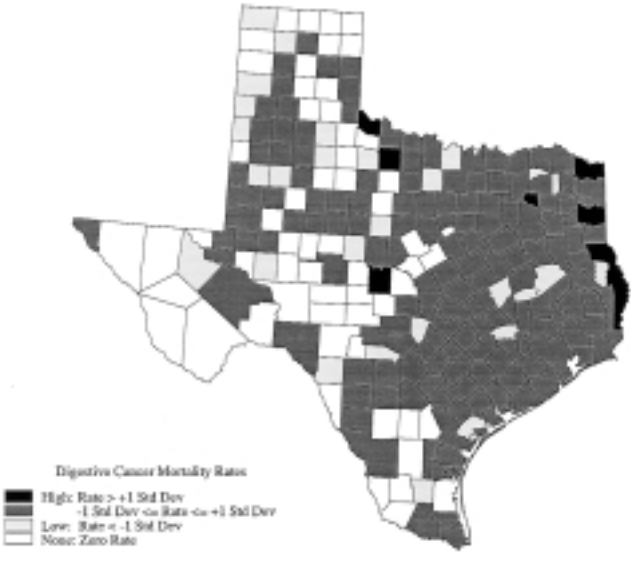

Figure 1. Age-, Race-, Sex-Adjusted Digestive Cancer Mortality Rates for Texas, 1986–1994.

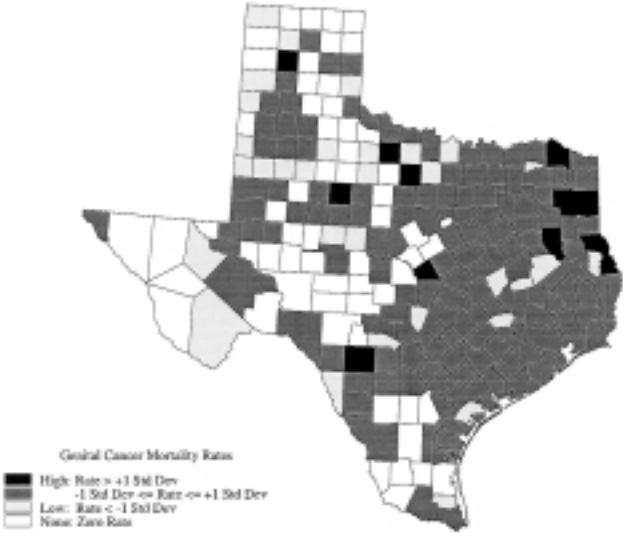

Figure 2. Age-, Race-, Sex-Adjusted Genital Cancer Mortality Rates for Texas, 1986–1994.

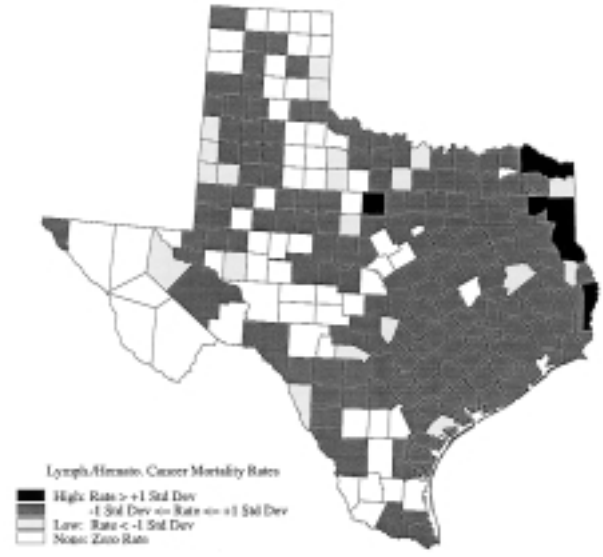

Figure 3. Age-, Race-, Sex-Adjusted Lymphatic/Hematopoietic Cancer Mortality Rates for Texas, 1986–1994.

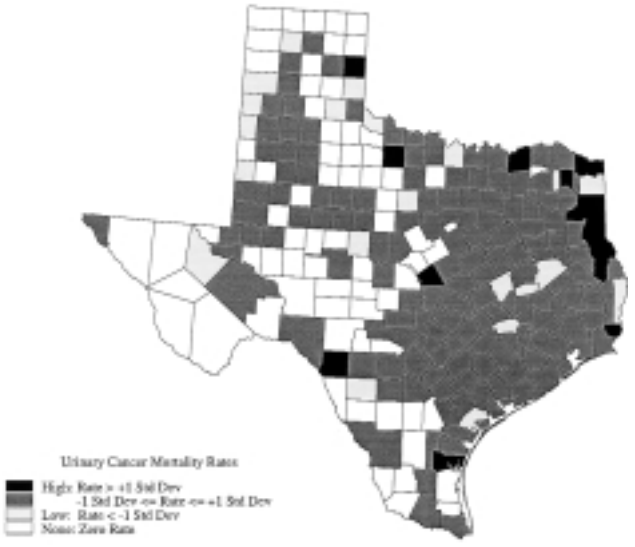

Figure 4. Age-, Race-, Sex-Adjusted Urinary Cancer Mortality Rates for Texas, 1986–1994.

were collinear (r = .849; TOLs < .3; VIFs > 3.9) in each of the cancer mortality models. Although herbicide-related correlation coefficients are reported in Table 2, the variable was deleted from subsequent analyses because its statistical associations with other variables were less than those associations by the insecticide variable.

Death rates tend to have non-normal univariate distributions. Consequently, magnitudes of the Cook's D value and a plot of the residual errors against estimated mortality rates determined case influence (i.e., leverage conditions). Cook's D measures the influence of the i^{th} case on all estimated regression coefficients, or equivalently all *n* predicted cancer

Table 2. Simple Statistics and Bivariate Correlations of Socio-economic and Industrial Characteristics, and Site-Specific Cancer Mortality Rates for Texas Counties.

Variables[a]	BLK	HSP	FBN	INC	URB	MAN	TRI	INS	HER	DIG	GEN	LYM	URI
BLK	1.000												
HSP	- .434‡	1.000											
FBN	- .311‡	.756‡	1.000										
INC	.095	- .464‡	- .283‡	1.000									
URB	- .230‡	.016	- .111	- .491‡	1.000								
MAN	.159†	- .038	.104	.301‡	- .312‡	1.000							
TRI	.140*	- .031	.038	.317‡	- .264‡	.530‡	1.000						
INS	- .071	.211‡	.185†	.028	- .193‡	.024	.083*	1.000					
HER	- .059	.137*	.027	.039	- .116	- .012	.066	.849‡	1.000				
DIG	.416‡	- .249‡	- .201†	.191†	- .428‡	.122*	.133*	.054	.039	1.000			
GEN	.466‡	- .245‡	- .172†	.198†	- .474‡	.148*	.138*	.037	.014	.843‡	1.000		
LYM	.477‡	- .219‡	- .167†	.124*	- .371‡	.113	.105	.075	.082	.636‡	.757‡	1.000	
URI	.459‡	- .231‡	- .184†	.165†	- .385‡	.116	.126*	.041	.038	.592‡	.690‡	.532‡	1.000
Mean	7.6	21.1	3.7	16,353	2.7	3,690	943,679	23,320	39,218	23.9	11.9	11.1	4.6
S.D.	8.9	22.2	4.2	3,334	1.0	16,786	4,654,950	35,915	45,098	19.4	9.5	10.6	4.4

[a]BLK = average Black percentage of population, 1970-90 ; HSP = average Hispanic percentage of population; FBN = average percentage of population that is foreign-born population; INC = average median family income; URB = urban-rural status of a county in 1990; MAN = average employment in manufacturing; TRI = accumulated number of pounds of TRI carcinogenic chemicals, 1988-1994; INS = average number of acres of applied insecticides, 1972-87; HER = average number of acres of applied herbicides, 1972-87; DIG = digestive cancer death rate; GEN = genital cancer death rate; LYM = lymphatic/hematopoietic cancer death rate; URI = urinary cancer death rate. All death rates are per 100,000 for the period 1986-1994. Statistically significant for rho = 0; $p < .05$ (*), $p < .01$ (†), and $p < .001$ (‡), N = 254.

mortality rates. No values were observed greater than one (the absolute cutoff or elimination point), but 9 to 12 cases (i.e., counties) produced values greater than .0157 (i.e., 4/n, where n = 254) in the regression models. The latter threshold value is the size-adjusted cutoff point for unusually influential cases (Hamilton 1992). Often referred to as "outliers," these cases were retained in the analysis for lack of a sufficient theoretical or mismeasurement reason to justify their elimination (Dietz, Frey and Kalof 1987).

A plot of each model's residuals against the predicted mortality rates was conducted to verify OLS regression's homoskedasticity assumption. In addition, the SPEC option in SAS (1990) was used to test if the residual errors were homoskedastic and independent of the regressor variables, and if each model was correctly specified (White 1980). Heteroskedastic variances of residuals lead to biased and inefficient standard error estimates and undermine the rationale for t- and F-tests (Dietz, Frey and Kalof 1987; Hamilton 1992). No condition of heterskedasticity was detected in any of the models. The OLS regression was conducted using the following model:

$$Y_i = \alpha + B_1X_1 + B_2X_2 + B_3X_3 + B_4X_4 + \ldots\ldots + B_8X_8$$

where:
- Y_i = age-, race-, and sex-adjusted death rates for digestive, genital, lymphatic, respiratory, and urinary cancers
- α = y-intercept
- X_1 = average Black percentage of population (BLK)
- X_2 = average Hispanic percentage of population (HSP)
- X_3 = average percentage that is foreign-born population (FBN)
- X_4 = average median family income (INC)
- X_5 = urban-rural status of a county in 1990 (URB)
- X_6 = average employment in manufacturing (MAN)
- X_7 = accumulated number of pounds of TRI carcinogenic chemicals (TRI)
- X_8 = average number of acres of applied insecticides (INS)
- B_i = unstandardized regression weight of each regressor variable

An important limitation of the model should be noted. The model ignores individual-level data that would include health histories and exposure vectors, doses, and durations which would affect when, where, and what cancers occur (Wagener, Selevan and Sexton 1995). The absence of these data from the analysis suggests that caution be exercised with the aggregate county-level measures used in this study and that causal inferences be avoided. Relationships among aggregate data used in ecological models can differ radically when observed at alternative levels of analysis, especially among individual level data (Morgenstern 1995).

Results

Table 2 presents the bivariate correlation coefficients. Discussion of the correlational findings is divided into three sections: associations among the regressor variables, associations among the cancer mortality death rates, and associations between the regressor and response variables. Relationships with the number of herbicide treated acres are reported for only this part of the analysis.

Three interesting patterns emerged among the intercorrelations of the regressor variables. A "metropolitan pattern" was indicated by Black proportion of a county population, which was mostly associated with urban county status and manufacturing employment. Small positive associations between this variable with manufacturing employment and volume of TRI releases were not statistically significant. Black proportion of the population was negatively correlated with Hispanic and foreign-born proportions of population, and negligibly correlated with acres treated with pesticides. These findings support demographic patterns in the state that show counties with large percentages of Black population generally do not have sizeable Hispanic populations, and vice versa (Murdock and Hogue 1992). An "agricultural pattern" was defined by large Hispanic and foreign-born proportions of a county population. Both variables were positively intercorrelated and positively associated with acres treated with pesticides. They were negatively associated with family income and only negligibly related to urban county status, manufacturing employment, and volume of carcinogenic TRI releases. Finally, a "toxic risk" pattern involved positive relationships between industrial measures (i.e., manufacturing employment and volume of carcinogenic TRI releases), between pesticide application measures (i.e., acres treated with insecticides and herbicides), and between urban county status and manufacturing employment measures.

Intercorrelations of cancer mortality rates had large, positive coefficients ($.532 < r < .843$), that were all statistically significant. The strongest associations involved genital cancer mortality rates with digestive and lymphatic cancers; the weakest associations were between urinary cancer mortality rates and the rates of lymphatic and digestive cancer mortality. The magnitude and statistical significance of these relationships were more than coincidental, indicating that cancer mortality could be highly clustered among a specific group of counties in the state. Indeed, a listing (not presented here) and examination of the top ten counties with the highest mor-

tality rates for each site-specific cancer shows that 8 particular counties were ranked in the top 10 counties for two or more cancers. Seven of these eight counties are located along the eastern border of the state. Two of the counties in that area were ranked in the top ten counties for all four cancers. A comparison of Figures 1 through 4 provides further evidence of which counties had cancer mortality rates that were consistently greater than one standard deviation above the mean rate of the state.

Characteristics of counties with high mortality rates can be derived from an examination of the correlations between the regressor and response variables. All site-specific cancer mortality rates exhibited similar patterns of associations with the regressor variables. Each rate was positively related to the Black proportion of a county population, median family income, and urban county status. Digestive and genital cancer mortality rates were positively associated also with manufacturing employment and volume of carcinogenic TRI releases.

Lymphatic/hematopoietic cancer mortality had statistically insignificant relationships with these two industrial variables. Urinary cancer mortality was positively associated with the volume of carcinogenic TRI releases. Contrary to expectations, mortality rates of all the site-specific cancers had statistically significant, negative coefficients with Hispanic and foreign-born proportions of a county population. Moreover, all had negligible associations with the number of acres treated with insecticides and herbicides.

OLS regression results are shown in Table 3. The models' adjusted R Squares varied from .287 to .380. Unstandardized and standardized regression coefficients (**b**) are reported in each model. Attention is focused on the standardized coefficients because they are suited better than unstandardized coefficients for examining the comparative influences of regressor variables. In the *digestive cancer mortality* model, rates were most influenced by urban county status, followed by Black proportion of a county population. Foreign-born proportion of county population and median family income had notable negative influences at slightly higher significance levels ($.05 < p < .10$) on digestive cancer mortality rates and on digestive, genital, and lymphatic/hematopoietic cancer mortality rates, respectively. The regressors behaved similarly in the *genital cancer mortality* and *lymphatic/hematopoietic cancer mortality* models. However, foreign-born proportion of county population had less influence in these models and in the *urinary cancer mortality* model. Only Black proportion of a county population and urban county status had statistically significant relationships with urinary cancer mortality rates. Contrary to the research hypotheses, Hispanic proportion of county population and the industrial variables had negligible influences on cancer mortality rates.[10]

Table 3. Site-Specific Cancer Mortality Rates Regressed Against Socioeconomic and Industrial Characteristics of Texas Counties, 1970-1994.

Independent	Digestive			Genital			Lymphatic			Urinary		
Variables[a]	***B***	**b**	**P>|T|**	***B***	**b**	**P>|T|**	***B***	**b**	**P>|T|**	***B***	**b**	**P>|T|**
Intercept	61.038		.000	29.928		.000	-26.455		.000	10.334		.000
BLK	.515	.234	.000	.310	.291	.000	.420	.352	.000	.162	.328	.000
HSP	-.091	-.104	.286	-.044	-.105	.261	-.031	-.066	.500	-.008	-.041	.671
FBN	-.686	-.148	.075	-.223	-.099	.214	-.253	-.100	.228	-.130	-.123	.140
INC	-.001	-.151	.056	-.000	-.148	.051	-.000	-.147	.064	-.000	-.104	.192
URB	-8.332	-.453	.000	-4.348	-.484	.000	-3.603	-.360	.000	-1.533	-.369	.000
MAN	-.000	-.021	.747	-.000	-.011	.863	-.000	-.001	.888	-.000	-.023	.175
TRI	.000	.039	.542	.000	.022	.717	.000	.008	.897	.000	.030	.648
INS	.000	.034	.531	-.000	-.008	.885	.000	.067	.226	.000	.026	.636
R Square	.323			.380			.320			.301		
Adj. R Square	.301			.360			.298			.287		
Prob. > F	.000			.000			.000			.000		

[a]Variables for ordinary least squares (OLS) regression analysis: BLK = average Black percentage of population, 1970-90 ; HSP = average Hispanic percentage of population; FBN = average percentage of population that was foreign-born population; INC = average median family income; URB = urban-rural status of a county in 1990; MAN = average employment in manufacturing; TRI = accumulated number of pounds of TRI carcinogenic chemicals, 1988-1994; HER = average number of acres of applied herbicides, 1972-87; DIG = digestive cancer death rate; GEN = genital cancer death rate; LYM = lymphatic/hematopoietic cancer death rate; URI = urinary cancer death rate. All death rates were per 100,000 for the period 1986-1994. B = parameter estimate and b = standardized coefficient; N = 254.

Discussion

This ecological study of cancer mortality rates showed that metropolitan counties and counties with large percentages of Blacks had the highest rates of digestive, genital, lymphatic and urinary cancer mortality in Texas. Moreover, median family income was inversely related to cancer mortality rates in all the models, except that of urinary cancer. These findings confirmed the research hypotheses and supported results of other studies. Contrary to expectations, Hispanic proportion of county population, level of manufacturing employment, accumulated pounds of toxic chemical wastes, and number of insecticide treated acres were unimportant influences on cancer death rates. Foreign-born proportion of county population was associated with only digestive cancer mortality. The lack of influence by these variables especially differed from the results obtained by Stokes and Brace (1988), who employed the same site-specific cancer groups, but focused on males in rural US counties. Additional unexpected findings were the high intercorrelations of the four cancer mortality rates. Comparisons of plotted rates in figures and of the rankings among counties with high rates clearly demonstrated that certain counties consistently had high rates for two or more site-specific cancers.

Interpretation of these results is cautiously made because of measurement constraints and the limitations of ecological studies. A twenty-year latency period was assumed for each of the site-specific cancers. Latency periods and etiologies vary for different cancers; for example, leukemia has a latency period of less than ten years. Also, genetic and physical susceptibilities of individuals, toxic properties of chemicals, and vectors and exposure dosages vary greatly, affecting the onset of morbidity and mortality. Such variation introduces many confounding factors that can influence cancer-related illness and death and that are often poorly or not measured in research.

Another constraint was the use of broad categories of toxic chemicals. Only two classes of agrochemicals (i.e., herbicides and pesticides) were examined here with no attention to specific active chemicals in these products, to other agrochemicals (e.g., fungicides, rodenticides, and nematocides), and to non-agricultural applications of these products. Direct measures of these chemicals and their pounds of applications were not available at the county-level. Similarly, no distinctions were made regarding types of employment in manufacturing industries. The measurement of manufacturing employment needs to be refined to identify specific high-risk jobs by industry and to determine who holds these jobs and their job history. Further, no distinctions were made among particular toxic chemicals in the TRI, their methods of release (i.e., into the air, water, etc.) as potential exposure vectors, volumes of toxic waste transferred away from manufacturing facilities and import locations, and the interactive combinations of carcinogenic chemicals in the environment. In short, the presence of toxic chemicals in the workplace and environment was understated and crudely measured for Texas counties. Consequently, findings of no influence by the number of insecticide-treated acres, level of manufacturing employment, and pounds of TRI carcinogens on cancer mortality rates should not be used to minimize the importance of these factors to cancer mortality. Rather, better-refined measures should be developed at the county and other levels of analysis to more accurately and reliably convey the presence of chemical hazards and their impact on human health and safety.

Finally, because of its ecological design, this study could not measure causal connections among people who were exposed to toxic chemicals in the environment and workplace and those people who died of cancer. Although some findings agreed with those reported elsewhere, many did not. Statistically significant and insignificant relationships identified at the county-level could drastically change when measured at the individual level and in the presence of other measured factors. Therefore, consideration of policy implications of the study's findings would be premature and tenuous at best. Attention should be given in future individual-level research to Texas counties where multiple high cancer mortality rates exist and where large concentrations of Black population live in urban areas. Though less than definitive, the relationships demonstrated by statistically significant variables in the OLS models suggest contentious, inequitable cancer risks may exist for residents of these counties. That research should address also the sociological and psychological factors related to human susceptibility to cancer. (Thomas, Kodamanchaly and Harveson 1998).

Endnotes

1. This research contributes to the project "Industrial Organization and Environmental Quality: Toxic Chemicals and Human Exposure Risks in Texas Counties (H-8571)," funded by the US Department of Agriculture and the Texas Agricultural Experiment Station. We thank Don Renchie, Extension Specialist with the Texas Agricultural Extension Service for providing pesticide-use information and Jeff Jordan with the State Data Center for his programming assistance. Correspondence should be addressed to John K. Thomas, Department of Rural Sociology, Texas A&M University, College Station, TX 77843-2125. E-mail address: **jthomas@rsocsun.tamu.edu.**

2. Only a few hundred of commercial chemicals are actively regulated for unreasonable risks (Wagener 1994), are deemed to be hazardous by the Resource Conservation and Recovery Act (RCRA), or are

monitored by the Environmental Protection Agency's (EPA) Toxic Release Inventory (TRI). Gerrard (1994) estimated that the total annual US waste production amounted to 10.8 *billion* tons, of which 79.6 percent are oil and natural gas production wastes.

3. The limitations of ecological studies are well noted by March and Caplan (1987) and Morgenstern (1995). Some of the limitations are lack of control of confounding factors and their effects on the observed exposure-outcome relationship, migration changes in the population at risk prior to and during the study period, and the potential of multicollinearity and higher correlations among predictor variables than would occur at the individual level. While these problems can not be ignored, their effects can be marshalled by: (1) use of as many risk factors as possible in an ecological regression model, (2) use of data grouped into the smallest geographic units of analysis as possible, subject to the constraints of intergroups migration and unstable rate estimation, and (3) determination of how groups were formed and use of all factors thought to influence the grouping process.
4. Major nonagricultural uses of pesticides include, for example, structural pest control in homes and other buildings; turf and lawn maintenance; rights-of-way maintenance on highways, power transmission lines, railroads; water treatment; and public health efforts to control rodents and mosquitoes. They represented collectively 26 percent (or 301 million pounds) of the U.S. total pesticide consumption in 1979 and 21 percent (or 202 million pounds) in 1995 (Aspelin 1995). Nonagricultural pesticide use and exposure at the county level was not measured in this study.
5. Manufacturing facilities in the SIC codes of 20 to 39 are: food products; tobacco; textile mill products; apparel; lumber and wood products; furniture and fixtures; paper and paper products; printing and publishing; chemicals; petroleum refining; rubber and plastics; leather; stone, clay, glass, and concrete products; primary metals; fabricated metal products; industrial and commercial machinery and computer equipment; electronic and electrical equipment; transportation equipment; measuring, analyzing, and controlling instruments; photographic, medical, and optical equipment; and miscellaneous manufacturing industries. Facilities in these industries must also have the equivalent of ten or more full-time employees and meet the established thresholds for manufacturing, processing, or other use of chemicals listed by EPCRA. Thresholds for manufacturing and processing are currently 25,000 pounds for each listed chemical; the threshold for other uses is 10,000 pounds (US Environmental Protection Agency 1996).
6. TRI listed chemicals are reported also if they are "transferred" to other locations, which are geographically or physically separate from the manufacturing source of the wastes. Transfer may be for recycling, energy recovery, treatment, or disposal. The volume of transferred chemicals were not included in this study. Moreover, the release vector (i.e., air emission, release to water, land storage, and underground injection to wells) was not examined. The first two types of releases would have the greatest potential implications for human exposure.
7. Proportion of population equal to and less than the 1990 poverty threshold (e.g., $13,359 for a family of four) was omitted from the analysis because of high multicollinearity with per capita income. Multicollinearity was determined by the magnitude of each variable's variance inflation value (Myers 1990).
8. Albrecht and Murdock (1990) point out that the definition of a "farm" changed nine times between 1850 and 1982. The 1982 definition describes a farm as any place from which $1,000 or more of agricultural products are sold, or normally would have been sold, during the census year. This variation of definitions complicates the comparison of data from the censuses.
9. The NCHS stopped reporting mortality data for counties with less than 100,000 people after 1989. It may provide these data, however, in response to special requests at a cost for each year of data requested.
10. Results of OLS models with the variable "average number of herbicide treated acres" were very similar to the results obtained with the insecticide measure.

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