

Economic and Toxic Chemical Influences on Rates of Gynecological Cancer Mortality in Texas

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Abstract

The influences of economic factors, agricultural pesticides, and industrial carcinogenic wastes on rates of cervical and ovarian cancer mortality were examined for 254 Texas counties. Regressor variables included: median family income, county proportion of state female employment in agriculture, county proportion of state female employment in the chemical/petrochemical industries, percentage of pesticide-treated acres in county land area, and accumulated pounds per acre of known carcinogenic wastes released by manufacturing industries in a county. Data for most of the variables were averaged for the period 1980 to 1990 to stabilize values for rural, sparsely populated counties. Levels of carcinogenic wastes reported by the Toxics Release Inventory were summed for the years 1988 to 1994. Standardized age and race-adjusted mortality rates were based on the average number deaths due to each of the two gynecological cancers for the period 1986 to 1994 and the 1990 size of population subgroups in Texas. Bivariate correlations were computed and ordinary least squares regression (OLS) was conducted. The OLS model explained 83 and 77 percent of the variation in cervical cancer and ovarian cancer mortality rates. Regression findings indicated that cervical and ovarian cancer mortality rates were positively influenced by both of the employment measures and by median family income. Contrary to the research hypotheses, these rates were negatively influenced by pesticide coverage and the per acre volume of accumulated toxic wastes.

Keywords: *cervical cancer, ovarian cancer, mortality rates, Toxic Release Inventory, Texas*

Introduction

Carcinogen and other health effects due to exposure to toxic chemicals in the workplace and environment are important societal issues garnering much attention from many segments of the socio-political spectrum (Szasz 1994). Concerned lay and professional groups claim that little is known and understood about the effects of most chemicals, especially those in the national waste stream, on human health and safety (Thomas et al. 1999). For example, of the 658 million to 786 million pounds of pesticides applied annually since 1980 in the United States (US Environmental Protection Agency 1997), only 70 among 2,800 pesticide products have been tested and are known to cause cancer in people (Bullard and Wright 1993). In their battle cry for environmental justice, these groups contend that people of color, children, the elderly, and women are more vulnerable to exposure and particular health risks than others in our society (Bullard and Wright 1993; Goldman 1991).

Epidemiologists and social scientists have devoted considerable attention to unraveling the complex relationship between toxic chemicals and cancers among women (Hartge and Stewart 1994; Shen et al. 1998; Vasama-Neuvonen et al. 1999). Their evidence shows that incidence and mortality trends of gynecological cancers, for example, have varied over time and by race, age, and geographical region (Devesa et al. 1989; Oriel et al. 1999; Wingo et al. 1999). Scientists have investigated only recently the occupational and environmental factors that may cause these female cancers in the United States (Wolff et al. 1996). Much of this work is hampered by the lack of longitudinal datasets on women's employment and health histories and an incomplete understanding of which and how environmental factors affect female health (Wolff and Weston 1997).

Although the incidence of gynecological cancers lags far behind the incidence of breast cancer and has shown a downward trend since the 1970s, they are among the leading cancers among U.S. women (Devesa et al. 1989, Wingo et al. 1999). For all races, cancer of the corpus uteri (endometrial cancer) had an age-adjusted incidence rate of 21.1 per hundred thousand (expressed as 10^5) population, which ranked it 4th in the top ten disease sites among women during the period 1990 to 1996. The incidence of ovarian cancer was 14.8 per 10^5 population with a rank of 5th, followed by cancer of the cervix uteri with an incidence of 9.0 per 10^5 population and a rank of 8th (Wingo et al. 1999). Age-adjusted mortality rates due to these cancers were over the same period: ovarian (7.69, ranked 4th), cervix uteri (2.47, ranked 10th), and corpus uteri (3.37, ranked 8th). Ovarian cancer had the greatest mortality-to-incidence ratio (52%). Cancers of the cervix uteri (19%) and the corpus uteri (16%) had lower ratios, largely possibly because of successful widespread cytologic testing programs (Devesa et al. 1989).

An ecological design was used in this study to determine if mortality rates of cervical and ovarian cancers were influenced by two groups of factors at the county-level of analysis in Texas.² One group included economic factors of median family income, county percentages of all women employed in agriculture and in particular high-risk manufacturing jobs. The other group involved environmental factors such as the proportion of pesticide-treated acres to county land acre, and the per acre volume of chemical carcinogens released by manufacturers in each county.

Texas and Gynecological Cancers

Texas was selected as the study area for several reasons. Its average annual age-adjusted mortality rate for cervical cancer (3.3 per 10^5 population) was significantly greater than the national rate (2.8 per 10^5 population) for the period 1991 to 1995 (Ries et al. 1998). Among ethnic groups, cervical cancer was the third most diagnosed cancer for Hispanic women and the fifth most diagnosed cancer for African American and other ethnic groups in 1995 (Carozza et al. 1999). Although Texas' rate for ovarian cancer (7.0 per 10^5 population) was lower than the national rate (7.7 per 10^5 population), it more than doubled the mortality rates for the combined other gynecological cancers in the state. Moreover, ovarian and cervical cancers were among the top five cancer sites diagnosed for all age groups of women in Texas for 1995; endometrial and cervical cancers were among the top sites diagnosed for race/ethnic groups (Carroza et al. 1999).

Texas was chosen also because it is a leading agricultural state in which 18 of 46 regulated pesticides were detected by the Environmental Protection Agency in groundwater levels that exceeded health advisory levels for 1988 (Kellogg et

al. 1992). The state ranks fifth nationally in the number of acres (6.4 million) which has a high risk of ground water contamination by pesticides (Kellogg et al. 1992). Exposure to particular classes of pesticides over a long period might pose high cancer risks because of their potential to mimic female hormones (Wolff and Weston 1997).

Other reasons for focusing on Texas were that it has ranked annually either first or second since 1988 in the total volume of toxic chemicals released into the environment by approximately 1,200 manufacturers who reported to the Toxic Release Inventory (TRI) (US Environmental Protection Agency 1993, 1996). Further, Texas counties have averaged 1,304 women employed in manufacturing jobs since 1980. Some of the most high-risk jobs are in the chemical and petrochemical industries, which account for the largest volumes of carcinogen releases in Texas. Most of these two industries' facilities are located predominately in metropolitan areas and in counties along the Gulf Coast (Thomas and Harveson 1997). Although these manufacturers have historically employed large numbers of male workers, they have increasingly employed more women in professional and wage-related positions as more women have entered the workforce.

Finally, this study was designed to minimize problems associated with ecological studies of cancer. The focus on counties in a single state avoided regional variations observed by other researchers in analyses of gynecological cancer incidence and mortality (Devesa et al. 1989; Ries et al. 1998; Wingo et al. 1999). Next, calculation of standardized age- and race-adjusted mortality rates controlled variations in the size of population sub-groups among counties. Furthermore, age and race are associated typically with cancer risk as confounders by being related to both exposure and disease outcome (Tsai and Wen 1986). Standardization of rates controls for these confounders and adjusts for unequal sizes of subgroup populations among counties. Finally, cervical and ovarian cancers differ in etiology, morphology, diagnostic approaches, and prognoses (Hulka 1997). These differences necessitated the independent study of the mortality rates of the two cancers. Endometrial cancer was not included in the study because of its lower mortality-to-incidence ratio.

Gynecological Cancers and Risks

Epidemiologists have identified several risks associated with gynecological cancers. Cervical cancer includes a group of diseases that arise from the surface of the cervix. Higher risks are associated with infections by the human papilloma virus or the Herpes Simplex Type 2 virus, multiple sex partners, early age at first intercourse, cigarette smoking, and use of oral contraceptives, particularly high-dose estrogen pills (Devesa et al. 1989; Koutsky et al 1992). When

compared to women afflicted with cervical cancer, ovarian cancer patients have a lower probability of survival after metastases to distant organs in the body. Ovarian cancer typically arises in the layer of epithelial cells that surround an ovary, but can also occur in any of several types of ovarian cells. Women who are 55 to 80 years of age and who have familial history of breast or ovarian cancer are the most at risk groups. Other risk factors include nulliparity (Shen et al. 1998), and high fat, protein, and caloric consumption (Kushi et al. 1999). Findings on the use of talcum powder on the perineal area (Hartge and Stewart 1994; Vasama-Neuvonen et al. 1999) and hormonal replacement therapy (Shen et al. 1998; Schairer et al. 1999) remain inconsistent.

Risks related to exposure to toxic chemicals in the workplace and environment are of increasing interest to scientists and the general public (Wagener et al. 1995). Many of these chemicals and their metabolites behave in vivo and in vitro much like estrogen or other endocrine hormones produced in women (Hulka 1997). Some of these agents bind to estrogen receptors and promote prolific cell growth, increasing the risk of tumorigenesis and carcinoma. Long term exposure to other chemical agents such as the pesticide TCDD (2,3,7,8, -tetrachlorodibenzo-p-dioxin) and some dioxin-like PCBs (polychlorinated biphenyls) act anti-estrogenically thereby causing premature anovulation (Wolff and Weston 1997).

A Human Ecological Framework

Human ecology theory provided the framework for this study. According to a contemporary version of the theory, the "human ecological complex," or model, is composed of population (P), social organization (O), environment (E), and technology (T) components (Duncan and Schnore 1959). The POET model embraces the idea that human ecology and biological ecology are closely related, as shown in recent applications of the model involving toxic waste issues (Thomas et al. 1999).

In the POET model, *population* is an aggregate of people who reside in a county and collectively adapt to environmental conditions. Economic opportunities accrue to a place (county) by virtue of both its size of population and its access to larger and more diverse economies of metropolitan areas. These benefits dissipate as the distance from large economic centers increases. Counties that are contiguous to a large metropolitan area are more likely to benefit economically from metropolitan growth than rural, isolated counties.

The population component also includes demographic processes and outcomes such as mortality that accompany collective human adaptation. Mortality rates of breast cancer victims are an aggregated, long-term response by residents to environmental and workplace conditions in a county (Thomas et al. 1999). Differences in distributions of age and

race in subpopulations can distort rates. Therefore, mortality rates were standardized to control for these differences.

Social organization is the manner in which human populations evolve a system of differentiated, sustainable relations and activities that utilize the environment in terms of providing resource materials, living space, and a repository for wastes (Freese 1988). Human ecologists often regard this component as the industrial and occupational structure of an area (Frisbie and Poston 1975). Median family income and the county percentages (i.e., share) of all women employed in the agriculture and the chemical/petrochemical industries in Texas partially indicated in this study the economic or sustenance structure that supported this group of employed women.

The natural *environment* is a composite of external factors (e.g., climate, soil, water and topography) to which a population is actually or potentially responsive (Hawley 1986). *Technology* includes all techniques, practices, and tools utilized by a population in its social organization as it adapts to the environment (Duncan and Schnore 1959). Greatly diversified economies tend to produce by their nature more environmental pollution and risks of human exposure to toxic hazards (Freese 1988). Although the production of synthetic and natural chemicals has expanded the productive capacity of the environment, volumes of industrially released carcinogens and pesticide use are dangerously toxic to humans. Hawley (1986, 49) termed these conditions of ecosystem disruption and pollution "disturbances" between social and biophysical systems. In the present study, volume of toxic industrial chemical wastes and the number of farm acres treated with pesticides indicated environmental quality (i.e., the greater the volume of the waste stream and the number of pesticide-treated acres, the poorer the environmental quality). It also represented the level of technological development (i.e., the greater the volume of the waste stream, the more economic development or industrialization in a county; conversely, the greater the number of pesticide-treated acres, the less urbanized and economically diversified a county).

Empirical Evidence and Hypotheses

Based upon empirical evidence reported in past research, this study tested five research hypotheses for rates of cervical and ovarian cancer mortality in Texas. Because of the ecological nature of the research design, the terms "affected" and "influenced" were used in lieu of "predicted" in each research hypothesis to avoid implying a causal effect (Morgenstern 1995).

H1a and 1b: Median family income is related (a) negatively to the rate of cervical cancer mortality and (b) positively to the rate of ovarian cancer mortality among women. Median family income is often a proxy for socio-economic

status (SES). Research evidence shows that prior to the 1990s, socio-economic status was linked to gynecological cancer risks. Women who had higher SES were more likely to experience ovarian cancer and possibly endometrial cancer, unlike women with lower SES who were more prone to cervical cancer (Faggiano et al. 1997). Najem and Greer (1985) reported evidence of a positive correlation between ovarian cancer mortality and per capita income and a negative relationship between cervical cancer mortality and per capita income in their study of New Jersey residents. Possible factors that contributed to these rate differences among SES groups were life-style differences such as accessibility to preventative care/treatment (Pukkala and Weiderpass 1999) and diet related behavior (Kushi et al. 1999).

H2a and 2b: Employment in the agricultural industry is related (a) positively to the rate of cervical cancer mortality and (b) negatively to the rate of ovarian cancer mortality among women. Employment in agriculture can occur on the farm as an owner/operator, family worker, or hired worker, or it can occur in companies which process agricultural commodities for consumers. Some research findings have indicated excess cervical cancers among farm workers (Blair et al. 1993; Stubbs et al. 1984), as well as among employees in the agricultural products, processed foods, and farm machinery industries (Sala et al. 1998). While women employed in the farm products (i.e., raw materials, wholesale) and farm supplies industries have higher mortality risks for endometrial cancer, female farm workers have lower risks of ovarian cancer mortality (Sala et al. 1998; Shen et al. 1998).

H3a and 3b: Employment in the chemical and petrochemical industries is positively related to (a) the rate of cervical cancer mortality and (b) to the rate of ovarian cancer mortality among women. Occupational exposure to carcinogens and the effects of exposure are difficult to assess because of the unavailability of these data, particularly for working women who have cervical cancer (Wolff et al. 1996). Moreover, in their comprehensive review of articles published from 1970 to 1997, Shen and colleagues (1998) found considerable variation internationally in the incidence rates for ovarian cancer and concluded that the evidence for ovarian cancer risks associated with job titles, industries, and environmental chemical agents was not convincing. Goldman (1991) provided some marginal evidence when he reported an increased risk of breast cancer mortality due to petrochemical employment in the United States. Breast cancer and ovarian cancer share several risk factors.

H4a and 4b: Agricultural pesticide use is positively related to (a) the rate of cervical cancer mortality and (b) to the rate of ovarian cancer mortality among women. The Occupational Safety and Health Administration (OSHA) recently identified pesticides such as dichlorovos and hep-

tachlor as carcinogenic (Beim et al. 1998). The EPA decertified arsenic compounds and chlorodane for agricultural use in the early 1990s because of their carcinogenicity. Other research evidence indicates triazines (atrazine being the most common) increase the occurrence of uterine adenocarcinoma and lymphatic and hematopoietic tissue cancer in female laboratory mice. No clinical or epidemiological evidence exists, however, that affirms a clear association between these pesticides and gynecological cancers among women (Sathiakumar and Delzell 1997). According to Wolff and her colleagues (1996), most chemical pesticides, except for chlorinated hydrocarbons, that are used today do not persist in the environment. This short-lived characteristic of pesticides makes it difficult to quantify body burden after several months or years following human exposure.

H5a and 5b: Accumulated volume of industrial waste carcinogens in the environment is positively related to (a) the rate of cervical cancer mortality and (b) to the rate of ovarian cancer mortality among women. Chemical wastes reported to the TRI are by their very definition hazardous to human health (US Environmental Protection Agency 1996). Of the 299 toxic chemicals listed in the TRI prior to 1994, 121 chemicals were known carcinogens, according to criteria set forth in the OSHA's Hazard Communication Standards (US Environmental Protection Agency 1996). Fifty of these chemical carcinogens were present in industry releases in Texas during the period 1988 to 1994. Chemical carcinogens released in the largest volumes (> 1 million pounds) were: styrene, benzene, acetaldehyde, acrylonitrile, 1,3-butadiene, formaldehyde, lead, tetrachloroethylene, and chloroform (Thomas and Harveson 1997). In addition to causing cancers, many of these chemicals produce genetic and chromosomal mutations, reproductive and developmental toxicities, and neurological risks (Geschwind et al. 1992; Stockwell et al. 1993). Najem and Greer (1985) found a consistent and significant positive association between breast, cervical, and ovarian cancers and the number of disposal sites for toxic chemical waste in New Jersey.

Methods

Regressor Variables

Data were compiled for this study from several sources and were averaged over approximately a ten-year period to produce stable estimates of frequency for rural, sparsely populated counties. Among the economic variables, *median family income* was obtained from the Bureau of the Census (US Department of Commerce 1983a, 1993a) and was averaged for the 1980 and 1990 census periods (mean = \$21,200). Employment in agriculture (mean = 96 women) and in the chemical and petrochemical industries (mean = 1,304

women) were based on women 16 years of age and older. Data used to calculate the average number of women employed full time in each of these industries were obtained from the 1980 and 1990 Summary Tape File 4 provided by the US Department of Commerce (1983b, 1993b). The chemical and petrochemical industries (hereafter shortened to "chemical industry") had the same standard industrial codes (28 and 29) of industries required to report to the TRI. *County percentages of female employment to total female employment in each industrial sector* were calculated to show the relative distributions across the state. In most cases, employment was less than one percent and skewed toward a few predominant counties. Two counties accounted for 15.2 percent of the total female agricultural employment in Texas. More dramatically, three counties provided 58.8 percent of the female chemical employment; approximately a third (36%) of all women employed in the Texas chemical industry worked in one of these counties.

Agricultural acreage data were obtained for three time periods (1978, 1982, and 1987) from the Census of Agriculture (US Department of Commerce 1980, 1984, and 1992). *Pesticide use/coverage* was measured initially as the average number of acres (mean = 34,244 acres) treated with insecticides and herbicides during the three agricultural census reporting periods. The proportion of treated to total number of land acres was determined next for each county (mean = .056 or 5.6 percent). Fourteen counties had more than 20 percent of land acre treated in pesticides. Overall, 84 percent of the counties had less than one in every 10 land acres treated in pesticides. The measurement of pesticide usage had two limitations. Agricultural chemicals are often repeatedly applied to the same acres thus creating a lack of independence in their measured acreages. Another limitation was that pesticide use did not distinguish specific chemicals (i.e., chlorinated hydrocarbons such as atrazine and methoxychlor) by toxicity and longevity in the environment. Data for the applied pounds of particular agricultural pesticides, though preferred, were unavailable at the county level.

Finally, volume of carcinogenic chemical releases was measured in dry pounds (mean = 943,678 pounds for all counties). A list of 121 known carcinogens, that represented less than one-half of the list of other toxic chemicals monitored by the EPA prior to 1995, was standardized to maintain comparability for the TRI reporting years of 1988 to 1994, and to measure the potential accumulation of these wastes.³ Only 38 percent of Texas' 254 counties had reported carcinogenic wastes (mean = 2,496,815 pounds for 96 counties). *Pounds of carcinogenic wastes per acre* was calculated for each county (mean = 1.3 pounds). Seven counties had greater than 30 pounds of carcinogenic waste releases per acre. None of these counties coincided with the counties that

reported large proportions of pesticide-treated acres. As was the case with agricultural pesticides, limitations of this measure of chemical carcinogens were that toxicities of waste chemicals are not all equal, these chemicals do not cause the same cancers and illnesses in humans, and their environmental longevities vary (Stockwell et al. 1993).

Response Variable

Mortality rates for cervical and ovarian cancers were computed for each county and based on decedents' counties of residence. The National Center of Health Statistics (NCHS) provided mortality data for the years 1986 to 1994. The ninth edition of the International Classification of Diseases (ICD) reported classification codes for cervical (ICD 180) and ovarian (ICD 183) cancers (US Department of Health and Human Services 1988).

Mortality rates were standardized first by totaling and then averaging the numbers of deaths for the years of 1986 to 1994 for each county to stabilize fluctuations that might have occurred annually in the number of deaths (Morgenstern 1995; Shryock and Siegel 1976). Second, the expected number of age- and race-adjusted deaths due to reproductive cancer were calculated (Shryock and Siegel 1976). Sizes of county subgroup populations reported in the 1990 US Census were used to adjust the rates for women 15 years of age and older. Age categories were 15-24, 25-34, 35-44, 45-54, 55-64, and 65 years of age and older. Racial/ethnic categories were Anglo, Black, Hispanic, and Other (i.e., Asians, American Indians, and other groups) (Murdock and Hoque 1992). Finally, use of the direct method and the sizes of age-race subgroup populations standardized adjusted death rates per 10⁵ population in Texas (Shryock and Siegel 1976).

Epidemiologists and demographers are accustomed to substituting a non-zero value when a standardized mortality rate (SMR) is calculated to be zero. They assume that the incidence of death due to a site-specific cancer could be unmeasurable in the presence of cancer illness among a given population at a given point in time. Twenty-eight counties had a standardized cervical cancer mortality rate equal to zero for their total female populations in this study; in other words, no incidence of cervical cancer mortality occurred over a nine-year period. Twenty-one counties had a standardized ovarian cancer mortality equal to zero. These SMRs were converted to a value of .0001. Figure 1 is a histogram of the distribution of cervical and ovarian mortality rates. Figures 2 and 3 show respectively the SMRs for cervical and ovarian cancers among Texas counties. The greatest rates occurred in counties shown in black as having rates larger than one standard deviation above the mean rates. Counties with mortality rates equal to or greater than one standard deviation from the mean rates are of particular interest

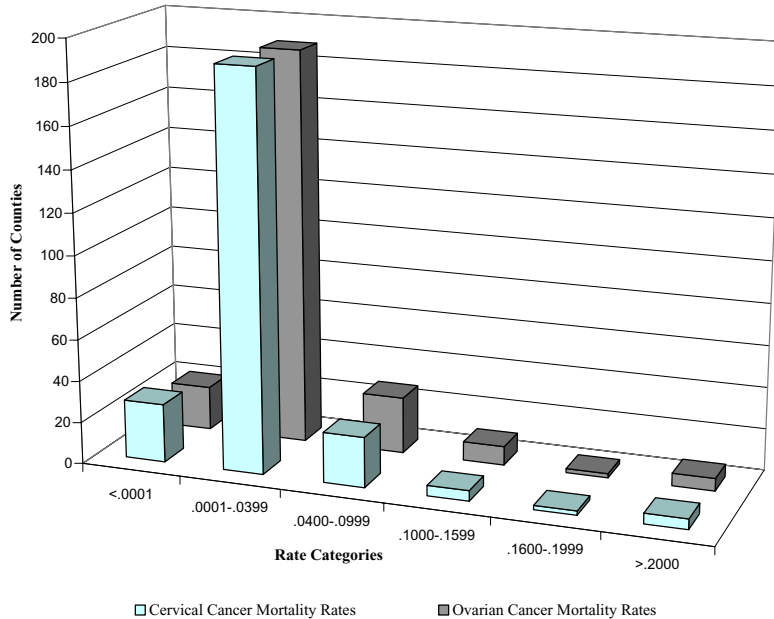


Figure 1. Histogram of Rates of Female Cervical and Ovarian Cancer Mortality in Texas, 1986-1994

Analytical procedures

Bivariate correlation coefficients were calculated and their statistical significance were determined for the hypothesis $\rho = 0$. Ordinary least squares (OLS) regression was conducted next for rates of cervical and ovarian cancer mortality (SAS Institute, Inc. 1990). Computed values for the variance inflation factor (VIF) and tolerance (TOL) determined the occurrence of multicollinearity among the regressor variables (Hamilton 1992). The SPEC option in SAS (1990) assessed whether the residual errors in the OLS models were homoskedastic and independent of the regressor variables, and if the analytical model was correctly specified (White 1980). Heteroskedasticity produces biased and inefficient standard error estimates and undermines the rationale for t- and F-tests (Dietz et al. 1987; Hamilton 1992). OLS procedures assume homoskedastic error terms (see Hamilton 1992 for a discussion of other assumptions of OLS). The SPEC chi square value for the cervical cancer model had a borderline probability value ($p = .0532$).⁴ The chi square value was statistically insignificant ($p = .2999$) in the ovarian cancer model. Both error variances were considered homoskedastic.

Finally, Cook's D values and plots of the residual errors against estimated mortality rates determined case influence

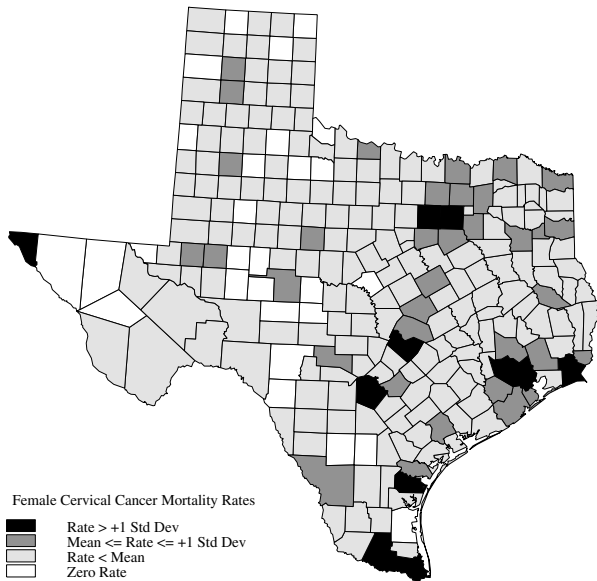


Figure 2. Age- and Race-adjusted Female Cervical Cancer Mortality Rates for Texas, 1986-1994

because they would have the greatest leverage in the regression analysis. Seven of ten counties highlighted in black were the same in both maps. These counties have large industrialized, metropolitan areas and high concentrations of chemical industry employment.

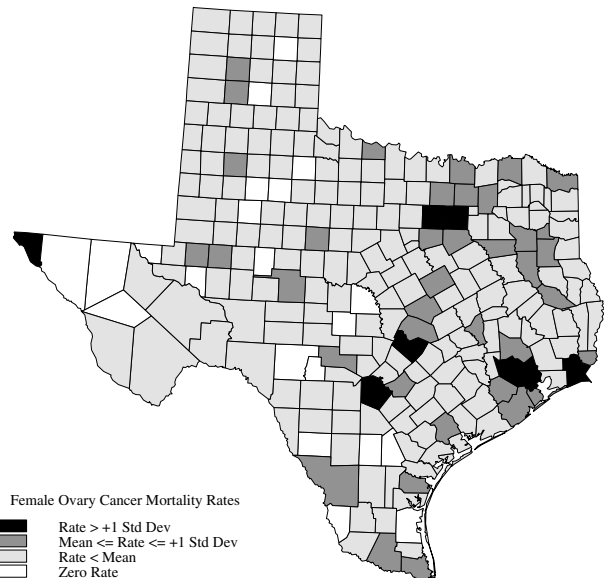


Figure 3. Age- and Race-adjusted Female Ovary Cancer Mortality Rates for Texas, 1986-1994

or leverage conditions (Neter et al. 1985). Cook's D measures the influence of the *i*th case on all estimated regression coefficients, or equivalently all *n* predicted cancer mortality rates. Eight counties had values that were greater than one (the absolute cutoff or elimination point), or values greater than .0157 (i.e., 4/N, where N = 254) in both of the OLS models. The latter threshold value is the size-adjusted cutoff point for unusually influential cases (Hamilton 1992). These "outliers" remained in the analysis because they were counties with prominent levels of female employment in the chemical industry and industrial carcinogenic wastes and because no theoretical or methodological reason existed for their exclusion (Dietz et al. 1987).⁵

Several reasons exist for cautiously reporting our findings. Ecological analyses such as the one conducted ignore individual-level data, which include health histories of decedents and exposure vectors, doses, and durations that would affect when, where, and what cancers occur (Wagener et al. 1995). The analytical attention given to aggregate county-level measures warrants that causal inferences be avoided. Relationships among aggregate data used in ecological models can differ radically when observed at other levels of analysis (Morgenstern 1995). Nevertheless, such models and their results may encourage further investigation at the individual-level of analysis. Finally, the examination of only cancer mortality rates in the study ignored incidences of cancer morbidity, that is to say acute and chronic health-related responses to exposure.

Results

Table 1 shows the descriptive statistics and bivariate correlation coefficients of all the study variables. Except for the

negligible relationship with pesticide coverage, rates of cervical and ovarian cancer mortality had statistically significant associations with all other variables, including each other ($r = .993$). Positive relationships were strongest with chemical and agricultural industry employment, followed by the number of accumulated pounds per acre of carcinogenic releases, and median family income. Neither rate had a strong association with pesticide coverage.

Although not addressed in the research hypotheses, intercorrelations among the regressor variables were as expected. Median family income had a weak positive association with the female employment in each industry and per acre level of carcinogenic chemical waste. Agricultural and chemical industry employment of women was moderately related to each other and each of these variables was positively associated with the level of carcinogenic chemical waste. Only agricultural employment had a statistically significant positive association with pesticide use.

Table 2 presents results of the OLS regression analysis for the rates of cervical and ovarian cancer mortality. The model regressed cancer mortality rates against the five independent variables. It explained 83 percent (adjusted multiple correlation coefficient or RSQ) of the variation in cervical cancer mortality rates and 77 percent of the variation in ovarian cancer mortality rates. As hypothesized, all of the economic regressors were statistically significant influences on the rates of cervical cancer mortality. The finding that median family income had a positive effect on ovarian cancer mortality rates differed, however, from results reported elsewhere (Najeem and Greer 1985). Overall, female employment in the chemical and agricultural industries had respectively the strongest effects, which were in line with other empirical results.

Table 1. Simple statistics and bivariate correlations of socio-economic and toxic chemical factors with standardized cervical and ovarian cancer mortality rates for females in Texas counties (N=254).

Variables ^a	INCOM	AGEMP	CHEMP	PEST	CHEM	CERVIX	OVARY
INCOM	1.000						
AGEMP	.145†	1.000					
CHEMP	.276‡	.491‡	1.000				
PEST	.053	.448‡	-.020	1.000			
CHEM	.333‡	.427‡	.672‡	.001	1.000		
CERVIX	.280‡	.748‡	.869‡	-.004	.504‡	1.000	
OVARY	.292‡	.706‡	.835‡	-.015	.470‡	.993‡	1.000
Mean	\$21,200.00	.394	.394	.056	1.310	.032	.038
Stand. Deviation	\$4,400.84	.764	2.472	.064	5.348	.108	.127

^a INCOM = average median family income, 1980-1990; AGEMP = county percentage of the state total number of females employed in agriculture, 1980-1990; CHEMP = county percentage of the state total of females employed in chemical and petrochemical industries, 1980-1990; PEST = proportion of the number of pesticide-treated acres in the total land area of a county, 1978-1992; CHEM = number of the accumulated pounds of carcinogenic chemical waste per acre in a county, 1988-1994; CERVIX = age- and race-adjusted mortality rates for female cervical cancer; and OVARY = age- and race-adjusted mortality rates for female ovarian cancer. Death rates were per 100,000 population for the period 1986 to 1994. Coefficients are statistically significant for rho = 0; p < .05 (*), p < .01 (†), p < .001 (‡).

Results for the environmental variables differed from our hypotheses and supporting literature. The effect by pounds of wastes per acre was statistically significant in both models. However, pesticide coverage had a small influence that was statistically significant only in the model for rates of ovarian cancer mortality. The negative effects by these environmental variables in both models were counter intuitive and contrary to the claims of environmental justice proponents.

Collectively, these findings indicated that cervical and ovarian cancer mortality rates were higher in counties that had greater percentages (shares of the state total) of women employed in the agricultural and chemical/petrochemical industries and higher median family incomes, but fewer pounds of carcinogenic wastes per acres and in the case in the ovarian cancer mortality model, less pesticide usage.⁵

Discussion

The results of this study suggest that rates of cancer mortality were greater in more industrialized counties where

Table 2. Cervical and ovarian cancer mortality rates regressed against economic and toxic chemical factors for Texas counties (N=254), 1990.

Mortality Models and Regressor Variables ^{a, b}	BETA ^c	SE ^c	beta ^c	P > t	Adjusted		
					RSQ	RSQ	P > F
Cervical Cancer Model							
Intercept	-.029	.015	.000	.0044	.832	.828	.0001
INCOM	.000	.000	.075	.0077			
AGEMP	.046	.005	.328	.0001			
CHEMP	.031	.002	.720	.0001			
PEST	-.089	.046	-.053	.0525			
CHEM	-.002	.001	-.113	.0020			
<i>(TOL > .357; VIF < 2.804; SPEC p = .0531)</i>							
Ovarian Cancer Model							
Intercept	-.047	.020	.000	.0187	.774	.770	.0001
INCOM	.000	.000	.102	.0017			
AGEMP	.051	.007	.310	.0001			
CHEMP	.036	.003	.709	.0001			
PEST	-.123	.062	-.062	.0489			
CHEM	-.003	.001	-.144	.0008			
<i>(TOL > .357; VIF < 2.804; SPEC p = .2999)</i>							

^a INCOM = average median family income, 1980-1990; AGEMP = county percentage of the state total number of females employed in agriculture, 1980-1990; CHEMP = county percentage of the state total of females employed in chemical and petrochemical industries, 1980-1990; PEST = proportion of the number of pesticide-treated acres in the total land area of a county, 1978-1992; CHEM = number of the accumulated pounds of carcinogenic chemical waste per acre in a county, 1988-1994.

^b The dependent variables were standardized age- and race-adjusted mortality rates for female cervical and ovarian cancers for the period 1986 to 1994. Death rates were per 100,000 population.

^c Unstandardized (BETA) and standardized betas (beta) and standard errors (SE) were rounded.

female employment and family incomes were greater than in non-industrialized areas. The fact that income is a function of employment could account for the importance of median family income in the model. In other words, regarding gynecological cancer mortality rates, where women work and what they do occupationally were as important as how much they earned. The findings for carcinogenic wastes in the model and pesticide usage's negative effects were contrary to the research hypotheses and to the positive bivariate correlations between carcinogenic waste releases and mortality rates.

Internal dynamics of the regression model (i.e., other regressor variables controlling the effects of the waste variable on mortality) notwithstanding, these findings could be attributable to interesting labor market conditions in the state. For example, women might have lived in a county that had very few carcinogenic wastes per acre and perhaps little agricultural pesticide coverage. Nevertheless, they commuted to jobs in agricultural and chemical industries located in the next county, where the volume of carcinogenic releases was greater. The family incomes of these women would have been above average because of higher paying jobs. Their risks of exposure to hazardous chemicals on the job or during their commuting to work would also have been greater. Consequently, they might have developed cervical or ovarian cancer. As these women died due to their cancers, counties of residence would have experienced above average cancer mortality rates. Although this scenario begs for individual level data on the life-course health/illness experiences of these women, it has some plausibility in Figures 2 and 3. Counties that have above average cancer mortality rates (as shown in dark gray) border and outlie the heavily industrialized metropolitan counties located in the eastern half of the state. Other studies have reported commuting-to-work patterns as just described for these areas (Perkinson 1999; see also Bokemeir, et al. 1983) and that hazardous industrial wastes released in these counties may be more toxic than the releases in more industrialized areas (Ying 2001).

These findings marginally contributed to human ecological theory (i.e., POET model). Researchers have restricted previous applications of the POET model to only selected features of the population and social organization components. More importantly, they have generally ignored framing human health and mortality issues with the model. This study demonstrated a connection between the economic organization (O) of Texas counties and rates of cervical and ovarian cancer mortality (P) through women's possible chemical (T) exposure experiences in *workplace* environments (O or E). It provided some understanding of the interrelationships among employment, chemical exposure, and health circumstances of women. Because of the unexpected negative

behavior of the chemical waste and pesticide variables in the research model, refinement and more explication of the environmental/technological components of the POET model remains, particularly below the county level of analysis and in applications to other highly vulnerable groups such as the elderly and children.

Indeed, identification of complex linkages between exposure to environmental factors and gynecological cancer mortality among women is difficult. Several research issues worsen this situation. The extension of experimental findings based on animal subjects to human subjects has produced inconsistent, unreliable results (Wolff et al. 1996). Also, ecological designs differ greatly from individual levels of analysis, especially in their ability to show causality (Morgenstern 1995). Moreover, use of direct and indirect standardization procedures produce dubious mortality rates when disease incidences are few and sizes of population subgroups are small (Tsai and Chen 1986). These rates may also differ when they are calculated using different geographical units. For example, county-level mortality rates based on population subgroups in a state may differ from rates based on the subgroup sizes for the nation.

Future ecological studies could be improved in several ways. Researchers need to make better distinctions regarding female employment in agricultural and manufacturing industries and specific workplace risks associated with each job type. Also, they should devote more attention to particular racial/ethnic groups who are over-represented in certain jobs. For example, other research has shown that large proportions of Hispanic and foreign-born populations were positively correlated with agricultural pesticide use and high site-specific cancer mortality rates, especially among male farm workers (Hoar et al. 1986; Moses et al. 1993; Stokes and Brace 1988; Thomas et al. 1999). In-depth field studies of women, who are employed in these occupations and are distinguished by ethnic group membership and life-course age groups, would fill a critical gap in the literature on women's workplace risks. Such studies are, however, expensive to conduct and require data on health history, vector and duration of exposure, and other potentially cancer-causing factors.

Next, the measurement of toxic chemicals should be improved by including over-the-counter pesticides, which accounted nationally for 21 percent (or 202 million pounds) of the total pesticide consumption in 1995 (Aspelin 1997). These products contain known or probable carcinogens, whose regular use by consumers prolongs exposure and perhaps accumulation in consumers over a long time period. A related refinement would distinguish among the broad categories of pesticides (i.e., herbicides, insecticides, fungicides, rodenticides, and nematocides). Direct measures of the

active agents in these chemicals and their pounds of applications are not readily available at the county-level.

Finally, this study addressed only on-site carcinogenic releases by manufacturers. Toxic wastes that were transferred off-site by in-state and out-of-state manufacturers to Texas counties were not examined (Thomas et al. 1999). Counties that had no TRI wastes reported as on-site releases may have received wastes transferred from elsewhere for recycling, energy recovery, treatment, or disposal. Consequently, counties' volumes of toxic chemical wastes were likely understated.

The risks and outcomes of human exposure to toxic chemicals and their wastes are controversial in several ways. Although this study explained much of the variation in cervical and ovarian cancer mortality, its focus was on rates, not incidence, of mortality. Therefore, it was unable because of its ecologic design to investigate known and other possible causal factors at the individual level. Controversy is also at the center of the jobs versus environment debate. The employment benefits derived from economic development and diversification are counter-balanced by industrial impacts on the environment and human health and safety in both the workplace and community at large. Environmental and social justice advocates have narrowly argued about the presence of these wastes in local communities and neighborhoods giving too little attention to workplace risks. At the heart of these issues is the citizen/worker's right to know about such risks, right to participate in regulatory reforms that protect against unsafe toxic chemical conditions, and arguably their right to sue for remediation of these conditions, as well as for negative health consequences they might experience (Grant 1997).

Endnotes

1. Address correspondence to John K. Thomas, Department of Rural Sociology, Texas A&M University, College Station, Texas, 77843-2125. E-mail: jthomas@rsocsun.tamu.edu.
2. An ecological study has several advantages. It is less costly than other epidemiological designs given its use of secondary data that are readily available at the county level of analysis. Because it provides a logical first step to identify specific counties with high incidences of specific female cancer mortality, it does not require *a priori* knowledge of an area's morbidity and mortality patterns (Morgenstern 1995). Among its disadvantages 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 a potential of multicollinearity and higher correlations among predictor variables than would occur at the individual level (Morgenstern 1995). While these problems can not be ignored or eliminated, researchers can mitigate such effects to some degree 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 inter-group migration and unstable rate estimation, and (3) determination of how groups were formed and use of all factors thought to influence the grouping process (Morgenstern 1995).
3. The list of TRI chemicals varies from year to year. It had 343 chemicals listed in 1994. The EPA expanded the list to include about 600 chemicals in 1995. It has modified the list by adding chemicals because of public and interest groups' requests and because recent research found particular chemicals to be toxic. Other chemicals were removed from the list because they were produced in insignificant quantities (i.e., less than 500 pounds) or they were no longer considered to be dangerous to humans and the environment according to scientific studies. Some manufacturers have retroactively amended their Form R reports which produced other changes in the list (US Environmental Protection Agency 1996).
 4. Because of the marginal probability value, the cervical cancer model was rerun after all of the regressor variables, except median family income (which was normally distributed), were transformed using log and square root power procedures. The adjusted multiple correlation coefficient decreased to .304, but the SPEC chi square probability value improved to $p = .5874$. Employment and pesticide usage findings were consistent with those reported for the original cervical cancer model. However, median family income and number of pounds of carcinogenic wastes per acre were statistically insignificant.
 5. The models were rerun after prominent outliers were omitted to determine what effects, if any, the deletion of outliers would produce on cervical and ovarian cancer mortality rates. Seven of the eight outliers were the same counties in both models. These outliers were large metropolitan counties that coincided for the most part with the counties shown in black (highest mortality rates) in Figures 2 and 3. The adjusted multiple correlation coefficient decreased to 69.9 percent in the model for cervical cancer and to 64.1 percent in the model for ovarian cancer. Levels of female employment in the chemical and agricultural industries were again the most important influences on cancer mortality rates. Median family income had a statistically significant, small positive influence in both models. The negative influence by county percentage of pesticide-treated acres became statistically significant in the cervical cancer model but was statistically insignificant ($p = .0529$) in the ovarian cancer model. Finally, negative influences by number of pounds of chemical toxic wastes per acre became statistically insignificant in both models. More importantly, the two models failed the SPEC test, which indicated heteroskedastic error variances.

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