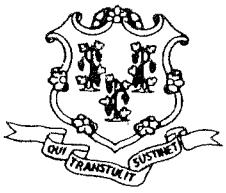
Cancer Incidence and Birthweight in Relation to Exposure to Raymark Waste Stratford, Connecticut



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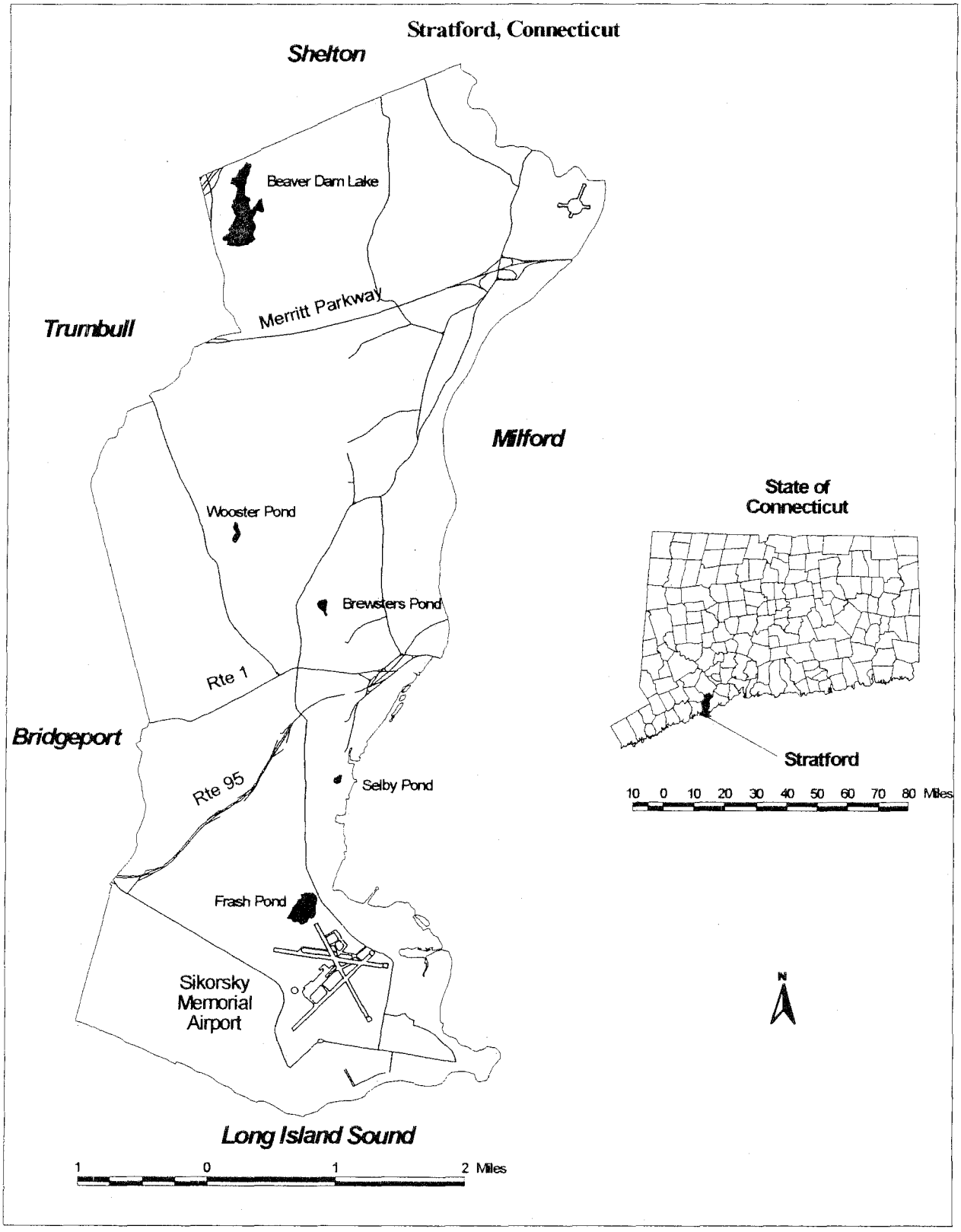
Stratford, Connecticut
Shelton
Beaver Dam Lake
Merritt Parkway
Trumbull
Milford
Wooster Pond
Brewsters Pond
Rte 1
Bridgeport
Rte 95
Selby Pond
Frash Pond
Sikorsky
Memorial
Airport
Long Island Sound
1 0 1 2 Miles
State of
Connecticut
Stratford
10 0 10 20 30 40 50 60 70 80 Miles
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ABSTRACT 1

I. STUDY OBJECTIVE 2

II. BACKGROUND 3

A. HISTORY 3
1.) Raymark 3
2.) Off-Site Locations 3
3.) Additional Site Investigations and Identification 5
4.) Environmental Sampling and Potential Health Impact Determination 5
5.) Environmental Remediation 6
B. PRELIMINARY EVALUATION OF HEALTH DATA 6
1). Blood Lead Screening Program 6
2.) Birth Defects 7
3.) Cancer Occurrence in Stratford from 1958 to 1991 7
C. RATIONALE FOR STUDY OF CANCER 8
1.) Mesothelioma 9
2.) Bladder cancer 9
3.) Testicular Cancer 10
4.) All Sites Less Than Age 25 - Early Onset Cancer (EOC) 11
D. RATIONALE FOR STUDY OF BIRTHWEIGHT 11
E.) EXPOSURE ASSESSMENT NEAR HAZARDOUS WASTE 14

III. METHODS 17

A.) STUDY DESIGN 17
B.) DATA SOURCES 17
1.) Environmental Data 17
2.) Cancer Incidence Data 17
3.) Birth Data 19
C.) USE OF GEOGRAPHIC INFORMATION SYSTEMS (GIS) 20
D.) EXPOSURE ASSESSMENT 21
1.) Block Group Exposure Model 21
2.) Distance to the Nearest Waste Site 22
3.) Cumulative Exposure 22
E.) METHODS USED TO ANALYZE CANCER INCIDENCE 25
1.) Time Trend Analysis 1958-1991: Comparative Morbidity Figures for Stratford vs Connecticut 25
2.) Spatial Analysis: Crude Incidence Rates per 1,000 by Block Group 1968-1991 27
3.) Spatial Analysis: Odds Ratios using Logistic Regression Models 1968-1991 27
F.) METHODS USED TO ANALYZE BIRTHWEIGHT 28
1.) Spatial Analysis: Using Regression Models 28

IV. RESULTS 30

A.) CANCER 30
1.) Time Trend Analysis 1958-1991: Comparative Morbidity Figures for Stratford vs. Connecticut 30
2.) Spatial Analysis: Crude Incidence Rates per 1,000 by Block Group 1968-1991 31
3.) Spatial Analysis: Odds Ratios using Logistic Regression Models 1968-1991 32
B.) BIRTHWEIGHT 34
1.) Block Group Exposure Model 34
2.) Distance to Nearest Waste Site Exposure Model 34
3.) Cumulative Exposure Model 35

V. DISCUSSION 36

A.) CANCER 36

1.) Time Trend Analysis 1958-1991: Comparative Morbidity Figures for Stratford vs. Connecticut *36*
2.) Spatial Analysis: 1968-1991 ..... *37*
B.) BIRTHWEIGHT 39
C.) EXPOSURE ASSESSMENT 40
1.) Block Group Model *41*
2.) Distance to Nearest Waste Site Model *41*
3.) Cumulative Exposure Model *42*
D.) USE OF EXISTING DATA SOURCES 42
E.) LATENCY BETWEEN EXPOSURE & DISEASE 42
F.) MOVEMENT OVER TIME 43
G.) USE OF 1990 CENSUS DATA AS CONTROLS 44
H.) OCCUPATIONAL EXPOSURE 44
I.) USE OF GIS 44

VI. CONCLUSIONS **46**

VII. RECOMMENDATIONS **47**

VIII. TABLES **48**

IX. MAPS **68**

X. AUTHORS **76**

XI. ACKNOWLEDGMENTS **77**

XII. REFERENCES **78**

ABSTRACT

Residential, recreational, commercial, and industrial properties in Stratford, Connecticut, were identified as posing health threats to the surrounding community. These sites were contaminated with lead, asbestos, and polychlorinated biphenyls owing to the waste disposal practices of Raymark Industries, a facility on the Environmental Protection Agency's National Priorities List. The hypothesis tested in this study is that residential proximity to these waste sites, as a surrogate of exposure, is associated with certain types of cancer and low birthweight.

In Stratford, waste was distributed at many locations. Based on residential proximity to these sites, three exposure models were developed: (1) block groups with waste, (2) distance from the nearest site, and (3) a cumulative exposure model. Each model refined the classification of exposure. The block groups with waste model being the least refined, and the cumulative exposure model being the most refined. If distance can effectively model exposure, ecological studies become an efficient way to address health concerns. Any existing database that contains residential addresses and measures of health can be investigated. In this report, the three exposure models are applied to data from birth certificates and two models are applied to data from a tumor registry.

Comparative morbidity figures were used to compare the incidence of the cancers in Stratford versus the State of Connecticut and to assess trends in cancer incidence for four categories of cancers: bladder cancer, mesothelioma, testicular cancer, cancers at any site in individuals under age 25 (early-onset cancer [EOC]). All cancers had been diagnosed between 1968 and 1991. While no trend could be found for testicular cancer, mesothelioma, or EOC, bladder cancer among males and males and females combined increased during the study period.

After controlling for age and gender and assigning geographic coordinates to case residential address at diagnosis, the association between exposure and the cancers was modeled using logistic regression. An innovative approach to estimate the control population was the use of census records. Crude incidence rates by block group were calculated to assist in the interpretation of the spatial analysis of cancer incidence.

No evidence of an association between exposure and mesothelioma was found. Evidence of an association between bladder cancer, testicular cancer, or EOC and exposures was limited and inconsistent.

After controlling for child's race, maternal age, and marital status, excluding multiple births, and assigning geographic coordinates to maternal addresses, 5,271 records (92.2% of 5,719 births) from 1983 to 1992 were used to test the association between exposure and birthweight. Missing values limited the ability to control for additional confounders (e.g., maternal education, smoking, weight, occupation, pregnancy interval, prior adverse outcomes, prenatal care, and medical risk factors). Birthweight was modeled as a continuous outcome using linear regression. Logistic regression was used to predict low birthweight (< 2,500 gm) and very low birthweight (< 1,500 gm). No evidence of an association between birthweight and exposure to the waste sites was found.

I. STUDY OBJECTIVE

The objective of this study was to test the hypothesis that residential proximity to the Raymark waste sites and health outcomes (birthweight and cancer) are associated. Routinely collected regulatory and surveillance data were used to address the public health concerns expressed by the Stratford residents. Computerized public health and environmental databases were integrated and analyzed, a technique previously employed in both nationwide[1] and statewide[2] analyses. Although ecological analyses of existing data have the advantage of being considerably less resource-intensive than cohort and case-control studies, the latter are subject to much less misclassification. In small populations using existing data bases, misclassification of exposure severely limits ecological analyses. For these studies to be effective, exposure misclassification has to be minimized and statistical power has to be sufficient.

To increase statistical power to test for associations in the small population of Stratford, continuous exposure variables were calculated based on the distance from waste sites to residences. Many prior studies have used postal codes or census designations to define exposure and establish a cutoff point of a given radius from a waste site to differentiate exposed and unexposed neighborhoods.[1] Since the geographic boundaries established by the U.S. Post Office or the U.S. Census are arbitrary with respect to chemical exposure, this methodology introduces substantial misclassification. Initially, the exposure models in this investigation were developed in an attempt to reduce this misclassification. The development of exposure models based on distance was complicated by the fact that waste materials were distributed at various sites throughout Stratford. Ultimately, the models developed in this study incorporated both the possibility of exposure to multiple waste sites and the different probabilities of exposure at the various site types where waste was found.

Exposure models based on geographic proximity can be applied to any existing database that contains residence locations and health outcome information. For this report, vital statistics and tumor registry databases provided the data for the two applications of the exposure models -- i.e., birthweight and cancer incidence. The birth certificate database contains information on all births (both cases and controls). In contrast, the tumor registry data do not contain information on the non-diseased control population. Therefore, for the cancer investigation, the U.S. Census of Population and Housing was used to estimate control populations. If the assumption underlying this study that distance from waste sites correlates with exposure proves correct, the modified ecological design developed for this study could be widely applicable. Any existing data source containing residential addresses and measures of health can be used to investigate a hypothesized association between exposure and disease.

II. BACKGROUND

A. HISTORY

On May 26, 1993 the Agency for Toxic Substances and Disease Registry (ATSDR) issued a Public Health Advisory to alert government agencies and the public of an imminent public health hazard in Stratford, Connecticut. This hazard was associated with past, present, and potential future exposures to waste from past operations and disposal practices of Raymark Industries, Inc.. The primary contaminants of health concern included asbestos, lead, and polychlorinated biphenyls (PCBs).

Following the release of the Public Health Advisory, the Environmental Protection Agency (EPA), the Agency for Toxic Substances and Disease Registry (ATSDR), the Connecticut Department of Environmental Protection (DEP), the Connecticut Department of Public Health (DPH) and the local health department initiated a number of activities to identify additional waste areas, reduce or eliminate exposure to known sites and address public health questions. These activities included extensive surface soil and depth sampling, review of environmental data, development of health consultations, voluntary blood lead screening, and preliminary health statistics reviews. A Public Health Assessment has been completed for Raymark and provides details on the history of the site, and pathways of exposure.[3]

1.) Raymark

Raymark Industries, Inc. operated a facility on 75 East Main Street in Stratford from 1919 until September 1989 when operations ceased. The Raymark Industries site covers approximately 33.4 acres. The facility produced brakes, clutch parts, and other friction based products. During the manufacturing process, wastes generated included ignitable and corrosive waste, solvents, liquid adhesives, phenolic resins, alcohol, caustic, phenolic mixtures, lead, asbestos, PCBs, and dioxins/furans. Table 1 lists contaminants identified on the Raymark property.

Raymark routinely disposed of its waste on the facility property and at other locations in Stratford. From 1919 to July 1984, Raymark used a system of lagoons in an attempt to capture the waste lead and asbestos. Over this 65 year period, these lagoon systems were located throughout the western and central portions of the Raymark site. As the lagoons filled up with sludge, they were often dredged and the material was used as fill in locations around Stratford. At several locations, the fill was evident in surface soil by the presence of brake parts and friable asbestos.

2.) Off-Site Locations

Based on historical town records, fourteen known or suspected waste disposal areas in the town were identified for evaluation. These areas included: Wooster Junior High School/playing fields; Raybestos Memorial Field; Morgan Francis Property on East Broadway; the Spada Property/commercial properties on Ferry Boulevard; Ferry Creek; the

Housatonic Boat Club; Elm Street Lot K; the Airport Clear Zone; Forth and Fifth Avenue; the landfill on Dorne Drive; Short Beach Park; Lordship Boulevard; Honeyspot Road; and Beacon Point Road. Of the fourteen waste disposal areas, eight were prioritized for evaluation. Schools, recreational areas and locations easily accessed by the public were of greatest concern. The following is a description of the eight priority sites:

Wooster Junior High School: Surface soil in the north playing field at the school was contaminated with lead, as high as 1797 ppm, asbestos, as high as 30% and PCBs, as high as 44 ppm. Fill was placed in this area in the mid to late 1960's. Children who attended the school and played on the field, teachers, coaches as well as residents living in the neighborhood adjacent to the school were likely exposed to contamination at this site.

Short Beach Park: Contamination was identified in surface soil at a number of recreational areas at Short Beach Park. These areas included softball, little league and soccer fields as well as the basketball and platform tennis court areas. Screening data from these areas indicated that lead was identified as high as 860 ppm, asbestos as high as 15% and PCBs as high as 5 ppm. The use of waste for fill in this area began in the 1950's as an extension of the Dorne Drive landfill located adjacent to Short Beach Park. People who participated in activities on any of these fields or recreational areas were likely exposed to waste.

Vacant Lot/End of 4th/5th Avenue: This vacant lot, located within a highly residential area was contaminated with lead as high as 8400 ppm, asbestos as high as 80% and PCBs as high as 15 ppm. The lot covers approximately three acres and was used by children in the neighboring residential area as a recreational area. It is believed that fill material was disposed of in this area in the 1960's.

Spada Area/Commercial Properties on Ferry Boulevard: This area is composed of commercial properties to the west of Ferry Creek and residential properties to the east of Ferry Creek. Some of the highest levels of contamination were located in this area with lead concentrations as high as 10,000 ppm, asbestos as high as 90% and PCBs as high as 160 ppm. People who work or shop at the commercial properties and people who live in the residential area were likely exposed to the contamination identified in surface soil. This area was filled with waste in the 1960's and 70's.

Morgan Francis Area: This commercial property houses a small tool shop and a large parcel of land. Lead was identified as high as 10,000 ppm, asbestos as high as 90% and PCBs as high as 44 ppm. Workers at the tool shop and trespassers were likely exposed to this contamination.

Lot K/Elm Street: This is an occupied private residence. Residents of this home may have been exposed to elevated levels of lead, asbestos and PCBs identified in surface soil.

Housatonic Boat Club: The Housatonic Boat Club is a private yacht club. During the late 1960's and 70's fill was used to build up the area where the Boat Club is now located. Lead was identified in surface soil as high as 10,000 ppm, asbestos as high as 90% and PCBs as

high as 100 ppm. Members of the Boat Club and their guests were likely exposed to this waste.

Raybestos Memorial Field and some adjacent properties: This field is located adjacent to the Raymark facility and covers approximately 13 acres. The area was used as a disposal site for Raymark waste as early as the 1940's through the 1980's. Team members that played on the field as well as spectators were likely exposed to asbestos, the predominant contaminant identified at this site.

3.) Additional Site Investigations and Identification

In addition to the eight priority areas, investigations were initiated at the remaining six areas and residential, recreational, and commercial properties located near and within the priority areas. This included the investigation of approximately 25 commercial areas, approximately ten recreational and municipal areas, and approximately 350 residential properties. By the fall of 1996 waste materials were identified on 46 private residential properties.

For every property where waste was identified, site visits were made to inform the residents, owners and/or workers of the findings. During these visits a representative from the health agency responded to any health questions and a representative from the EPA provided information on the sampling results and future plans for site remediation.

4.) Environmental Sampling and Potential Health Impact Determination

Soil sampling included grab samples at 0-3 inch depths. All samples were screened for lead, asbestos and PCBs. These contaminants were selected because: 1.) they are indicative of Raymark waste; 2.) there are screening methods available for these contaminants; and 3.) these contaminants presented the most significant health threat. Ten percent of all the surface soil samples were sent for laboratory confirmation. This also included analysis for volatile and semi-volatile organic compounds, pesticides, dioxin and metals.

For every site that was investigated, staff from the Agency for Toxic Substances and Disease Registry (ATSDR) and the Connecticut Department of Public Health (DPH) reviewed the data and made a determination of whether the waste presented a risk to human health. The health determinations were made on a site specific basis and considered the level of contamination, extent of contamination, current land use (whether the property is residential, recreational, commercial or industrial influences the estimate of the amount of exposure people have to the contaminants), and the location of the contamination on the property. Although inhalation of fugitive dust and vapors is possible, the primary route of exposure was considered to be direct contact with the waste material. A well survey revealed that property in the area relied on the public drinking water system which was not contaminated by Raymark waste.

Once all of these factors were considered, each property was assigned a health categorization:

No problem: No evidence that there is waste present on the surface of residential or recreational property, or no evidence that people have access to the contamination on commercial or industrial property.
Possible health hazard: Evidence that waste may be on the property below the surface, or at low levels. There is little or no potential for people to be exposed to the waste.
Health hazard: Evidence that waste is on the surface of the property with the potential for people to be exposed to the waste.
Imminent health hazard: Evidence that waste is throughout the property with a high potential for people to be exposed to the waste.

5.) Environmental Remediation

EPA and DEP conducted interim measures to ensure that human contact with the waste would be minimized on properties that were determined to pose a health hazard or imminent health hazard. Interim measures included placing temporary cover over waste and fencing areas to limit access.

Permanent remedies have been completed on many properties. On residential properties the waste was removed and returned to the Raymark site for permanent disposal. At Wooster School the waste was also removed and returned to Raymark. The Raymark facility itself is in the process of being stabilized in preparation for development as a shopping plaza. The buildings at Raymark have been removed and the waste has been capped as part of the permanent clean up process for Raymark.

Despite efforts to quickly identify the waste and stop exposures, residents of Stratford expressed numerous concerns about adverse health effects associated with past exposures. These health concerns motivated a number of preliminary health data evaluations.

B. PRELIMINARY EVALUATION OF HEALTH DATA

Existing data bases including the Connecticut Tumor Registry and the Connecticut Birth Defects Surveillance Program were used to assist in the preliminary evaluation of health data. In addition, a blood lead screening program was offered to respond to citizen concerns about lead exposure.

1). Blood Lead Screening Program

A blood lead screening program was initiated as a result of concern about lead exposure associated with Raymark contamination. Because lead was found at very high levels in surface soil at locations easily accessed by the public, the DPH and local health officials recommended blood lead screenings, particularly for children under the age of six. Any screening result of 10 ug/dL (micrograms of lead per deciliter of blood), or greater, was followed up for a confirmatory test.

The DPH reviewed the blood lead levels and questionnaires of 1287 people who participated in the voluntary blood lead clinics offered by the Stratford Health

Department. Of the 1287 people (342 were children less than age 6), only 13 people (6 were children less than age 6) had confirmed blood lead levels of 10 micrograms of lead per deciliter of blood (ug/dl) or greater. Ten micrograms of lead per deciliter of blood is the lowest level at which adverse health effects have been documented.

Based on national statistics and the percent of older housing stock in Stratford, DPH expected the number of people found with an elevated blood lead level to be higher than what was seen in this evaluation. However, because the blood lead screening program was voluntary, it is likely that the people tested did not accurately reflect the blood lead levels of all persons in Stratford. Blood lead levels may have been low because of several factors. People who were at greatest risk of exposure may have chosen not to participate or may have gone to a private physician. The screening clinics began in the spring when outdoor activities were just beginning and access to known waste sites was already being restricted. Exposures that may have resulted from playing on soil contaminated with lead in the fall may have been undetectable at the time of the lead screening clinics which were held several months after exposure stopped.

2.) Birth Defects

The number and rate of birth defects in Stratford and the State of Connecticut were evaluated using Data from the Connecticut Birth Defects Surveillance Program (CBDSP). The CBDSP was funded for only a short period and data was available for the years 1983, 1985, and 1986 only. The CBDSP used information gathered from the birth certificate, death certificate, and hospital discharge summary. The rate of birth defects per 1,000 live births for Stratford residents were compared to the same rates for the state.

Holger Hansen, of the University of Connecticut School of Medicine, Division of Epidemiology and Biostatistics summarized the three years of data by stating that "No birth defect stands out as particularly excessive in Stratford. The slightly elevated rates of cleft lip and palate and of musculoskeletal anomalies are not statistically significant, and neither is the total incidence of birth defects in Stratford, compared to the rest of the state."

3.) Cancer Occurrence in Stratford from 1958 to 1991

Information on the occurrence of cancer in Stratford was obtained from the DPH, Tumor Registry on cancer of the bladder, brain, breast, kidney, liver, lung, rectum, and testis; and non Hodgkin's lymphoma, mesothelioma, leukemia, soft tissue sarcoma and all sites combined for the years 1958-1991.

The rates of cancer incidence in Stratford are what would be expected based on State rates for the majority of the cancer sites studied adjusting for age and population. Cancer of the brain, breast, kidney, liver, lung, rectum, testis; and non Hodgkin's lymphoma, leukemia, and soft tissue sarcoma were studied and were found to be not statistically significantly different from State cancer rates. For bladder cancer, mesothelioma, and the total of all cancer sites combined there were some differences in Stratford rates in comparison to State of Connecticut rates. Table 2 presents these results.

The rate of bladder cancer among Stratford residents was 14 percent higher than the state rate. The most common risk factors associated with the development of bladder cancer are certain occupational exposures (including working with benzidine based dyes), history of frequent bladder infections, and smoking. Some studies have also linked bladder cancer with drinking water that has high levels of chlorination by-products. For mesothelioma there were five more cases than would be expected based on state rates from 1958 to 1991. Mesothelioma is a very rare cancer of the lining of the lung that is associated with exposure to asbestos. This tumor is most commonly linked with persons who had an occupational exposure to asbestos. While the rate of mesothelioma was higher in Stratford than the State, the number of cases was small and not considered statistically significant.

DPH also reviewed more detailed information on cancers that occurred to persons less than 25 years of age. Records in the Tumor Registry were reviewed to determine if there is any type of cancer that was more likely to be diagnosed among younger persons. For the period 1958 to 1991 there were a total of 130 cases of cancer among persons less than 25 years of age while it was expected that 107 cases would occur. While there was a 22 percent increase in the number of cancers among younger persons there was no apparent pattern in the type of cancers that occurred among this age group. No one cancer type demonstrated a significant excess of cases. Since no one type of cancer was more common among this group it is less likely to indicate a common cause. However, reviewing cancer incidence among this group may serve as an indicator of cancer risk.

C. RATIONALE FOR STUDY OF CANCER

Cases of bladder and testicular cancer, mesothelioma, and all types of cancer diagnosed among persons 25 years of age and younger in Stratford were selected for inclusion in this study based on the preliminary review of cancer incidence, epidemiological studies, and community concerns. The preliminary review of cancer incidence (presented above) indicated elevations in the incidence of bladder cancer, cancer among those less than age 25, and mesothelioma. Epidemiological studies have shown that exposure to asbestos is a risk factor for mesothelioma and exposure to solvents is a risk factor for bladder cancer. Asbestos and solvents are components of Raymark waste. Testicular cancer was also included in this study because of concerns raised by the citizens of Stratford. Citizens expressed concerns regarding cancer among persons less than 25 years of age and testicular cancer because some of the Raymark waste was used as fill at recreational areas at Short Beach Park and Wooster Middle School. It was felt by the citizens that younger persons would have had the potential for more exposure to contaminants at these recreational areas and may have been at greater risk of developing cancer from that exposure.

Two different time periods were utilized in this study. The preliminary evaluation of health data and the time trend analysis both focused on tumors occurring between 1958-1991. The wide range of time allows for the examination of cancer incidence in five year intervals before and after off-site exposure to Raymark waste would have occurred. This provided an in-depth overview of cancer incidence in Stratford, as

compared to the State of Connecticut, over time.

Spatial analyses of cancer incidence were then conducted using Geographic Information Systems (GIS) for the years 1968-1991 to evaluate the distribution of cancer cases within the town of Stratford.

The following is a brief review of the epidemiology of the tumor sites selected for study with emphasis on possible environmental risk factors.

1.) Mesothelioma

Approximately 2,000 cases of mesothelioma are diagnosed annually in the United States. The incidence of mesothelioma has increased since the 1950's with the rates being highest among those born around 1910 and declining among those born later. [4] Mesothelioma is defined as a primary malignant neoplasm of the pleura, a part of the respiratory tract.[5] The relationship between asbestos exposure and mesothelioma was first reported by Wyers.[6] Since then, there have been numerous studies that link asbestos to mesothelioma. Work by Wagner, Sleggs, and Marchand has shown that the exposure could come from environmental sources as well as from occupational sources. In Wagner et Al.'s study of thirty-three cases from the North West Cape Province of South Africa, exposure was occupational in some of the cases and residential or environmental for the others. One of the findings of this study was that there appeared to exist a carcinogenic hazard for relatively low levels of asbestos fiber exposure.[7] Concern continues regarding the potential effect of asbestos in buildings such as schools, however, the levels are believed to be low and present little risk of mesothelioma.[8]

There is a long latency period between first exposure to asbestos and the development of mesothelioma. Cancer rarely develops in less than 20 years and incidence may not peak until 45 years after exposure.[4,9] Rates of mesothelioma have continued to increased since the 1950's and the increase may not end until the late 1990's.[4]

2.) Bladder cancer

Approximately 52,900 new cases of bladder cancer occur in the United States each year and 11,700 die from the disease. Bladder cancer occurs almost three times more often among men than women. Whites have a higher incidence of bladder cancer than blacks, although blacks are more likely to die from the disease.[10] The rate of bladder cancer among males increased 72% from the late 1940s to the mid 1980s; however, mortality has declined possibly due in part to an increase in the proportion of cases determined as in situ.[11]

Cancer of the bladder has been shown to be elevated among certain occupational groups including those who manufacture dyes, aromatic amines, leather, rubber, and aluminum. Painters, dry cleaners, truck drivers, and those who work with organic chemicals have also experienced a higher risk of bladder cancer.[12,13,14,15] Among women the occupations found at greater risk included metal working, and chemical processing.[16] A study by Meigs and coworkers of a benzidine manufacturing facility in Connecticut

found bladder cancer to increase with exposure to benzidine, and also found that the risk of bladder cancer decreased with the cohort of workers after control procedures had been instituted.[17] A later study of the same facility determined that workers experienced increased risk of bladder cancer even though process changes had been made to replace benzidine production with production of dichlorobenzidine, ortho-dianisidine and ortho-tolidine.[18] The risk of bladder cancer attributable to occupational exposures is estimated to be approximately 11 percent for women and 21-27 percent for men.[13,14,16]

Smoking is consistently linked with bladder cancer and has been found to increase a person's risk by two to three times compared with risk of non-smokers.[19] Coffee, alcohol, and artificial sweeteners have all been extensively studied as risk factors for bladder cancer, however, none of these have demonstrated consistent positive associations.[12] Drinking water containing disinfectant by-products has been associated with increasing the risk of bladder cancer first in ecologic[20,21] and then in case control studies.[22,23] A meta-analysis of studies linking disinfectant by-products with bladder cancer suggests the attributable risk may be nine percent.[24]

A study of a cluster of bladder cancer cases in northern Illinois found an elevated rate of bladder cancer in a town which had its municipal water supply contaminated with solvents which had leached from a landfill. The increased risk for males was RR= 1.7 (95% CI 1.1, 1.9) and for females was RR=2.6 (95% CI 1.2, 4.7). The study had been initiated to evaluate higher bladder cancer mortality in the northeastern counties of Illinois. Incidence data were gathered from local hospitals and age standardized incidence ratios were calculated for counties and zip codes in the area. Records for ground water contamination were evaluated for the county with the bladder cancer "cluster". Only the town with the "cluster" was found to have any ground water contamination. Two of four municipal drinking water wells were located within a half mile of a 22-acre landfill that had leached trichloroethylene, tetrachloroethylene, and 1,1-dichloroethane into the water supply.[25]

3.) Testicular Cancer

Approximately 7,400 new cases of testicular cancer occur each year while only 370 die from the disease. The incidence of testicular cancer has been increasing; however, screening and improved treatment have contributed to a decrease in mortality.[26] Whites tend to have higher incidence rates than blacks, with Asian and Hispanic males having rates between those of blacks and whites.

Unlike most solid tumors the incidence rate for testicular cancer does not increase with age, but reaches peak incidence among men 24-44 years of age, then decreases, and is relatively rare among men greater than 55 years of age. While much of the etiology of testicular cancer is unknown, the age distribution supports the hypothesis that exposure to risk factors in utero, or early in life may be more important than exposure to risk factors in adulthood.

Genetic factors are associated with some cases of testicular cancer including a specific structural chromosomal abnormality, isochromosome (12p). Undescended testes and inguinal hernia are risk factors for testicular cancer. Infectious diseases have been

explored as possible risk factors because of the age distribution of testicular cancer. The epidemiology of testicular cancer and that of Hodgkin's disease possess similarities, with each having elevated titers of antibodies to cytomegalovirus and Epstein-Barr virus. While trauma to the testis and increased temperature have been associated with testicular cancer, riding a horse, bike or motorcycle have not consistently been shown to increase risk.[27]

While epidemiological evidence does not link testicular cancer with compounds identified in Raymark waste, citizens expressed concern about the incidence of testicular cancer among Stratford residents, and therefore, testicular cancer was included in this study.

4.) All Sites Less Than Age 25 - Early Onset Cancer (EOC)

Nationwide, cancer is the fifth leading cause of death among persons less than age 25. The majority of those cancer related deaths are either leukemia, brain and central nervous system cancers or non-Hodgkin's lymphomas.[10]

Different cancer sites have different etiologies. Therefore, grouping all cancer sites together is not indicative of a common cause but serves more as a benchmark for tracking cancer in the overall population. Cancer under age 25 was selected because this was the population of interest to the Stratford community. Cancer among persons in this age group is referred to as early onset cancer (EOC) for this study.

D. RATIONALE FOR STUDY OF BIRTHWEIGHT

Newborn infants were selected for this investigation because the developing fetus is particularly sensitive to chemical exposures. Birth certificate data from Stratford during the decade prior to interventions (1982-1992) were used to evaluate whether infants had been adversely affected by exposures to hazardous waste during gestation.

More than 900 compounds are known to cause birth defects in laboratory animals.[28] In people, reduced birthweight has been shown to be associated with environmental exposures during pregnancy to wood preservatives.[29] Xu et al. found significant associations between gestational age and ambient exposures to suspended particulates and sulfur dioxides.[30]

Of the chemicals found at levels of health concern in the Raymark waste, lead and PCBs have been shown to be associated with adverse pregnancy outcomes. Many lead compounds have been shown to be teratogenic in the laboratory. Lead crosses the placenta as early as the 12th week of pregnancy, and may accumulate in the fetus.[31]

Occupational exposure to lead has been shown to be associated with spontaneous abortion and low birthweight.[32,33] At lower environmental exposures, the evidence of an association between lead exposure and adverse pregnancy outcomes has been contradictory. Even with high levels of lead exposures experienced by pregnant women living near lead smelters, the results have been inconclusive. An association between proximity to the smelter and spontaneous abortion was found by Nordstrom in Sweden[33],

while McMichael et al. and Murphy et al. did not find associations in their studies conducted in Australia and Yugoslavia.[34,35] Factor-Litvak et al. investigated gestational age and birthweight near the smelter in Kosovo, Yugoslavia.[36] They compared birthweight and gestational age in the town surrounding the smelter to another town. No association was observed. The study was strengthened by measurement of the maternal blood lead measured at mid-pregnancy, at delivery, and in the umbilical cord. Comparisons of gestational age and birthweight across blood lead concentrations did not demonstrate a significant difference.

Evidence of an association between environmental exposure to PCBs and birthweight or other reproductive outcomes is sparse. Exposure to PCBs can occur from ingestion of contaminated soil and food, inhalation of contaminated dusts, and dermal absorption. PCBs are stored in body fat, and excretion is extremely slow. Fetuses are particularly sensitive to PCBs because PCBs readily cross the placenta and fetuses lack the liver enzymes that are responsible for breakdown and excretion of the contaminant.[3] In a study of the reproductive outcomes of 13 pregnant women who had been poisoned by rice oil contaminated with PCBs, 12 of the 13 newborns were smaller than the national average.[37]

Fein et al. investigated the birthweights of 242 infants born to mothers who had consumed large quantities of Lake Michigan fish contaminated with PCBs.[38] PCB levels in umbilical cord blood at birth were positively correlated with lower birthweight. Interestingly, fish consumption rates correlated poorly with PCB levels in the cord blood, and fish consumption did not predict birth size. These findings underscore the value of using biological markers as a way of modeling exposure in preference to dietary or geographic surrogate variables.

In contrast to the Fein study, Rogan et al. found no association between PCB levels and birthweight in their study in North Carolina.[39] They measured PCB levels in the placenta, maternal and cord serum, and breast milk. The levels reported represented background body burdens and were lower than those reported by Fein et al. in the Lake Michigan study. Similarly, Berkowitz et al. detected no differences in the maternal serum PCB levels between 20 women who had a spontaneous preterm birth and 20 matched women who delivered at term.[40]

Toxicological studies of exposure to PCBs during pregnancy and its relation to birthweight suggest a threshold effect, with a dose-response relationship seen at sufficiently high exposures. Although evidence of an association between PCB exposure and LBW is ample, it remains contradictory and inconclusive. For example, in a study of acute exposure in rats, Spencer found no association at dietary doses of Aroclor 1254 as high as 100 mg/kg/day.[41] On the other hand, other studies showed that birthweights decreased after acute exposure in mice, rats, and guinea pigs.[42] Experiments using rhesus monkeys have demonstrated an association between chronic exposure to PCBs and abnormally small off-spring and early births.[43] Although these findings do not prove a causal association between PCB exposure and LBW in humans, such an association is certainly plausible.

The latency period from an environmental exposure to a health outcome at birth is relatively short. This contrasts with investigations of carcinogenic outcomes, in which the latency between chemical exposure and resulting disease can be three decades or

more. Long latency periods require estimation of exposures that occurred decades before individuals became symptomatic. In an investigation of birth outcomes reported on the birth certificate, the time since exposure is usually much shorter and the residence history is usually more stable.

Another advantage of focusing on birth outcomes is that the birth certificate provides information on the entire population. In an ecological cancer investigation, only cases are recorded by the tumor registry, and there is not comparable information on the individuals who did not develop the disease. With the birth certificate, information is recorded systematically on all births, irrespective of the birth outcome.

Birthweight is measured routinely at birth. It can be expected to be measured and reported accurately at all birth facilities, and it is arguably the most accurate of all the health outcome measures coded on the birth certificate. And, although it is not a disease, birthweight does have a strong correlation with infant survival[44] and is indicative of intrauterine growth.

Very low birthweight babies (VLBW) are less than 1500 grams, while low birthweight babies (LBW) are less than 2500 grams. VLBW babies, in particular, are subject to numerous adverse health conditions which affect their survival and their quality of life. If chemical exposures were adversely affecting the developing fetus, it is likely that this effect would be manifest in the birthweight of the exposed infants. Conversely, if reduced birthweight is associated with the exposure, this may be a strong indication of an adverse public health outcome.

In the United States, LBW is the major determinant of neonatal infant mortality.[44] Mortality rates increase with decreasing birthweight below 2500 grams. LBW infants are 40 times more likely to die within the neonatal period (< 28 days of age) as compared to babies over 2500 grams, and the neonatal mortality of VLBW babies is 200 times that of normal birthweight infants.[45] The measure of the association between LBW and infant (< 1 year of age) mortality is highly variable, depending on the age at death and the cause of death.[46] Most infant deaths in developed nations occur to babies in this neonatal period,[44] and almost two thirds of all neonatal deaths occur to LBW infants. After the first month, the association between LBW and infant mortality declines, and one fifth of post neonatal deaths occur to LBW babies.[45]

There has been a dramatic decline in infant mortality in the United States from 100 per 1000 live births in 1900[44] to 8.3 per 1000 in 1993.[47] The decline in the number of low birthweight infants is presumed to have been less pronounced, and there were 71 LBW infants per 1000 births in 1991.[48] As infant mortality has decreased, a larger proportion of deaths occur in the neonatal period. The contribution of low birthweight to this remaining infant mortality has increased.[44]

In addition to the relationship between birthweight and mortality, LBW also contributes to morbidity and the use of health care resources. LBW babies who survive are at much greater risk of developing neurologic handicaps,[44] having a serious congenital anomaly[49], and of developing lower-respiratory-tract conditions[50]. The length of stay in the hospital[51] and the number of hospital re-admissions[52] are inversely correlated with birthweight. Even by the age of 8-10 years old, there is some evidence that children who were VLBW are still three to four times more likely to be hospitalized during a given year.[53] The public health consequences of LBW extend even beyond the child's health

status and costs of care. LBW infants are more difficult for their family to care for, and their care can place a great deal of financial and emotional strain on the family.[54]

E.) EXPOSURE ASSESSMENT NEAR HAZARDOUS WASTE

ATSDR emphasizes the importance of understanding exposure by requiring the identification of completed exposure pathways as part of the Public Health Assessment process. As defined by ATSDR, a completed exposure pathway consists of the following five elements: source of contamination; environmental media and transport mechanisms, such as water, air or soil; point of exposure such as a well or playground; route of exposure; and the receptor population (the exposed persons).[55]

In addition to defining the exposure pathway, it is helpful to quantify the exposure by determining the level of exposure and the length of time of exposure. Ideally, there would also be a measurable biological index of the exposure. Unfortunately, all of these components are rarely present when assessing exposure in a health study. Therefore, environmental epidemiology often relies on various methods of estimating exposure. One method employed in studies of health effects near hazardous waste sites is to estimate the distance of the receptor population from the source of contamination.

In a study of congenital malformations near hazardous waste sites by Geschwind and colleagues, a Geographic Information System (GIS) was used to map the maternal residential address of 9,313 children born with congenital malformations and 17,802 controls.[2] The authors combined the New York State Congenital Malformations Registry with the Hazardous Waste Site Inspection Program. The residential addresses were mapped to the center of the Census block, or the center of the postal carrier route (which the authors say is comparable to a Census block group), or center of the zip code area. Cases and controls were defined as higher risk of exposure if they lived within one mile of the 590 hazardous waste sites mapped in New York state. Higher rates of congenital malformations were associated with higher risk of exposure OR=1.12 (95% CI 1.06,1.18). In addition to measuring distance to the waste site an exposure risk index was developed that was based on the distance to the hazardous waste site and a hazard ranking score. The hazard ranking score was derived during EPA inspections that evaluated the toxicity of identified chemicals at the hazardous waste site and their ability to migrate; the probability of contaminants transporting from the site; and the relative importance of environmental exposure routes. By employing the hazard ranking score and by improving the accuracy of the exposure assessment, the authors were able to demonstrate a dose response relationship between hazard estimation and risk of congenital malformations: no exposure risk OR=1.00; low exposure risk OR= 1.09 (95% CI 1.04, 1.15); high exposure risk OR=1.63 (95% CI 1.34, 1.99).

Despite the limitation of the one mile buffer zone, Geschwind and coworkers present two important methodological advances in this study. These two advances include a three-level exposure score, and a tiered approach of progressing from the most general hypothesis tests to the most specific. They categorized the three levels of exposure based on a formula incorporating terms that included chemical toxicity,

contaminant transfer off-site, and the route of exposure. This three level score enabled the investigation of a potential dose-response relationship between increasing exposure and congenital malformations. Secondly, the authors focused their study to include tests of possible associations between particular congenital defects and particular chemical groups. Their findings were suggestive of an association between proximity to waste sites and congenital malformations. However, the authors could not rule out the possibility that residual confounding or factors they were not able to consider was responsible for the observed relationship. The demographic characteristics of the population living near the waste is not comparable to those living farther away. Incomplete control of confounding is a limitation of the analysis of existing data bases.

Sosniak et al. explored the possible association between proximity to hazardous waste sites and low birthweight nationwide.[1] The authors did not show an association between maternal proximity to hazardous waste and increased risk of low birthweight OR=0.99 (95% CI 0.86-1.16). Their methodology involved the use of zip code centroids to establish both the maternal and NPL site locations. Mothers with zip code centroids within one mile of an NPL site were considered exposed. The mother's address was not available in the National Maternal and Infant Health Survey. The study used a one mile buffer zone as its exposure classification. Highly correlated confounding variables were used in the same logistic regression model, including education, income, age, smoking, and drinking status. The authors point frankly to many important limitations of their ecological methodology. They conclude that their methodology was innovative and economical, but not efficient due to the limitations in exposure definition.

Linos et al. found a statistically significant association between residential proximity to a factory and leukemia and Non-Hodgkin's lymphoma.[56] They measured exposure by asking cases and controls to recall if factories were within 1/2 and 2 miles of their home, and those who reported living near factories were classified as having been exposed. Many more controls than cases were excluded from their analysis, and recall bias in combination with residual confounding could explain their results.

The above methods focused on measuring distance from an exposure. Another method of defining exposure is to assign exposure classification based on established political boundaries such as states, counties, towns, postal codes, or census tracts. The advantage of this method is that population data are often collected or aggregated for these political subdivisions and thereby enable the calculation of incidence rates based on the population.

The rates of gastrointestinal cancer in counties in New Jersey were evaluated in relation to the distribution of waste disposal sites. Death certificate data for the years 1968 to 1977 were used to compare the rates of various gastrointestinal cancers with national and SEER area cancer rates. Twenty of the 21 New Jersey counties studied were found to have higher rates of gastrointestinal cancer. The factors most often correlated with higher rates of cancer mortality were degree of urbanization, population density, and presence of chemical waste disposal sites.[57]

Location of hazardous waste sites has been included in an analysis by Griffith that demonstrated increased cancer death in U.S. counties with hazardous waste sites.[58]

Mallin evaluated bladder cancer incidence in northern Illinois for the period 1978 to 1985. Incidence was first studied at the county level and then narrowed to postal

codes. One of the two postal codes with excess bladder cancer was in a town which had drinking water wells contaminated with trichloroethylene, tetrachloroethylene, and other solvents from a landfill site. This study evaluated potential exposure to environmental contaminants in the drinking water for only the county containing the postal codes with elevated bladder cancer risk. The authors reviewed records from the USEPA and the Illinois Environmental Protection Agencies, and the Illinois Department of Energy and Natural Resource to identify sources of groundwater contamination.[59]

Shaw et al. Also found no association between residence location and congenital malformations or birthweight.[60] They defined exposure by categorizing each of the census tracts in the San Francisco bay Area into one of three categories representing contamination levels. Births to mothers living within a census tract were assigned the categories associated with that census tract.

Goldberg et al. conducted an investigation of the possible association between proximity to a solid waste landfill in Montreal Canada and low birthweight and cancer incidence. Persons were believed to be exposed to chemicals in the biogas from waste decomposition and air contaminants released from incineration of the waste. They defined exposed zones based on the proximity of postal code areas to the landfill. They compared the postal codes nearest the landfill to postal codes with similar demographic characteristics. The odds ratios for certain cancers were elevated, although not significantly.[61] The odds of low birth weight (1.20) and small for gestational age (1.09) were significantly elevated, while very low birth weight and preterm birth were not increased.[62] Although suggestive of a possible association, the study was inconclusive. Most importantly, one can not assume that the control and exposure neighborhoods were comparable in all respects except for the exposure of interest. Residual confounding can not be dismissed as a likely explanation for the findings of their investigation. The results do warrant further investigations, and suggest the possibility of using ecological study designs to identify exposures needing further investigation.

In the evaluation of cancer near Love Canal in New York, cancer incidence in the census tract containing Love Canal was compared to data from the New York Cancer Registry. Standard Incidence Ratios (SIRs) were calculated for all sites of cancer for the period 1966 to 1977. Liver cancer, lymphoma, and leukemia were examined for the period 1955 to 1965 as well. The authors concluded that there was no evidence for higher cancer rates associated with residence near the Love Canal.[63]

III. METHODS

A.) STUDY DESIGN

This is an ecological study using routinely collected regulatory and surveillance data. Although ecological, the measures of proximity to the Raymark waste sites are calculated to all maternal addresses in the birthweight analysis, and to all cancer cases in the cancer investigation. Similarly, the demographic factors controlled for in these analyses are also considered at an individual level. Only the U.S. Census population used to define the control population in the cancer investigation must be aggregated at the block group level, as is characteristic of ecological designs.

B.) DATA SOURCES

1.) Environmental Data

The majority of sampling for contaminants from Raymark Industries was conducted by the Environmental Protection Agency to determine the extent of contamination and the location of off-site disposal sites.

The Environmental Protection Agency produced maps using geographic information systems (GIS) technology which detailed the sampling locations. For each of the properties that were determined to be a health hazard or an imminent health hazard by ATSDR or DPH the property outlines were digitized using GIS by the University of Connecticut, Department of Geography. As of September, 1994, thirty nine separate waste locations were identified. Figure 2 Map of Exposure Models Utilized In Stratford, Connecticut, September 1994 presents these locations and forms the basis of the exposure models described below.

2.) Cancer Incidence Data

Information on all incident cases of bladder, mesothelioma, testicular, and all sites less than age 25 diagnosed in Stratford residents was obtained from the Connecticut Department of Public Health Tumor Registry (CTR). The CTR has been gathering data on every tumor diagnosed to residents of Connecticut since 1935. It is the oldest population based tumor registry in the world and the largest in the United States. Connecticut participates in the National Cancer Institute's SEER (Surveillance, Epidemiology, and End Results) program. SEER is a program which collects and analyzes cancer data.

Two different time periods were utilized in this study. The preliminary evaluation of health data and the time trend analysis both focused on tumors occurring between 1958-1991. Data for these analyses were obtained from the CTR computerized file system.

Spatial analyses of cancer incidence were then conducted using Geographic

Information Systems (GIS) for the years 1968-1991 to evaluate the distribution of cancer cases within the town of Stratford. Data on individual cases for these analyses were extracted manually from CTR paper files.

At the time this study started, 1991 was the last year the CTR had complete data. This time period for the spatial analyses was selected because it allowed for 10 - 15 years to pass from when Raymark Industries started to dispose of waste within the community (start of exposure) and when tumors possibly resulting from this exposure would be expected to appear due to latency.

For the tumor sites selected, the data extracted for the spatial analyses included diagnosis, address at diagnosis, date of diagnosis, gender, and date of birth. Additional data were considered but could not be used due to missing or incomplete information on the majority of the records. Such data included smoking history, occupation, and family history of cancer. The CTR does not have address information on all cases in their electronic database. The hard copy tumor files were reviewed and address information was obtained for use in the GIS.

The CTR goes to great length to ensure accurate reporting and coding of tumor information.[64] All incoming tumor records are reviewed by a medical record technician for completeness of data. Incomplete records are sent to a field unit for follow-up. Complete records are sent to the coding staff where proper ICD-9 Oncology codes are assigned. The coded record is verified and sent for data entry. There are also quality control processes in the data entry programs. Pre-edit checks and validations help to ensure accurate data entry. CTR staff members attend monthly in-house training conferences. These conferences serve to review the results of the various quality control activities. Coding and abstracting workshops are also presented to the CTR staff during these meetings.

The CTR receives reports from a variety of sources including hospitals, private laboratories, nurses, autopsy reports, and death certificates. Hospital reporting has remained fairly constant at approximately 97% of all reported cases.[65] Connecticut had an estimated completeness of case reporting rate of 96.1 percent.[66]

The reporting is currently accomplished by having the reporting source send in a form with the appropriate information provided. The CTR has begun to design and construct a database application which will facilitate on-line, electronic reporting of tumors. They are also in the process of entering the older data, pre-1973, into an electronic database.[67] This will greatly aid future studies of this type.

The 1990 census data at the block group level were used to estimate the population at risk or referent population for the cancer incidence analysis. Population and demographic information has been extracted from the 1990 U.S. Census Summary Tape 3A. Data were imported into the GIS and statistical software that was utilized for this project.

The 1990 census population figures were used in part to maintain geographic integrity. The 1990 census geographic boundary files were used by the EPA and in preliminary studies, therefore, 1990 demographic data was used. The total population remained constant over the study period of 1968 to 1991, however, the population tended to age during this time. Table 3 presents the 1970, 1980, and 1990 population data stratified into the age groups used in the cancer analyses.

The geographic unit of analysis used in this project is the block group. Census blocks are the smallest geographic area for which the Bureau of the Census tabulates

decennial census data. The block group is the next level above census blocks. Block groups are a combination of blocks which are a subdivision of a census tract. Block groups are also the smallest geographic area that the Census tabulates and publishes sample data. The Census states that a block group should ideally contain 400 housing units with a maximum of 500 and a minimum of 250 housing units.[68]

3.) Birth Data

The Connecticut Department of Public Health Vital Records (CTVR) maintains electronic databases of all births occurring in the state of Connecticut. All birth data for the time period 1982 through 1991 was obtained from the CTVR mini-computer. During the period of 1982 to 1991 the management of Vital Records was in a period of transition. In 1988 a change in the birth certificate occurred which required that the birth data be received in two data sets. The first data set was from 1982 to 1987 and the second data set was from 1988 to 1991. Some variables were in different formats between the two time periods.

This is an ecological study using routinely collected regulatory and surveillance data. Although ecological, the measures of proximity to the Raymark waste sites are calculated to all maternal addresses in the birthweight analysis. Similarly, the demographic factors controlled for in these analyses are also considered at an individual level.

The birth certificate data base was reviewed for variables that could confound or modify a relationship between exposure and birthweight. Preliminary models were developed that incorporate the known risk factors for LBW. The variables from the birth certificate that were reviewed include: maternal age, education, race, smoking status, weight, marital status, pregnancy interval, prior births, prenatal care, occupation, medical risk factors, previous fetal or infant death, and multiple births. These variables were re-coded as necessary and the validity of each variable was considered.

Unless evidence was observed to suggest an improvement in the LBW models from an alternative coding scheme, coding of variables for the LBW and VLBW analyses were chosen for consistency with the mean birthweight analyses. Simple linear models predicting birthweight were developed. Multiple regression models predicting birthweight were developed subsequently.

Variables that were used to control for confounding in this study were those that were recorded consistently on the birth certificate, and are strong predictors of birthweight. These demographic variables per se were not causally associated with LBW. Instead they can be expected to be associated with other risk factors that were not measured (or were incompletely measured) on the birth certificate. For example, marital status, maternal age, and race could be associated with nutritional status during pregnancy, which in turn is a cause of adverse birth outcomes. Since the underlying risk factors are not recorded on the birth certificates, it is essential to control for these demographic characteristics when evaluating the exposures of interest.

Several other variables may be important causal risk factors of low birthweight, and are recorded on the birth certificate. These include smoking, maternal weight at delivery, the interval between births, parity, prenatal care, medical risk factors, and

occupation. However, these variables were not included in many of the final models in order to preserve statistical power (they are missing for a large proportion of the data), or because limitations in validity introduced concern about bias.

In the beginning of the study period paper copies of birth certificates were sent to DPH. Data entry occurred at DPH with each record being entered twice to ensure accuracy of data entry. Data entry programs were utilized for some variables to identify values that were possibly inaccurate. Birth certificates with discrepancies were sent back to the hospitals for verification. During the study period an automated data entry system was initiated. Hospitals participating in the automated data entry system would perform data entry and then electronically transmit the records to DPH. During the transition period to an automated data entry system the same data entry screens were utilized by DPH and by the hospitals participating in automated data entry.

DPH reciprocates with other states and the provinces of Canada in exchanging copies of birth records for non-residents. Registration of births in Connecticut is essentially 100% complete.

C.) USE OF GEOGRAPHIC INFORMATION SYSTEMS (GIS)

A Geographic Information System (GIS) is an organized collection of computer hardware, software, geographic data, and personnel designed to efficiently capture, store, update, manipulate, analyze, and display all forms of geographically referenced information.[69] The GIS assisted in the mapping of cases, measuring distances between points, delineating census block group areas, and managing spatial data.

Geocoding involved processing the address information against a geographic reference database which placed a point at the approximate location of the address. The address was then converted to latitude and longitude.[70]

Geocoding was accomplished using a commercial upgrade of the US Census TIGER files. The GIS locates the best possible place for each address and places a marker. If the reference database does not have the exact address, it interpolates and places the address in the best logical location.[70] The file that contains the address information was appended with latitude and longitude, census tract, census block group, and census block.[70] Accurate residential address information in the health outcome database and the referencing database are crucial in obtaining high rates of geocoding.

The geocoding was performed by Dr. Ellen Cromley, Associate Professor in the Department of Geography at the University of Connecticut, and by research assistants Stephen McGee and Brian Kuerbitz. Address quality was good and the match rate was relatively high. Only 273 (4.8%) of the maternal addresses and 4 (0.86%) of the cancer cases were not geocoded. The records that did not geocode were excluded from the analyses of exposure based on residence locations.

The address location interpolation can introduce error into the exposure classification process. In this study exposure classification is based on the geocoded location of each birth and tumor record, therefore, errors in geocoding can cause errors in exposure classification. Errors in geocoding can also result in incorrect spatial attributes

such as census tract, block group and blocks being appended to a record.

In order to assess the potential errors introduced by geocoding, the addresses were then verified. Verification involved selecting a ten percent sample of the tumor and birth cases and cross-checking them against the town assessors maps or driving by the actual location in Stratford.

The geocoding process was found to be fairly accurate. Of the 10% sample that were verified, only 0.8% were found greater than 500 feet from the actual location. Some locations were placed on the wrong side of the street (3.5%) which could result in assigning the case to the wrong census block (3.4%) or wrong census block group (0.6%). Because the census block is smaller than the block group there were more errors in assigning the correct block than the block group.

A new database containing the personal identifiers of the maternal residential address for the birth records, and the residential address at the time of diagnosis for the tumor cases, the location code (latitude and longitude), and census block group designation was then developed. The census data (cancer reference population) were assigned the centroid of the census block group as their residential location. This database was used to build the GIS coverage's (map layers) and perform the statistical analyses.

D.) EXPOSURE ASSESSMENT

The geocoded tumor records, birth records, and waste site locations were used to develop three exposure models based on geographic proximity of the case address to the waste sites. Many prior studies have used postal codes or census designations to define exposure. Since geographic boundaries established by the U.S. Post Office or the U.S. Census are arbitrary with respect to chemical exposure, this methodology introduces substantial misclassification. The exposure models in this investigation were developed in an initial attempt to reduce this misclassification.

The three types of models are detailed below. The three models include block groups with waste, distance to the nearest site, and a cumulative exposure model to all sites based on continuous measurements of distance. The development of exposure models based on distance was complicated by the fact that waste was distributed throughout the town. Ultimately the most refined model incorporated both the possibility of exposure to multiple sites and the different probabilities of exposure at the various types of sites. These models were intended to have increasing precision in their use of distance to approximate exposure. The exposure models are presented in Figure 2 Map of Exposure Models Utilized In Stratford, Connecticut, September 1994.

1.) Block Group Exposure Model

A "block group" is a geographic designation for areas that contain approximately 400 housing units. According to the U.S. Census Bureau, they must contain at least 250 and no more than 550 units. Boundaries of block groups follow visible features such as roads, rivers, and railroads.

The town of Stratford contains 46 block groups. In this investigation, block

groups with Raymark waste were defined as "exposed", while those without Raymark waste were defined as "unexposed". The geocoded records (births and cancer cases) were assigned their census block group of residence. Mothers or cancer patients residing in any of the 9 census block groups with waste were considered to be exposed; the unexposed referent population was defined as the remaining 37 census block groups without waste.

2.) Distance to the Nearest Waste Site

The geocoded data facilitated calculation of the distance from each tumor and each birth residence to each waste site. Using these continuous measures of distance, one can develop exposure models that should be more accurate estimates than buffer zones. The simplest of these models is the distance to the nearest waste site from each residence.

Since the birth cohort contains both low birth weight and normal birthweight babies, this type of model works well. However, for the cancer data, only information on the cases exists. Because of this, census data was utilized as the referent population. This has its limitations in that the centroid of each block group was used as the location for the referent population.

Distances were converted from feet to miles in logistic regression models so that the odds ratios could be used to estimate the odds of LBW, VLBW or cancer for each mile of distance. The odds ratio of distance to the nearest site compared lower and higher exposure categories. An association between distance to the nearest site and LBW, VLBW or cancer would yield an odds ratio significantly less than 1.00. To eliminate this potentially confusing problem, the inverse odds ratios and opposite parameter estimates were determined. These inverses represent the odds associated with living each mile closer to the nearest site.

3.) Cumulative Exposure

This model attempts to estimate the potential exposure by adding the probability of being exposed to each of the 39 waste sites. The model incorporated the distance to each waste site, the area of each waste site, and the type of waste site.

Assuming that distance from waste sites is correlated with exposure, this model describes the relationship between distance and the probability of exposure to waste contaminants. This model incorporates the measured distance from each case residence to all the waste sites and includes terms for relating probability of visiting a waste site to exposure.

To relate the combined distance of each residence to the cumulative probability of spending a unit of time at any of the 39 waste sites, the area of each waste site is considered. There are some parts of Stratford where there are numerous contaminated properties near one another. These properties have a set of distance measurements, one for each property boundary. To prevent the number of properties from influencing the exposure score, each distance is multiplied by the area of the property.

It is assumed that the likelihood of visiting a property falls off exponentially with distance. Morrill and Kelly propose that, on average, properties that are three times as far away are one third as attractive since it takes three times as much effort to get there.[71] Lowe and Pederson explain that

> 'Although the exact paths and destinations of any particular individual are unpredictable, the general routes and types of destination can be predicted with confidence....In a survey of British towns, the distance-decay effect was found to be greatest for school and shopping trips, with 75 percent of them within 4 kilometers of home. The effect was less for work and social trips, but even for these, 75 percent were less than 10 kilometers from home.'[72]

The normal distribution is a very common distribution among continuous random variables. In the absence of data, an assumption is that the relationship between distance and the probability of visiting a remote site at that distance can be described using the normal distribution. The use of the normal distribution was suggested by Dr. Heping Zhang, Associate Professor of Biostatistics at Yale University's Department of Epidemiology and Public Health.

An individuals location is described on the X and Y coordinate axis. Centered over each individual's residence is a set of Z axis values, or heights. These heights form a three dimensional bell shaped curve. The Z axis values estimate the probability of the individual visiting the location described by its corresponding X and Y coordinates. At the summit (largest z axis value) of the bell shaped curve, an individual is predicted to have full probability of visiting their own home.

The probability of visiting nearby sites is described by the top of the bell shaped curve. The use of the normal distribution results in the prediction that individuals perceive the difficulty of visiting two nearby sites, both within walking distance, nearly equally. The formula predicts a similar probability of visiting waste sites within 100 feet and 200 feet of one's residence. As distances increase, however, the probability of visiting a site declines at an accelerating rate, described by the steep part of the bell shaped curve. As the bell shaped curve reaches the tail, distances become far enough away that they are equally unattractive.

An example of the use of the 3-D normal distribution to predict probabilities of visiting a site would be to predict the locations where a given individual has a 50% chance of visiting. Place this individual on the map at the x and y coordinates of their home in Stratford. The z axis measures the probability that the person will visit the location described on the x and y axes. There is a circle of places around the maternal location that the mother is predicted to have a 50 % chance of visiting. This circle of locations is described by the x and y coordinates that have a z axis value of 0.5.

By setting the 'standard deviation' sigma value, and including the area of each waste site, the normal distribution can be used to predict the probability of finding an individual at a waste site using the location of their home. This probability, added up over all waste sites, is the cumulative probability of finding an individual at any of the waste sites. At each waste, one determines the average of the waste site's elevation from the normally distributed mountain centered on that mother's home.

Each residence has 39 average z values. This matrix of values is the model's prediction of the probabilities that each of the residents visited each of the 39 waste sites in a unit of time. To refine this model further, distance-decay values based on the site type were assigned σ. The standard deviation σ was defined as follows:

σ= 250 feet for commercial, residential sites, and the Housatonic Boat Club
σ= 500 feet for the Raymark facility
σ= 2000 feet for Wooster Park, Beacon Point Road, Raybestos Memorial Field
σ= 3000 feet for Short Beach Park.

These distance decay values were arbitrarily assigned based on our common-sense estimates. The practical meaning of the standard deviation value is that those properties with the smaller standard deviation values are likely to be visited by those living closer to the site, where as, those properties with larger standard deviation values are likely to draw visitors from a further distance. These 39 z values were summed for each of the residences, resulting in one cumulative exposure score per residence, according to the following formula:

$$Exposure = \sum_{f=1}^{39} area(f) \times \frac{1}{2\pi\sigma^2} \times e^{\left(\frac{1}{2}\right)\left(\frac{\left(\frac{X\{f\}-X\_Coord}{5280}\right)^2+\left(\frac{Y\{f\}-Y\_Coord}{5280}\right)^2}{\sigma}\right)}$$

where:

f = waste site #
a{f} = area of waste site f in square miles
X{f}= x coordinate of waste site f
Y{f} = y coordinate of waste site f
X_Coord = x coordinate of each residence
Y_Coord = y coordinate of each residence
σ= standard deviation (rate of distance decay)
250 feet for commercial, residential sites, and the Housatonic Boat Club
500 feet for the Raymark Facility
2000 feet for Wooster Park,Wooster School, Beacon Point Road,Raybestos Memorial Field
3000 feet for Short Beach Park

This equation conforms to a common-sense expectation of how the exposures distributed in the town, if exposure is determined by proximity.

Even if this model accurately predicts the probability of finding an individual at a remote site, this is a surrogate for actual exposure. The relationship between attractiveness, or probability of finding an individual at a site, and biological exposure is an estimate, and would need to be tested. Without exposure data, the equation is not intended to be a good-fitting model. Instead, the equation is proposed to further explore the use of continuous exposure measurements to estimate exposure, to identify the variables that may be important in relating distance to exposure, and to suggest a direction for future research.

E.) METHODS USED TO ANALYZE CANCER INCIDENCE

Time trend analysis of cancer incidence in Stratford in comparison to the State of Connecticut was conducted by calculating Comparative Morbidity Figures using computerized CTR data files for the period 1958-1991. Spatial analyses of individual cancer cases within Stratford were then conducted using manually extracted information from paper CTR records for the period 1968-1991. Spatial analyses included calculation of crude incidence rates per 1,000 by block group, and use of regression models to compute the odds ratios for the risk of cancer in exposed versus unexposed groups using the three exposure models described above.

The CTR collects data on individuals whom have been diagnosed with a tumor. There is no information on individuals who do not have such a diagnosis. The CTR, however, is a population based tumor registry meaning that all cases within the State must be reported to the CTR. This allows the use of Census data as the referent population for calculation of rates of disease and to control for the effects of age and gender in the analyses. The population data utilized for this project was extracted from the 1990 U.S. Census summary tape SFT3A by block group.

1.) Time Trend Analysis 1958-1991: Comparative Morbidity Figures for Stratford vs Connecticut

Age adjusted incidence rates for bladder cancer, testicular cancer, mesothelioma, and all cancers occurring to those under age 25 (EOC - early onset cancer) for males and females for Stratford in comparison to the State of Connecticut were computed. The age adjusted incidence rates were computed for seven time periods beginning in 1958 through 1991. The time periods consisted of six five year time periods and one four year time period. The four year time period was used because 1991 was the last full year of data available from CTR when the study began.

The direct method of standardization was utilized. Since a summary age and gender adjusted incidence rates for each of the seven time periods are going to be compared to each other, standardizing on a standard population will control for the effects of population changes and age distributions over time.[73] The U.S. 1990 Standard Million was used as the reference population for the direct standardization analysis. Breslow and Day recommend utilizing a published set of weights for direct standardization as it promotes comparability between series.[74] This enabled the analysis of trends in cancer incidence over time. Stratford's population has aged over time. It should be noted that the age adjusted incidence rates produced by this method is based on a fictitious population. The incidence rates are only for comparing cancer incidence in Stratford in relationship to the State of Connecticut in each time period and to assess trends. The incidence rates alone mean nothing.[75]

The tumor data were stratified into four age groups, <45, 45-64, 65-74, >74. Incidence rates computed using a direct method as described above are inappropriate to use when the cell numbers are small.[75] By using only four age groupings, this problem was addressed in advance. The direct standardized incidence rates for the State of Connecticut

and for the town of Stratford were compared by means of a comparative morbidity figure (CMF). The same four age groups were utilized whenever age was controlled for in the exposure models. For EOC, the data was not stratified into four age groups. It was maintained as one age group, < age 25.

The comparative morbidity figure (CMF) is used as a comparative measure of incidence.[74] In this study it provides a measure of the ratio of Stratford rates to Connecticut rates after they have been adjusted to control for the effects of age. The CMF was derived by dividing the age standardized incidence rate for Stratford by the age standardized incidence rate for Connecticut. Age was aggregated into four stratum. The formula for the CMF is

$$CMF = \frac{\sum_{j=1}^{4} w_j s_j / p_j}{\sum_{j=1}^{4} w_j c_j / t_j},$$

where w_j is the stratum specific population distribution weight from the 1990 U.S. Standard Million; s_j is the number of tumors in Stratford within the stratum; p_j is the Stratford population total for the stratum; c_j is the number of tumors in Connecticut within the stratum; t_j is the Connecticut population total for the stratum.

Confidence intervals were also constructed around the CMF from the standard error. Breslow and Day suggest transforming to the log scale to correct for skewness in the distribution of the CMF.[74] The formula for the confidence interval is

$$e^{(\ln(CMF) \pm 1.96(SE(\ln(CMF))))},$$

where

$$\text{SE(ln(CMF))} = \frac{\text{SE(CMF)}}{\text{CMF}}.$$

A CMF and 95 percent confidence intervals were calculated for each of the four tumor sites and seven time periods. The incidence ratios are used only for assessing trends and not as a statement of the cancer incidence in that town.

The CMF's give a crude indication of what is happening in Stratford as compared to Connecticut over time. This type of analysis does not evaluate exposure to the waste. The CMF's were stratified by gender except for mesothelioma due to small sample size. When examining CMF's it is important to remember that the number does not relate to actual incidence of disease. The CMF is the ratio between the age adjusted direct standardized incidence rates for Stratford and Connecticut.

Many CMF and confidence intervals were constructed (n=56). Therefore, it can be expected that some tests will obtain significance by chance. A Bonferrioni correction was not applied to the confidence intervals because the primary purpose of this analysis was to assess for time trends.

The test for trend was accomplished by utilizing a simple regression of the CMF for each time period on that time periods mid-point (1960, 1965, 1970, 1975, 1980, 1985, 1990).

2.) Spatial Analysis: Crude Incidence Rates per 1,000 by Block Group 1968-1991

The tumor data was analyzed by Census block group. The crude incidence rate of disease over the population of each block group per 1,000 for the twenty-four year study period was calculated. Because of the small numbers within each block group, the rates produced were not standardized on age or gender. The formula for the crude incidence rates is,

$$IR_{CRUDE} = \left(\frac{a}{a+b}\right)k,$$

where a equals the frequency with which the tumor has occurred during the study time period; (a + b) equals the population at risk for developing a tumor during the study time period; k equals 1,000.[76]

The crude rates are presented in Figure 3 Map of Mesothelioma and Testicular Cancer Incidence From 1968 - 1991, Stratford, Connecticut, Figure 4 , and Figure 3 and not in a table. They are presented to demonstrate the usefulness of GIS in analyzing health outcome data.

3.) Spatial Analysis: Odds Ratios using Logistic Regression Models 1968-1991

Regression models defining the association between exposure to Raymark waste and cancer were developed, using the CTR for case information and the census for the comparison group. The purpose of these models was to explore the possible association between exposure to Raymark waste and the incidence of bladder cancer, testicular cancer, mesothelioma, and all cancers occurring to persons less than 25 years of age or early onset cancer (EOC).

Residential location was reported only at the block group level in STF3A of the 1990 Census. The location of residence for Census data was assumed to be the centroid of a block group. There are 46 block groups in Stratford, and each resident was assigned one of the 46 possible block group centroids as the estimated location of their home. The distance from the centroid of each block group to the perimeter of the waste sites was calculated. For the individuals with cancer, the actual distance from their home to the waste site perimeters were calculated.

Data was not available on some of the important risk factors for cancer. For example, although occupational exposures to bladder carcinogens is an important risk factor, the CTR files were lacking in good occupational information. Most records had no occupation information at all. Smoking is another variable which should be considered, but CTR records were incomplete and/or inconsistent.

The effect of age and gender were controlled in the analysis of bladder cancer, with age categorized in four age groupings due to the evidence of a significant departure from linear trend. The effect of age was controlled in the EOC analysis with age maintained as a continuous variable. Because of very small sample sizes, age could not be controlled when examining mesothelioma or testicular cancers.

For the analysis of bladder cancer, age was categorized into four groups, <45, 45-64, 65-74, >74. This is the same age stratification utilized in the comparative morbidity figures, and these age groupings were selected to maintain consistency among the different analyses. Preliminary models were run comparing age as a continuous variable to the four age groups, and a test of whether there is a significant departure from linear trend was performed. The test statistic was calculated by subtracting the scaled deviance associated with the restricted model (without age or gender) from the scaled deviance associated with the full model (with age or gender included). Age was included as a categorical variable if results indicated a significant departure from linear trend. The analysis of bladder cancer was also stratified by gender.

When examining EOC, age was maintained as a continuous variable because there was no evidence of a departure from linear trend and all of the cases would fall in the category of <45.

Gender was also available for all cases, but was used as a control variable only for the bladder cancer analysis. Gender significantly improved the fit only for models predicting bladder cancer. EOC contains tumors of many different etiologic backgrounds, and its usefulness in an analysis of cancer is only as an indication of cancer trend among the young. Gender was also not controlled for in mesothelioma due to the small sample sizes.

Since several statistical comparisons were made it would be expected that some odds ratios would be elevated due to chance. Therefore, a Bonferroni correction was made to adjust for multiple statistical comparisons. The Bonferroni correction increases the confidence interval width, but does not change the odds ratio.[77] Analyses were performed in the Statistical Analysis System (SAS®) software utilizing the PROC LOGISTIC function.[78]

F.) METHODS USED TO ANALYZE BIRTHWEIGHT

1.) Spatial Analysis: Using Regression Models

There is a large body of literature establishing the association of numerous risk factors with the risk of having a low birthweight baby. These risk factors are described in Table 4. Regression models defining the association between these known risk factors and birthweight, LBW, and VLBW were developed, using the birth certificate data base. The purpose of these models is to explore the possible association between exposure to Raymark waste and birthweight, LBW, and VLBW after controlling for these known risk factors. The ability to control for the known risk factors of low birthweight is limited by numerous factors. Most of the limitations of this effort are a consequence of the use of the existing birth records data set for this study.

The birth certificate data base was reviewed for variables that might confound or modify a relationship between exposure and birthweight. The variables from the birth certificate that were reviewed include: maternal age, education, race, smoking status, weight, marital status, pregnancy interval, prior births, prenatal care, occupation, medical risk factors, previous fetal or infant death, and multiple births. These variables were re-

coded as necessary to maximize the statistical power of the analysis and the validity of each variable was considered. Simple linear models predicting birthweight were developed. Then multiple regression models predicting birthweight were built.

Variables that were used to control for confounding in this study were those that were recorded consistently on the birth certificate, and are strong predictors of birthweight. These variables were found to be child's race, marital status, and maternal age. These demographic variables are not causally associated with low birthweight in and of themselves. Instead they can be anticipated to be associated with other risk factors that are not measured on the birth certificate (or are incompletely measured). For example, marital status, age, and race could be associated with prenatal care during pregnancy, which in turn is a cause of adverse birth outcomes.

Several other variables may be important causal risk factors of low birthweight including smoking, maternal weight at delivery, the interval between births, parity, prenatal care, medical risk factors, and occupation. However, these variables were not included in the final model to preserve statistical power (they are missing for a large proportion of the data) or because they were correlated with other variables that were already in the final model.

The analyses were performed in SAS® utilizing the PROC LOGISTIC function.

IV. RESULTS

The cancer results are presented first, followed by the birthweight results. For cancer, the time trend analysis is presented first and utilized Comparative Morbidity Figures (CMFs) comparing Stratford to the State of Connecticut. The spatial analyses presents the cancer incidence rates by block group, and then two of the three exposure models (Block Group and Distance to Nearest Waste) are used to calculate the odds ratios for cancer risk within Stratford. The birthweight results are presented by each exposure model first with birthweight as a continuous variable and then as a categorical variable of low birthweight (LBW) and very low birthweight (VLBW).

A.) CANCER

Bladder cancer was analyzed controlling for gender and age. Due to the relatively small sample size, age was aggregated into four categories, <45, 45-64, 65-74, ≥75. Early onset cancers were analyzed controlling for age as a continuous variable. Mesothelioma and testicular cancer were analyzed in an uncontrolled model due to very small sample sizes.

1.) Time Trend Analysis 1958-1991: Comparative Morbidity Figures for Stratford vs. Connecticut

Comparative Morbidity Figures (CMFs) were computed for mesothelioma, bladder cancer, testicular cancer and EOC. As described earlier, each tumor site was stratified into seven time periods, six five year time periods and one four year time period. Table 5 shows the CMF and 95 percent confidence intervals for each site by gender and time period. Table 6 presents the number of cancer cases, for each time period by sex and tumor type.

In order to assess the CMF trend over time, a simple regression was performed for CMF on time. For all sites combined and bladder cancer the analysis stratified by gender. Because of small numbers, mesothelioma was analyzed by females and males combined. Testicular cancer was analyzed by males only. Table 7 presents the results of a test for trend over time.

There was no statistically significant trend for mesothelioma during the study period, $F(1,5)=3.20$ $p>F = 0.1338$.

The trend analysis for bladder cancer among males, and bladder cancer among males and females combined showed a statistically significant increase over time. The hypothesis of the trend line slope equaling zero is rejected for both $F(1,5)=19.63$ $p>F = 0.0068$ and $F(1,5)=12.82$ $p>F = 0.015$ respectively. There was no statistically significant trend for bladder cancer among females $F(1,5)=5.04$ $p>F = 0.075$.

There was no statistically significant trend for testicular cancer $F(1,5)=0.00$ $p>F = 0.962$.

The trend analysis for EOC: combined gender, females, and males failed to show a

statistically significant increase over time. The hypothesis of the trend line slope equaling zero is not rejected for all three EOC categories. The results were $F(1,5)=1.9$ $p>F = 0.226$, $F(1,5)=0.3$ $p>F = 0.8797$, and $F(1,5)=0.8$ $p>F = 0.4118$ respectively.

2.) Spatial Analysis: Crude Incidence Rates per 1,000 by Block Group 1968-1991

The crude incidence rates per 1,000 by Census block group were computed. Maps depicting these results are located at the end of this report. The maps contain block group crude incidence rates per 1,000 overlaid with the boundaries of block groups with Raymark waste highlighted in bright red. The incidence rates are for a 24 year time period.

Care should be taken when examining the block group incidence rates. These rates are obtained by applying a spatial filter (block group boundaries) to the cancer data. An implied significance between block groups, cancer incidence, and exposure may or may not exist.

a.) Mesothelioma

When examining mesothelioma block group incidence rates (Figure 3), no discernible spatial patterns exist. Out of the nine block groups with waste, seven have no mesothelioma cases reported over the last 24 years. Out of the 46 block groups in Stratford, only eleven have mesothelioma cases reported over the last 24 years. For mesothelioma, the block groups with waste most often have the same or lower rates as block groups without waste.

b.) Bladder cancer

Bladder cancer was examined by gender and combined males and females (Figure 4). For combined gender bladder cancer, the cases are distributed evenly throughout Stratford. Only three block groups have no bladder cancer cases (combined gender) reported in 24 years. The rates for block groups with and without waste are also similar.

When bladder cancer is examined by gender, a slightly different pattern presents itself. The block group rates for female bladder cancer for those block groups with cases reported is similar (.01-10 bladder cancer cases per 1,000) in all but one block group. Again all block groups with waste have female bladder cancer cases reported. For female bladder cancer, 14 block groups without waste had no cases reported. Male bladder cancer shows a more varied distribution among block groups with regard to the rates.

Block groups without waste show higher male bladder cancer rates than block groups with waste in all but two block groups Five block groups had no male bladder cancer cases reported in 24 years and all were waste free. Block group bladder cancer rates for males, females and combined gender demonstrate no discernible spatial patterns.

c.) Testicular cancer

When examining testicular cancer block group incidence rates (Figure 3), no discernible patterns exist. Out of the nine block groups with waste, seven have no testicular

cancer cases reported over the last 24 years. Out of the 46 block groups in Stratford, only fifteen have testicular cancer cases reported over the last 24 years. For testicular cancer, the block groups with waste most often have the same or lower rates as block groups without waste.

d.) Early Onset Cancers EOC (all sites less than age 25)

EOC combined gender block group rates are fairly evenly distributed across Stratford (Figure 5). Only seven block group, all without waste, reported no EOC cases in the 24 year study period. The pattern for female block group rates are similar. Twenty block groups reported no female EOC cases in the 24 year study period. Two of those had waste. The pattern for males is different. Of the nine block groups with waste, four of them reported no male EOC cases in the 24 year study period. Twice as many block groups with waste had no cases reported for males than females. Block group EOC rates for males, females and combined gender demonstrate no discernible patterns.

The south-eastern most block group in Stratford had the highest rate (10.51-14 per 1,000) of EOC for females and combined males and females. It also had the second highest rate (7.01-10.5 per 1,000) for males. Large portions of this block group contain wetlands and an airport. There are few locations in which people live. Because the population is small in this block group, a difference of one or two cases can dramatically increase the rate.

3.) Spatial Analysis: Odds Ratios using Logistic Regression Models 1968-1991

a.) Block Group Exposure Model

This model predicts the difference in cancer incidence between block groups with and without waste. The results are presented as the odds ratio (the odds of disease with exposure over the odds of disease without exposure). Table 8 presents the results of the cancer analyses. The results are shown for uncontrolled models and models controlling for age and gender where possible. Table 9 presents the results of the gender stratified bladder cancer analysis.

The odds ratio and 95% confidence interval for mesothelioma was 0.905 (0.109, 7.523). The odds ratio and 95% confidence interval for bladder cancer was 1.436 (0.996 2.072), and changed little after controlling for age and gender 1.417 (0.978, 2.051). When bladder cancer was stratified by gender while controlling for age, the odds ratios and 95% confidence interval for females and males were 1.960 (1.214, 3.167) and 1.257 (0.913, 1.731) respectively. The odds ratio and 95% confidence interval for testicular cancer was 0.754 (0.137, 4.149). The odds ratio and 95% confidence interval for EOC was 1.315 (0.706, 2.449) and increased slightly after controlling for gender to 1.387 (0.739, 2.572).

EOC did have an elevated odds ratio, although the odds ratio was not significantly different from unity. Bladder cancer also had an elevated odds ratio which was not significantly different from unity. However, when stratified by gender, the odds ratio for females was significantly different from unity while males was not.

b.) Distance to Nearest Site Exposure Model

This model predicts the difference in cancer incidence for each mile of distance to the nearest waste site. The results are presented as the odds ratio (the odds of disease for each mile closer to a waste site) and 95% confidence interval (Table 8).

The odds ratio for mesothelioma was 1.063 (0.369, 3.061). For bladder cancer the odds ratio was 1.93 (0.882, 1.354), and was greatly reduced after controlling for age and gender to 1.222 (0.986, 1.514). The odds ratio and 95% confidence interval for bladder cancer stratified by gender while controlling for age for females and males was 1.611 (1.118, 2.322) and 1.130 (0.953, 1.339) respectively (Table 9). The odds ratio for testicular cancer was 1.403 (0.522, 3.769). The odds ratio for EOC was 1.465 (0.940, 2.285) and after controlling for gender the odds ratio was 1.340 (0.845, 2.124). While statistical significance was only reached for female bladder cancer with the distance to the nearest waste site model, all of the tumor sites had odds ratios greater than one.

B.) BIRTHWEIGHT

The birthweight results are presented for birthweight as a continuous outcome and as a categorical outcome (low birthweight and very low birthweight). Using birthweight as a continuous variable provides substantial power to detect differences between exposed and unexposed individuals. One limitation of using birthweight as a continuous variable is that the distribution of birthweight is not the measure with health implications. LBW and VLBW babies are at increased risk for many adverse health outcomes, but this is not the case for babies with birthweights slightly below the mean. In addition to models that predict the distribution of birthweight as a continuous variable, models were constructed to predict LBW and VLBW. As a health outcome measure, continuous measurements of birthweight maximize the potential power to detect an association and avoids the problem of developing arbitrary cutoff points to establish categories. LBW and VLBW, on the other hand, are more direct measures of health outcomes. Table 10 presents mean birthweight LBW and VLBW by selected risk factors.

Each of the exposure models controlled for the effects of child's race, mother's age, and marital status. Figure 6 Map of Factors Associated With Birthweight From 1982 - 1991, Stratford, Connecticut presents these risk factors by census block group. However, the results are presented for both the uncontrolled and controlled models. This was accomplished to demonstrate the relationship the control variables had on the measure of effect.

1.) Block Group Exposure Model

a.) Birthweight: Continuous Outcome

The birthweights of the exposed and unexposed groups are significantly different. The birthweight of babies born to mothers living in the block groups with waste is higher. The mean birthweight and 95% confidence intervals around this mean are presented in Table 11. The mean birthweight by block groups are presented in Figure 7 Map of

Birthweight by 1990 Block Groups From 1982 - 1991, Stratford, Connecticut.

The results of regression analysis using birthweight as a continuous outcome are presented in Table 12 . The F value for the one variable regression model predicting birthweight from the exposure category was 5.080 with a Prob.>F = 0.024. The R-Square for this model was 0.001. The model equation is: Birthweight=3411.36 + 43.90(exposed). This model suggests that the birthweight of those living within exposed block groups is, on average, 43.9 grams higher than the birthweight of Stratford residents living outside the exposed block groups. When controlling for child's race, mother's age, and martial status, the parameter estimate of the exposure variable decreased from 43.9 grams to 24.27 grams. The difference is not significantly different from zero (T=1.25 (Prob.>|T|)=0.21).

b.) Birthweight: Categorical Outcome (LBW - VLBW)

The difference in the odds of LBW and VLBW between block groups with and without waste is predicted using a logistic regression model. The results are presented as the odds ratio (the odds of disease with exposure over the odds of disease without exposure). Table 13 presents the results of the categorical birthweight analyses. Figure 7 presents birthweight (LBW and VLBW) by block group.

The odds ratio and 95% confidence interval for LBW and VLBW for the uncontrolled model are 0.80 (0.56, 1.14) and 0.58 (0.23, 1.47) respectively. For the controlled model, the odds ratio and 95% confidence interval for LBW and VLBW are 0.92 (0.64, 1.32) and 0.73 (0.29, 1.92) respectively. Neither model (controlled or uncontrolled) achieved statistical significance.

2.) Distance to Nearest Waste Site Exposure Model

This model predicts the difference in health outcome by miles of distance to the nearest waste site. The results are presented as the odds ratio (the odds of disease for each mile closer to a waste site). The birthweight results are presented for birthweight as a continuous outcome (Table 12) and as a categorical outcome (low birthweight and very low birthweight (Table 13). The results are shown for an uncontrolled model and for a model controlling for child's race, maternal age, and marital status.

a.) Birthweight: Continuous Outcome

In a linear model predicting birthweight from the distance from the nearest site, there is no association (F Value=0.19, Prob. > |F| = .660, R-Square = 0.000). The model equation is: Birthweight=3405.27 + 4.8(distance). The model predicts that the birthweight of the baby would be 4.8 grams heavier for each mile of distance closer to a waste site. For the controlled model, the parameter estimate of the exposure variable increased from 4.8 grams to 10.5 grams heavier for each mile of distance closer to a waste site. The difference is not significantly different from zero (T=0.97 (Prob.>|T|)=0.33).

b.) Birthweight: Categorical Outcome (LBW - VLBW)

The odds ratio and 95% confidence interval for low birthweight and very low birthweight for the uncontrolled model are 1.01 (0.84, 1.22) and 1.27 (0.74, 2.16) respectively. For the controlled model, the odds ratio and 95% confidence interval for LBW and VLBW are 0.97 (0.78, 1.21) and 1.18 (0.68, 2.03) respectively. Neither model (controlled or uncontrolled) achieved statistical significance.

3.) Cumulative Exposure Model

This model predicts the differences in health outcome based on the cumulative probability of exposure to all the waste sites. The results are presented as the odds ratio (the odds of disease for the most exposed over the least exposed). The birthweight results are presented for birthweight as a continuous outcome (Table 12) and as a categorical outcome (low birthweight and very low birthweight (Table 13). The results are shown for a controlled and uncontrolled model.

a.) Birthweight: Continuous Outcome

A one variable linear regression model predicting birthweight from the percentile ranks of the exposure score was not significant (R-Square= 0.000, F=1.38, Pr > F = 0.239. The model is Birthweight=3404.16 + 31.43(exposure). Once race, mothers age, and marital status are controlled, the beta value for the exposure score increases from 31.43 to 44.44 grams. The difference is not significantly different from zero (T=1/68 (Prob.>|T|)=0.09).

b.) Birthweight: Categorical Outcome (LBW - VLBW)

The odds ratio and 95% confidence interval for low birthweight and very low birthweight for the uncontrolled model are 0.69 (0.44, 1.10) and 0.99 (0.34, 2.90) respectively. For the controlled model, the odds ratio and 95% confidence interval for LBW and VLBW are 0.62 (0.39, 0.99) and 0.79 (0.25, 2.46) respectively. Neither model (controlled or uncontrolled) achieved statistical significance. Table 14 presents LBW, VLBW and other reproductive characteristics according to distance to the nearest waste site and in the total population of Stratford.

V. DISCUSSION

A.) CANCER

The results from these analyses do not support the hypothesis that living near Raymark waste resulted in an increased risk of developing mesothelioma or testicular cancer. The results do somewhat support the hypothesis that living near Raymark waste is associated with an increased risk of developing bladder cancer or EOC. The results of the analyses are discussed with specific reference to the exposure models utilized and are summarized in Table 8 and Table 9.

Public health officials must respond when a community is concerned that a particular industry or waste site is causing an excess incidence of cancer. It is unlikely that the findings from a cluster investigation of small numbers of individuals exposed to low levels of ambient carcinogens will prove to be statistically significant unless there is a great increase in risk. The long latency period and infrequent incidence of most cancers make epidemiological investigations of cancer particularly difficult. This difficulty is compounded when financial and time constraints limit the investigation to an analysis of existing data sets, which often contain incomplete information regarding the exposure history as well as important confounders.

1.) Time Trend Analysis 1958-1991: Comparative Morbidity Figures for Stratford vs. Connecticut

The CMF's give a crude indication of what is happening in Stratford as compared to Connecticut over time. This analysis does not take into account exposure to the waste. The CMF's were stratified by gender except for mesothelioma due to small sample size. When examining CMF's it is important to remember that the number does not relate to actual incidence of disease. The CMF is the ratio between the age adjusted direct standardized incidence rates for Stratford and Connecticut.

Since the etiology of cancer differs with each site, grouping them together in an analysis (EOC) produces results that are difficult to interpret. Citizens' concerns drove the analysis of EOC so the results need to be reviewed with that purpose in mind. There was no significantly increasing trend for EOC: combined gender, females, and males over the seven time periods.

The trend analysis for bladder cancer combined gender and for males, showed a significantly increasing trend over the seven time periods. Female bladder cancer did not show a significant increasing trend.

Because of the nature of the exposure to the waste, there is no reason to believe that Stratford residents would have a greater exposure as time progressed. Waste was deposited over a fairly long period of time, with some waste being at the surface and then covered over many years later. However, the increasing rates of cancer may reflect latency, effects of cumulative exposures over time, other cumulative environmental exposures, or other risk

factors. The increasing trend might also be due to chance alone.

No significant test for trend was found for mesothelioma. The mesothelioma CMF's rise and fall with no consistencies. The trend analysis was also not significant for testicular cancer, however, there does appear to be a general increase over time. The number of testicular cancer cases from 1958 - 1991 is relatively small (n=21), and therefore, there is little statistical power to detect an association.

Care needs to be taken when interpreting the CMF results. Because so many CMF and confidence intervals were constructed (n=56), the concern over multiple-testing arises. With this many statistical tests being performed, it can be expected that some tests will obtain significance by chance.

2.) Spatial Analysis: 1968-1991

a.) Mesothelioma

None of the exposure models identified an increased risk of developing mesothelioma nor was there any discernible spatial pattern in the distribution of mesothelioma cases throughout Stratford over the entire 24 year period. There was no trend in the incidence of mesothelioma over time.

Although mesothelioma is a rare cancer, it was somewhat surprising that there were only 12 cases of mesothelioma in Stratford during the study period recognizing the occupational exposures probably are associated with the use of asbestos at Raymark. Some workers at Raymark were likely to have been occupationally exposed to asbestos and to have lived in Stratford.

The exposure models applied in this study were designed to assess environmental exposures. These models would not have assisted in identifying any increased risk to workers at the facility because worker exposure to asbestos would not be associated with their residential proximity to the waste sites. Unfortunately, Tumor Registry records do not provide complete information regarding occupation making it impossible to further explore the occupational aspects of risk and disease.

Conversations with the director of the Tumor Registry indicated diagnostic techniques were less definitive early in the study period and cases of mesothelioma may have gone unrecognized in Stratford and statewide.

b.) Bladder Cancer

The block group and distance to the nearest waste site exposure models indicated an increased risk for bladder cancer for men and women combined, although the odds ratios did not achieve statistical significance.

To explore further the increase in incidence of bladder cancer, the analyses were stratified by gender. The odds for bladder cancer was slightly higher and did achieve statistical significance among females but not males using both the block group and distance to the nearest waste site models (Table 9). The higher incidence of bladder cancer among women resulted primarily from two factors: a high incidence of bladder

cancer in women living in the exposed block group containing the Housatonic Boat Club, and the absence of bladder cancer in the southwest section of Stratford (see Figure 4 Map of Bladder Cancer Incidence From 1968 - 1991, Stratford, Connecticut).

Although the geographic pattern of bladder cancer in Stratford is of concern, one must be cautious not to infer from these results any suggestion of a causal relationship with ambient exposures to the Raymark waste. Other potential risk factors could result in a geographic distribution of the disease coincidental with the distribution of the Raymark waste sites. An individual's potential occupational exposures to bladder carcinogens, their environmental exposures from sources other than Raymark waste, their smoking and dietary history, the medications they have taken, and genetic factors must be considered as other potential explanations for the geographic pattern of bladder cancer found among women in Stratford.

The Tumor Registry records do not have complete information on bladder cancer risk factors including smoking or occupation. These factors may have influenced risk but cannot be considered in the analysis. The analysis stratified by gender indicated that the odds of bladder cancer were significantly elevated among women, but not among men. Women may be less likely to have worked with potential bladder carcinogens in an occupational setting, and the results of the analysis of women may be subject to less occupational confounding. This finding of increased odds of bladder cancer among women closer to the Raymark waste sites is suggestive of an association. However, one can not ascertain from this study if the association was the result of ambient exposure to the waste, or from some other factor with a similar geographic distribution as the waste sites.

The trend analysis for bladder cancer combined gender and for males only, showed a significantly increasing trend over the seven time periods. Female bladder cancer did not show a significantly increasing trend. The female bladder cancer CMFs rise and fall with no consistencies.

c.) Testicular cancer

The preliminary analysis in an earlier study indicated that the risk of testicular cancer was lower in Stratford than in the state, however, residents of Stratford requested that testicular cancer be included in this study. Because testicular cancer is typically diagnosed in young men, there appears to be heightened awareness and concern among the general population regarding this cancer.

While the block group exposure model indicated a reduced risk for testicular cancer, the risk for testicular cancer increased in the distance to the nearest waste site exposure model. Neither of these odds ratios achieved statistical significance. The incidence maps indicated no discernible pattern of cases near the waste sites. Because there were few cases, the statistical power of this analysis is very limited.

Much of the etiology of testicular cancer is unknown and even less is known about environmental risk factors. Because the age distribution supports a hypothesis that exposure to risk factors in utero, or early in life are more important than exposure to risk factors in adulthood, the use of residential address at time of diagnosis may not be indicative of the most critical exposure, assuming that exposure is associated with

development of testicular cancer.

d.) Early Onset Cancers

As with testicular cancer, citizen concerns drove the analysis of early onset cancers. Because the etiology of cancer differs for each tumor site, grouping all early onset cancers together produces results that are difficult to interpret with respect to environmental exposure.

While there was a slight elevation in the risk of EOC for both exposure models, neither of the odds ratios achieved statistical significance. The incidence maps indicate no geographic pattern in the incidence of EOC. The block groups in the southeastern portion of the town did have higher rates of EOC. This block group contains the airport, swamp areas, and commercial property. The population is low and the rates can be affected by just a few cases.

B.) BIRTHWEIGHT

Table 12 is a summary of the relationship between the exposure models and birthweight as a continuous variable. Table 13 presents the relationship between these distance models and LBW and VLBW. No significant associations with exposure were detected in any of these models. With the exception of distance from the nearest site and VLBW, exposure status predicted higher birthweight. This is likely a result of residual confounding because the demographic factors associated with LBW were not concentrated in the areas with highest potential exposure.

The concentration of demographic factors associated with lower birthweight is demonstrated in Figure 6 Map of Factors Associated With Birthweight From 1982 - 1991, Stratford, Connecticut. Three demographic factors strongly associated with lower birthweight are presented by block group. These demographic factors exhibit a spatial pattern that is strikingly similar to that of VLBW. Block groups in the south-western side of Stratford contain the highest percentages of the three demographic factors (unmarried, black race, and mothers < age 21). The levels of these variables associated with lower birthweights are concentrated in the southwestern section of the town, as opposed to the waste sites located along the Housatonic River on the eastern side.

Efforts to include many of the potential confounding variables recorded on the birth certificate were limited by missing information. During the late 1980s (1988 in Connecticut), birth certificates were revised nationwide to include many more variables than had previously been recorded. This period of transition may have contributed to the limitations in data validity. Responses to questions about maternal smoking and alcohol consumption, maternal weight, and medical risk factors were incomplete, and nonresponses were associated with adverse birth outcomes. The confounders that were included in the final models are significantly associated with birthweight, however the low R-square values in models predicting birthweight demonstrate that these factors predict a very small percentage of the variability in birthweight. The addition of other potential confounders with more limited validity from the birth certificate did not influence the results, nor result in models that were more predictive of birthweight.

These data suggest that the host of environmental and genetic factors determining birthweight are probably inadequately measured or are not measured at all on the birth certificate.

The results of these analyses do not support the hypothesis that there is an association between birthweight and exposure to Raymark waste. The suggestion in this study of a lack of an association, on the other hand, must be interpreted with caution due to the limited control of confounding and exposure misclassification.

The lack of associations between measures of proximity to the Raymark waste sites and birthweight were consistent. One can not refute the possibility that incomplete control for confounding overwhelmed these analyses, or that the power was not sufficient to detect an association (Type II error). The suggestion in this study of a lack of an association must therefore be interpreted with caution. One cannot rule out the potential for small associations between exposure to hazardous waste in soil and LBW in Stratford that might have gone undetected.

Consistent with a vast body of literature, the birthweight study did present overwhelming evidence of the geographic concentration of LBW in specific minority neighborhoods. There is a public health imperative to address the causes of LBW that resulted in such a large variability in LBW between neighborhoods. Children coded as black race on the birth certificate were almost 2 and a half times more likely to be LBW than white race children in Stratford. These findings underscore the alarming national trend of increasing ratios of LBW infants among black infants compared with that among white infants. The nationwide odds ratio of 2.3 in 1991[79] is comparable to our finding of 2.4 in Stratford. Although infant mortality has been declining steadily, the differences in the infant mortality, LBW, and VLBW rates between blacks and whites have steadily increased and are projected to be threefold by the year 2000.[79] The disparity in the risk of LBW by race can be attributed to a variety of clinical conditions[80], presumably resulting from the social inequities that result in differences in maternal health status.[81] There is a growing need for the public health community to continue to investigate and to remedy the causes of the strong associations detected between birthweight and race, maternal age, marital status, education, and other demographic and reproductive variables.

C.) EXPOSURE ASSESSMENT

Ecological studies often rely on population based estimates of exposure. Studies utilizing GIS sometimes compare political subdivisions (e.g. counties) or create "buffers" of geographic areas defined as exposed (e.g. within one mile of a site). This study improved upon those methods by comparing three exposure models, each refining the estimation of exposure. However, each of these models shared an important weakness by relying on the use of a single residential location and its proximity to waste in the estimate of exposure. Actual exposure to the Raymark waste is primarily dependent on physical contact with the soil. In addition, occupational exposures are not appropriately recognized or characterized based on residential location. Exposure misclassification

may occur using these exposure models because proximity to waste may not provide an accurate estimate of actual exposure.

1.) Block Group Model

The block group exposure model provided a categorical assignment of exposed and unexposed based on whether or not individuals lived in block groups with waste. While block groups were used because of their relatively small size and the availability of population or denominator data from the US Census there are many opportunities for misclassification in this first modeling effort.

Since block groups often use streets as boundaries, people living on different sides of the same street could receive a different exposure classification even though the probability of exposure could be the same. Within a block group, the probability of exposure could be different based on proximity despite the fact that everyone within the same block group received the same exposure assignment. Because an exposed classification was given to any block group containing waste, regardless of the area or type of waste site, the probability of true exposure in one "exposed" block group could be different than the probability of true exposure in another "exposed" block group.

Exposure misclassification can also occur during the geocoding process if a case is geocoded to the wrong block group. Since the misclassification is independent of the other study variables, this is considered nondifferential misclassification. This biases the results towards the null, and may underestimate the effect.[73] However, in this study, it was estimated that only 0.6 % of the subjects would have been assigned to the wrong block group.

2.) Distance to Nearest Waste Site Model

Because this model provides a continuous measure of distance as an estimate of exposure it refines the earlier categorical assignment provided by the block group model. Despite this improvement, this model also presents a number of opportunities for misclassification.

As with the block group model, this model treats all the waste sites equally with regard to area and site type. A large waste site may present a higher probability of exposure than a smaller site, however, two residences located equidistant from each of these two different sized sites would receive the same "exposure" classification. In addition, a recreational site may present a higher probability of exposure versus a commercial or industrial site but would be treated the same in the distance to nearest site model.

In this model, differential misclassification was introduced to the cancer analysis because of the absence of individual control data. Control data from the census were assigned the centroid of the block group for their location (latitude and longitude). Since there are 46 block groups in Stratford, this placed 49,353 people at 46 locations. This generalization of locations introduced differential misclassification to the cancer analysis. Any misclassification of exposure based on the distance measurements affected only the

controls. The birthweight data contains all births, therefore, this bias only pertains to the cancer analysis.

3.) Cumulative Exposure Model

In addition to producing a continuous estimate of exposure based on distance, the cumulative exposure model incorporated a number of factors that likely affect the probability of exposure. These factors included the area of each of the sites and the type of site (such as residential, recreational, or commercial property). In addition, this model is not based on the nearest site but provides a cumulative exposure "score" that includes all the sites. By incorporating these factors the probability of actually coming in contact with the waste becomes more refined. For example if a person lived equidistant from a recreational and a commercial property, they would be more likely to spend time at the recreational property. A major limitation of this approach is the lack of empirical data to support some of the assumptions used in the model. For instance, we do not know how much more appealing a recreational site is versus a commercial site. In addition, this model again relies on residential proximity and would not take into account an individual who works at a commercial property.

As discussed previously, modeling the risk of exposure to contamination in soil presents a number of difficulties. Unlike exposure to drinking water or ambient air, exposure to soil is contingent upon an individual's actions with respect to their interaction with the media. The models applied in this study refine the assessment of exposure to contamination in soil by incorporating factors that likely affect an individual's interaction with a site. Because the cumulative exposure model was flexible and allowed for the incorporation of various site characteristics, it could be applied to studies of health outcomes and environmental exposures, including the potential for exposure to multiple sources of environmental contaminants.

D.) USE OF EXISTING DATA SOURCES

An objective of this work was to utilize the existing data sources at the Connecticut Department of Public Health. While birth records and tumor records were complete and accurate in many respects, these data were not collected specifically for this study. Information concerning many potential risk factors for low birthweight and cancer were not available for analysis (including specific information on exposure to the Raymark waste).

E.) LATENCY BETWEEN EXPOSURE & DISEASE

The latency period between exposure and disease onset for the cancer cases can be ten to twenty years or longer. Because of this long latency period, issues such as estimation of exposure based on one residential address become particularly problematic. This is less of a consideration for the birthweight data as the latency period from conception to birth is nine months.

F.) MOVEMENT OVER TIME

A major limitation of both the cancer and birthweight analysis is the inability to track the movement of cases over time. An assumption is made that all the cases lived in the same house for the entire study period. If a mother relocated during her pregnancy or a cancer patient moved prior to diagnosis, the address reported on the birth certificate or medical record would not reflect where the individual lived during the relevant exposure period.

The birth certificate is completed at the end of pregnancy, while the important exposure may have occurred early in the pregnancy. Schulman et al. reported that substantial bias can result from exposure misclassification due to residential relocation during pregnancy.[82] Bias becomes a problem if mothers exposed early in pregnancy moved away from the exposed areas or mothers from unexposed areas moved into the exposed areas late in pregnancy.

As long as the movement occurs equally in both groups, exposed moving away from exposed areas and non-exposed moving into exposed areas, the bias will be non-differential. This has the effect of moving the point estimate towards the null. If movement is different between groups, more people move in than out or more people move out than in, the bias will be differential. This will have an unpredictable effect on the point estimate.

After 1988, Connecticut birth certificates contained information on how long mothers lived at their current address. A high percentage of mothers reported living at their current address one year or less. Of the 2,859 Stratford residents who responded to the question, 31.2% said they were at their current address for one year or less. With over 30% moving during pregnancy, using the address at the time of birth could introduce a considerable amount of misclassification.

The mobility of the population over time is even more of a concern with respect to the cancer analysis due to the long latency between exposure and tumor diagnosis. The cancer study rests on the assumption that the location of the individual at the time of cancer diagnosis is the address they lived at decades earlier when relevant exposures may have occurred.

Information regarding residential history on cancer cases was not available from the CTR records. A few cases had residential information if they moved and continued to receive treatment for their cancer. However, the address change only represented the address at the time medical care was received and not when the case moved.

When the mother's address or address at cancer diagnosis is used to approximate exposure, residential mobility becomes an important limitation. In future ecological studies that use existing datasets, an attempt needs to be made to obtain the information on residence history.

G.) USE OF 1990 CENSUS DATA AS CONTROLS

The Tumor Registry is a population based registry which provides demographic and disease data on the cases, but not on the comparison population. The comparison group for the cancer analysis was the 1990 census population. Since the population aged over the study period (see Table 3) the use of the 1990 census data could underestimate the odds ratios for the cancers that tend to occur later in life (bladder and mesothelioma) and overestimate the odds ratios for the cancers that tend to occur earlier in life (EOC and testicular).

H.) OCCUPATIONAL EXPOSURE

The tumor registry database did not include the occupational history of the patient in a consistent manner for the duration of the study period, and this important confounding factor could not be controlled. Similarly, the birth certificate did not record maternal occupation for the duration of the study period and, due to the large number for whom information was missing, adequate control of occupational confounding was not possible.

Starting in 1988, birth certificates began to include information on the parents' occupations and places of work. Maternal job classifications were associated with socioeconomic variables, which in turn are associated with birthweight. For example, the mother's reporting jobs in manufacturing occupations was associated with the child's race (chi-square = 20.7 [$p = 0.002$]), maternal age (chi-square = 59.8 [$p < 0.001$]), and marital status (chi-square = 110.1 [$p < 0.001$]). Further analysis of occupational associations with birthweight will therefore require adequate control of other confounding.

I.) USE OF GIS

This study demonstrated that existing health outcome data could be accurately geocoded and used in a GIS. In this study the GIS was used to geocode data, measure distances between exposures and residential locations and to produce maps of the waste locations and study results. The high quality residential address information in the health outcome database and the referencing database are crucial in obtaining high rates of geocoding.

Geographic Information Systems should continue to be used in conducting epidemiological investigations. There are many applications of GIS including: geocoding of health outcome data, graphic display of data, spatially relating health outcome data with other data including census information and environmental data, and the development of environmental exposure models.

Possible application of GIS technology should be considered as health outcome and environmental databases are developed. Accurate address information in the proper format is necessary to enable a GIS to geocode the information. Geocoded health outcome data (tumor, lead poisoning, birthweight, and birth defects) would allow for

faster implementation of health studies. Environmental exposure databases should be developed on a state-wide basis utilizing various sources such as the Point Source Inventory (PSI), Leaking Underground Storage Tanks (LUST), Toxic Release Inventory (TRI), as well as others. These data then could be incorporated in environmental exposure assessments.

GIS can be used to assist in the development of methods to construct estimates of populations. While the census provides very useful information, sometimes additional information is necessary. GIS can utilize existing spatial data layers such as land use/land cover or roads to provide more precise estimates of population density within a Census designated area such as the block group. Housing locations and populations can then be assigned within these areas. A digital phone book can also be used to provide locations of houses. These methods would be useful in ecologically based cancer analyses which do not have geocoded referent populations.

State health departments need to use standardized protocols for the investigations of health outcome data in relation to potential exposure to hazardous waste. These protocols should include the delineation of a known exposure pathway, biological plausibility between exposure and development of disease, existence of quality health outcome data, and sufficient statistical power to be able to demonstrate an effect.

VI. CONCLUSIONS

1. The time trend analysis did not indicate any increasing incidence of mesothelioma, female bladder cancer, testicular cancer, or early onset cancer (EOC). Bladder cancer combined gender and for males showed a significantly increasing trend over the study period. This increasing trend is a town wide analysis and is not associated with exposure to waste.
2. Mesothelioma and testicular cancer were not shown to be consistently associated with exposure to Raymark waste as estimated by exposure models based on residential proximity to waste sites. However, small sample sizes render the findings inconclusive, particularly since mesothelioma and testicular cancer are rare.
3. Bladder cancer was slightly elevated among those who lived closer to Raymark waste. The elevation was greater and statistically significant for women. The maps of the incidence rates by block group for men do not indicate that cancer rates were highest in the block groups believed to have the highest likelihood for exposure. Factors other than, or in addition to, geographic proximity to the Raymark waste sites could explain the elevated odds ratios for women.
4. Early onset cancer (EOC), cancer occurring to those less than 25 years of age, had a slight, non-statistically significant increase with increasing proximity to Raymark waste. Changes in the population demographics during the 24 year time period of the investigation may be a partial explanation of the EOC cancer findings.
5. Birthweight, low birthweight, or very low birthweight were not shown to be associated with exposure to Raymark waste as estimated by exposure models based on residential proximity to waste sites.
6. On a population basis, this study did not demonstrate an association between exposure and birthweight low birthweight, very low birthweight, mesothelioma, or testicular cancer. However, epidemiologic methods, especially ecological studies, cannot be used to derive conclusions regarding individual risk. Therefore, these results do not rule out the potential that an individual's health was adversely affected by exposure to Raymark waste.
7. The Geographic Information System (GIS) was successful in enhancing the ability to conduct epidemiological investigations. The address information was of sufficient quality to enable accurate geocoding of the health outcome data. The GIS calculated distances between points, which was a necessary component of the exposure models. Also, the GIS enabled graphic display of data to aid in the interpretation of results.
8. Mapping of low birthweight risk factor data present on the birth certificate proved useful for this study as well as the local health director in Stratford in targeting public health programs for teen age mothers. The mapped birth data can be used for public health planning purposes, targeting intervention activities, and focusing public health educational activities.

VII. RECOMMENDATIONS

1.) Educational programs need to be conducted involving DPH, the Stratford Health Department, citizens of Stratford, and other interested individuals to discuss the results and limitations of this study.

2.) These results do not suggest the need to conduct further ecological epidemiological investigations of the possible association between mesothelioma, testicular cancer, or birthweight and exposure to Raymark waste.

3.) Other researchers should consider environmental exposures in investigations of the causes of bladder and early onset cancer.

4.) The cumulative exposure model that was developed for use in this study should be tested in other studies involving multiple sources of environmental exposure.

VIII. TABLES

TABLE 1 RAYMARK INDUSTRIES, INC. ON-SITE SOIL CONTAMINATION LEVELS 49
TABLE 2 CANCER INCIDENCE IN STRATFORD IN COMPARISON TO CONNECTICUT FROM 1958 - 1991 50
TABLE 3 STRATFORD, CONNECTICUT CENSUS POPULATION SUMMARY FOR 1970, 1980, 1990 BY GENDER AND AGE 51
TABLE 4 RISK FACTORS ASSOCIATED WITH LOW BIRTHWEIGHT, 1982 - 1991, STRATFORD, CONNECTICUT 52
TABLE 5 COMPARATIVE MORBIDITY FIGURES (CMF) FOR STRATFORD AS COMPARED TO CONNECTICUT AND 95% CONFIDENCE INTERVALS BY TUMOR SITE, TIME PERIOD, AND GENDER, 1958 - 1991 53
TABLE 6 SUMMARY OF DATA UTILIZED IN THE CANCER TREND ANALYSIS, STRATFORD, CONNECTICUT, 1958 - 1991 54
TABLE 7 TEST OF TREND OVER TIME OF THE COMPARATIVE MORBIDITY FIGURES, 1958 - 1991 60
TABLE 8 CANCER ODDS RATIOS AND 95% CONFIDENCE INTERVALS BY TUMOR, EXPOSURE MODEL, AND CONTROLS FROM 1968 - 1991, STRATFORD, CONNECTICUT 61
TABLE 9 ODDS RATIOS (OR) AND 95% CONFIDENCE INTERVALS FOR BLADDER CANCER FOR TWO EXPOSURE MODELS BY GENDER, 1968 - 1991, STRATFORD, CONNECTICUT 62
TABLE 10 MEAN BIRTHWEIGHT, LBW, AND VLBW ACCORDING TO SELECTED RISK FACTORS, 1982 - 1991, STRATFORD, CONNECTICUT 63
TABLE 11 DISTRIBUTION OF BIRTHS AND MEAN BIRTHWEIGHT AND 95% CONFIDENCE INTERVALS WITHIN BLOCK GROUP EXPOSURE CATEGORIES, 1982 - 1991, STRATFORD, CONNECTICUT 64
TABLE 12 BIRTHWEIGHT REGRESSION ANALYSIS RESULTS - BIRTHWEIGHT AS A CONTINUOUS OUTCOME, 1982 - 1991, STRATFORD, CONNECTICUT 65
TABLE 13 BIRTHWEIGHT ANALYSIS RESULTS - BIRTHWEIGHT AS A CATEGORICAL OUTCOME (LBW-VLBW), 1982 - 1991, STRATFORD, CONNECTICUT 66
TABLE 14 DEMOGRAPHIC AND REPRODUCTIVE CHARACTERISTICS AMONG THE STUDY POPULATION IN STRATFORD, CONNECTICUT, 1982-1991 67

Table 1 Raymark Industries, Inc. On-Site Soil Contamination Levels

Classification	Compound	Concentration Range (in ppm* unless noted)
	asbestos	2-25%
Metals		
	copper	4.6 - 56,900
	lead	1.7 - 52,700
PCBs		
	aroclor 1262	.015 - 4,000
	aroclor 1268	.0096 - 6,400
VOCs		
	1,1,1 trichloroethane	.005 - 120
	1,2 dichloroethene	.002 - 240
	2-butanone	.007 - 280
	carbon tetrachloride	6.2
	tetrachloroethane	.002 - 15
	trichloroethene	.001 - 3,500
Semi-volatile Organics		
	2-methylnaphthalene	.096 - 75
	benzo(a)pyrene	.80 - 1.8
	benzo(a)anthracene	.75 -24
	benzo(b)fluoranthene	.069 - 20
	benzo(g,h,i)perylene	.067 - 6.7
	benzo(k)fluoranthene	.078 - 11
	bis (2-ethylhexyl) phthalate	.070 - 24
	chrysene	.078 - 21
	di-n-butylphthalate	.072 - 300
	dibenzo(a,h)anthracene	.93 - 5
	dibenzofuran	.068 - 18
	fluoranthene	.072 - 48
	fluorene	.071 - 12
	naphthalene	.079 - 49
	pentachlorophenol	.1 - 6.6
	m & p cresol	.070 - 9,600
Dioxins		
	dioxin TEF†	0 - .0413

* ppm-parts per million
†TEF-Toxic Equivalency Factor

Table 2 Cancer Incidence in Stratford in Comparison to Connecticut from 1958 - 1991

TUMOR SITE	NUMBER OF CASES OBSERVED/EXPECTED	AGE STANDARDIZED INCIDENCE RATIO*	95 % CONFIDENCE INTERVAL
ALL SITES	7467 / 7394	1.01	(0.99, 1.03)
ALL SITES LESS THAN AGE 25	130 / 106.56	1.22	(1.01, 1.42)
BLADDER	379 / 332.3	1.14	(1.03, 1.26)
BRAIN	108 / 108.2	1.00	(0.81, 1.19)
BREAST	1036 / 1084	0.96	(0.90, 1.01)
KIDNEY	179 / 172.4	1.04	(0.89, 1.19)
LEUKEMIA	201 / 203.7	0.99	(0.85, 1.12)
LIVER	43 / 45.75	0.94	(0.66, 1.22)
LUNG	1031 / 1017	1.01	(0.95, 1.08)
MESOTHELIOMA	16 / 11.35	1.41	(0.72, 2.10)
NON-HODGKIN LYMPHOMA	218 / 217.7	1.00	(0.87, 1.13)
RECTUM	411 / 385.8	1.07	(0.96, 1.17)
SOFT TISSUE SARCOMA	89 / 90.2	0.99	(0.78, 1.19)
TESTICULAR	23 / 29	0.79	(0.47, 1.12)

* The standard incidence ratio (SIR) is an overall summary measure of the cancer risk. The actual (or observed) number of cancers identified by the Tumor Registry are divided by the expected number to obtain the SIR. When the SIR is less than one (1.00) the risk of cancer is less than expected, when the SIR is greater than one the risk is more than expected. Since the SIR may be based on small numbers when studying rare cancers the 95% confidence interval gives the probable range of the SIR. When the range does not include the number 1.00 then the results are considered statistically significant.

Table 3 Stratford, Connecticut Census Population Summary For 1970, 1980, 1990 by Gender and Age

Census Year	Age Catagories	Females	Males	Total Population
1970	<25	9955	10111	
	<45	15692	15446	
	45-64	7166	6575	
	65-74	1713	1290	
	>=75	1159	734	
	Total	25730	24045	49775
1980	<25	8221	8418	
	<45	14584	14306	
	45-64	7450	6526	
	65-74	2873	2219	
	>=75	1724	859	
	Total	26631	23910	50541
1990	<25	6948	7151	
	<45	14440	14524	
	45-64	5721	4994	
	65-74	3395	2468	
	>=75	2449	1398	
	Total	26005	23384	49389

Table 4 Risk Factors Associated with Low birthweight ,* 1982-1991, Stratford, Connecticut

CATEGORY	RISK FACTOR
Demographic Risks	
	Mother's age
	Mother's race
	Mother's socioeconomic status
	Mother's marital status
	Mother's education
Medical Risks prior to this pregnancy	
	Parity (0 or >4)
	Low weight (adjusted for height)
	Selected disease (diabetes, hypertension)
	Previous low birthweight baby
	low weight at own birth
	Multiple spontaneous abortions
Medical risks during this pregnancy	
	Multiple pregnancy
	Poor weight gain
	Short interpregnancy interval
	Hypotenision
	Hypertension/preeclampsia/toxemia
	Selected infections
	First or second trimester bleeding
	placenta previa and abruptio placenta
	Hyperemesis
	Oligohydramnios/polyhydramnios
	Anemia/abnormal hemoglobin
	Isoimmunization
	Fetal anomalies
	Incompetent cervix
	Spontaneous premature rupture of membranes
Behavioral and Environmental Risks	
	Smoking
	Poor nutritional status
	Alcohol and substance abuse
	DES
	High altitude
	Stress
Health Care Risks	
	Inadequate prenatal care
	Iatrogenic prematurity

* Adapted from: Institute of Medicine, Committee to Study the Prevention of Low Birthweight, Division of Health Promotion and Disease Prevention, National Academy Press, Washington, DC 1985

Table 5 Comparative Morbidity Figures (CMF)* for Stratford as compared to Connecticut and 95% confidence intervals by tumor site, time period, and gender, 1958 - 1991

Tumor Site	Time Period	Comparative Morbidity Figure and 95% Confidence Interval		
		Female and Male Combined	Female	Male
Mesothelioma	1958-62	2.336 (.369, 14.8)	-	-
	1963-67	**5.096 (1.59, 16.3)**†	-	-
	1968-72	1.49 (.198, 11.19)	-	-
	1973-77	.598 (.081, 4.419)	-	-
	1978-82	1.01 (.331, 3.052)	-	-
	1983-87	1.48 (.310, 7.029)	-	-
	1988-91	.693 (.128, 3.736)	-	-
Bladder	1958-62	.859 (.372, 1.986)	.750 (.194, 2.898)	.818 (.310, 2.161)
	1963-67	.969 (.537, 1.746)	1.00 (.321, 3.120)	1.07 (.555, 2.067)
	1968-72	1.03 (.592, 1.791)	1.50 (.687, 3.275)	.875 (.471, 1.627)
	1973-77	.958 (.569, 1.612)	1.25 (.580, 2.695)	.941 (.519, 1.706)
	1978-82	1.167 (.754, 1.81)	1.00 (.333, 3.003)	1.17 (.702, 1.938)
	1983-87	1.268 (.829, 1.94)	1.25 (.593, 2.635)	1.17 (.705, 1.932)
	1988-91	**1.66 (1.063, 2.60)**	2.00 (.844, 4.739)	1.33 (.806, 2.206)
Testicular	1958-62	-	-	0.00 (.000, .000)
	1963-67	-	-	1.06 (.256, 4.395)
	1968-72	-	-	**2.97 (1.256, 7.04)**
	1973-77	-	-	.842 (.269, 2.635)
	1978-82	-	-	.422 (.071, 2.516)
	1983-87	-	-	.823 (.332, 2.037)
	1988-91	-	-	.917 (.373, 2.256)
EOC‡	1958-62	1.50 (0.88, 2.57)	1.50 (0.75, 2.99)	1.33 (0.72, 2.48)
	1963-67	1.00 (0.62, 1.62)	0.67 (0.27, 1.67)	1.00 (0.48, 2.10)
	1968-72	**1.67 (1.13, 2.46)**	**2.00 (1.24, 3.23)**	1.67 (0.98, 2.83)
	1973-77	1.33 (0.88, 2.02)	1.00 (0.50, 2.00)	1.33 (0.70, 2.53)
	1978-82	1.33 (0.82, 2.16)	1.33 (0.67, 2.65)	1.00 (0.51, 1.96)
	1983-87	1.00 (0.61, 1.64)	0.50 (0.17, 1.44)	1.50 (0.86,2.61)
	1988-91	1.00 (0.56, 1.80)	1.67 (0.90, 3.09)	0.67 (0.28, 1.59)

* CMF's were computed based on direct standardized incidence rates from Stratford, CT and the State of Connecticut. Data was obtained from the Connecticut Tumor Registry.

† Bold text indicates statistically significant finding

‡ Early Onset Cancers (EOC) - all tumors occuring to individuals under the age 25

Table 6 Summary of Data Utilized in the Cancer Trend Analysis, Stratford, Connecticut, 1958 - 1991

Time Period	Location	Gender	Tumor Site	Age Group	No. of Tumors	Population
1958-62	CT	Female	EOC *	<25	404	791837
1958-62	CT	Male	EOC	<25	450	530979
1958-62	Stratford	Female	EOC	<25	7	13880
1958-62	Stratford	Male	EOC	<25	10	12870
1963-67	CT	Female	EOC	<25	466	596726
1963-67	CT	Male	EOC	<25	513	603326
1963-67	Stratford	Female	EOC	<25	6	9543
1963-67	Stratford	Male	EOC	<25	9	9622
1968-72	CT	Female	EOC	<25	547	671615
1968-72	CT	Male	EOC	<25	601	675671
1968-72	Stratford	Female	EOC	<25	16	9955
1968-72	Stratford	Male	EOC	<25	14	10111
1973-77	CT	Female	EOC	<25	522	633702
1973-77	CT	Male	EOC	<25	599	643939
1973-77	Stratford	Female	EOC	<25	7	9088
1973-77	Stratford	Male	EOC	<25	11	9265
1978-82	CT	Female	EOC	<25	545	595788
1978-82	CT	Male	EOC	<25	607	612206
1978-82	Stratford	Female	EOC	<25	10	8221
1978-82	Stratford	Male	EOC	<25	10	8418
1983-87	CT	Female	EOC	<25	562	565634
1983-87	CT	Male	EOC	<25	664	585872
1983-87	Stratford	Female	EOC	<25	5	7585
1983-87	Stratford	Male	EOC	<25	13	7785
1988-91	CT	Female	EOC	<25	398	535478
1988-91	CT	Male	EOC	<25	444	559536
1988-91	Stratford	Female	EOC	<25	9	6948
1988-91	Stratford	Male	EOC	<25	3	7151
1958-62	CT	Female	Bladder	0-44	12	881687
1958-62	CT	Male	Bladder	0-44	41	876418
1958-62	Stratford	Female	Bladder	0-44	0	15754
1958-62	Stratford	Male	Bladder	0-44	1	15253
1958-62	CT	Female	Bladder	45-64	100	273913
1958-62	CT	Male	Bladder	45-64	385	260601
1958-62	Stratford	Female	Bladder	45-64	1	5055
1958-62	Stratford	Male	Bladder	45-64	7	5028
1958-62	CT	Female	Bladder	65-74	96	88359
1958-62	CT	Male	Bladder	65-74	412	74564
1958-62	Stratford	Female	Bladder	65-74	1	1450
1958-62	Stratford	Male	Bladder	65-74	7	1204
1958-62	CT	Female	Bladder	75+	143	47046
1958-62	CT	Male	Bladder	75+	272	32646
1958-62	Stratford	Female	Bladder	75+	3	752
1958-62	Stratford	Male	Bladder	75+	2	516
1963-67	CT	Female	Bladder	0-44	26	962963
1963-67	CT	Male	Bladder	0-44	88	955091
1963-67	Stratford	Female	Bladder	0-44	0	15723

1963-67	Stratford	Male	Bladder	0-44	0	15351
1963-67	CT	Female	Bladder	45-64	145	309392
1963-67	CT	Male	Bladder	45-64	519	290267
1963-67	Stratford	Female	Bladder	45-64	0	6111
1963-67	Stratford	Male	Bladder	45-64	16	5802
1963-67	CT	Female	Bladder	65-74	159	94653
1963-67	CT	Male	Bladder	65-74	509	74214
1963-67	Stratford	Female	Bladder	65-74	3	1582
1963-67	Stratford	Male	Bladder	65-74	7	1247
1963-67	CT	Female	Bladder	75+	155	59107
1963-67	CT	Male	Bladder	75+	375	37789
1963-67	Stratford	Female	Bladder	75+	3	956
1963-67	Stratford	Male	Bladder	75+	7	626
1968-72	CT	Female	Bladder	0-44	39	1044238
1968-72	CT	Male	Bladder	0-44	95	1033760
1968-72	Stratford	Female	Bladder	0-44	0	15692
1968-72	Stratford	Male	Bladder	0-44	2	15446
1968-72	CT	Female	Bladder	45-64	175	344870
1968-72	CT	Male	Bladder	45-64	618	319933
1968-72	Stratford	Female	Bladder	45-64	3	7166
1968-72	Stratford	Male	Bladder	45-64	16	6575
1968-72	CT	Female	Bladder	65-74	156	100947
1968-72	CT	Male	Bladder	65-74	500	73863
1968-72	Stratford	Female	Bladder	65-74	6	1713
1968-72	Stratford	Male	Bladder	65-74	12	1290
1968-72	CT	Female	Bladder	75+	207	71167
1968-72	CT	Male	Bladder	75+	465	42931
1968-72	Stratford	Female	Bladder	75+	4	1159
1968-72	Stratford	Male	Bladder	75+	1	734
1973-77	CT	Female	Bladder	0-44	31	1040203
1973-77	CT	Male	Bladder	0-44	61	1033013
1973-77	Stratford	Female	Bladder	0-44	0	15138
1973-77	Stratford	Male	Bladder	0-44	1	14877
1973-77	CT	Female	Bladder	45-64	261	348464
1973-77	CT	Male	Bladder	45-64	780	321080
1973-77	Stratford	Female	Bladder	45-64	6	7308
1973-77	Stratford	Male	Bladder	45-64	25	6551
1973-77	CT	Female	Bladder	65-74	167	112923
1973-77	CT	Male	Bladder	65-74	627	84341
1973-77	Stratford	Female	Bladder	65-74	5	2293
1973-77	Stratford	Male	Bladder	65-74	9	1755
1973-77	CT	Female	Bladder	75+	271	83809
1973-77	CT	Male	Bladder	75+	597	45814
1973-77	Stratford	Female	Bladder	75+	3	1442
1973-77	Stratford	Male	Bladder	75+	7	797
1978-82	CT	Female	Bladder	0-44	38	1036165
1978-82	CT	Male	Bladder	0-44	60	1032264
1978-82	Stratford	Female	Bladder	0-44	1	14584
1978-82	Stratford	Male	Bladder	0-44	1	14306
1978-82	CT	Female	Bladder	45-64	275	352057
1978-82	CT	Male	Bladder	45-64	737	322226
1978-82	Stratford	Female	Bladder	45-64	7	7450
1978-82	Stratford	Male	Bladder	45-64	22	6526
1978-82	CT	Female	Bladder	65-74	245	124899

1978-82	CT	Male	Bladder	65-74	777	94818
1978-82	Stratford	Female	Bladder	65-74	4	2873
1978-82	Stratford	Male	Bladder	65-74	24	2219
1978-82	CT	Female	Bladder	75+	340	96450
1978-82	CT	Male	Bladder	75+	698	48697
1978-82	Stratford	Female	Bladder	75+	4	1724
1978-82	Stratford	Male	Bladder	75+	11	859
1983-87	CT	Female	Bladder	0-44	24	1062099
1983-87	CT	Male	Bladder	0-44	81	1067065
1983-87	Stratford	Female	Bladder	0-44	0	14513
1983-87	Stratford	Male	Bladder	0-44	2	14416
1983-87	CT	Female	Bladder	45-64	219	344356
1983-87	CT	Male	Bladder	45-64	705	318445
1983-87	Stratford	Female	Bladder	45-64	5	6586
1983-87	Stratford	Male	Bladder	45-64	17	5761
1983-87	CT	Female	Bladder	65-74	275	134677
1983-87	CT	Male	Bladder	65-74	788	103300
1983-87	Stratford	Female	Bladder	65-74	11	3134
1983-87	Stratford	Male	Bladder	65-74	17	2344
1983-87	CT	Female	Bladder	75+	347	110778
1983-87	CT	Male	Bladder	75+	750	56632
1983-87	Stratford	Female	Bladder	75+	4	2087
1983-87	Stratford	Male	Bladder	75+	21	1129
1988-91	CT	Female	Bladder	0-44	13	1088030
1988-91	CT	Male	Bladder	0-44	37	1101862
1988-91	Stratford	Female	Bladder	0-44	2	14440
1988-91	Stratford	Male	Bladder	0-44	0	14524
1988-91	CT	Female	Bladder	45-64	151	336654
1988-91	CT	Male	Bladder	45-64	463	314663
1988-91	Stratford	Female	Bladder	45-64	8	5721
1988-91	Stratford	Male	Bladder	45-64	11	4994
1988-91	CT	Female	Bladder	65-74	191	144455
1988-91	CT	Male	Bladder	65-74	571	111782
1988-91	Stratford	Female	Bladder	65-74	6	3395
1988-91	Stratford	Male	Bladder	65-74	18	2468
1988-91	CT	Female	Bladder	75+	288	125104
1988-91	CT	Male	Bladder	75+	582	64566
1988-91	Stratford	Female	Bladder	75+	6	2449
1988-91	Stratford	Male	Bladder	75+	19	1398
1958-62	CT	Female	Mesothelioma	0-44	2	881687
1958-62	CT	Male	Mesothelioma	0-44	3	876418
1958-62	Stratford	Female	Mesothelioma	0-44	0	15754
1958-62	Stratford	Male	Mesothelioma	0-44	0	15253
1958-62	CT	Female	Mesothelioma	45-64	6	273913
1958-62	CT	Male	Mesothelioma	45-64	12	260601
1958-62	Stratford	Female	Mesothelioma	45-64	0	5055
1958-62	Stratford	Male	Mesothelioma	45-64	0	5028
1958-62	CT	Female	Mesothelioma	65-74	2	88359
1958-62	CT	Male	Mesothelioma	65-74	11	74564
1958-62	Stratford	Female	Mesothelioma	65-74	0	1450
1958-62	Stratford	Male	Mesothelioma	65-74	1	1204
1958-62	CT	Female	Mesothelioma	75+	4	47046
1958-62	CT	Male	Mesothelioma	75+	0	32646
1958-62	Stratford	Female	Mesothelioma	75+	0	752

1958-62	Stratford	Male	Mesothelioma	75+	0	516
1963-67	CT	Female	Mesothelioma	0-44	1	962963
1963-67	CT	Male	Mesothelioma	0-44	3	955091
1963-67	Stratford	Female	Mesothelioma	0-44	0	15723
1963-67	Stratford	Male	Mesothelioma	0-44	0	15351
1963-67	CT	Female	Mesothelioma	45-64	8	309392
1963-67	CT	Male	Mesothelioma	45-64	10	290267
1963-67	Stratford	Female	Mesothelioma	45-64	1	6111
1963-67	Stratford	Male	Mesothelioma	45-64	1	5802
1963-67	CT	Female	Mesothelioma	65-74	3	94653
1963-67	CT	Male	Mesothelioma	65-74	6	74214
1963-67	Stratford	Female	Mesothelioma	65-74	0	1582
1963-67	Stratford	Male	Mesothelioma	65-74	0	1247
1963-67	CT	Female	Mesothelioma	75+	0	59107
1963-67	CT	Male	Mesothelioma	75+	2	37789
1963-67	Stratford	Female	Mesothelioma	75+	0	956
1963-67	Stratford	Male	Mesothelioma	75+	0	626
1968-72	CT	Female	Mesothelioma	0-44	3	1044238
1968-72	CT	Male	Mesothelioma	0-44	5	1033760
1968-72	Stratford	Female	Mesothelioma	0-44	0	15692
1968-72	Stratford	Male	Mesothelioma	0-44	0	15446
1968-72	CT	Female	Mesothelioma	45-64	6	344870
1968-72	CT	Male	Mesothelioma	45-64	18	319933
1968-72	Stratford	Female	Mesothelioma	45-64	1	7166
1968-72	Stratford	Male	Mesothelioma	45-64	1	6575
1968-72	CT	Female	Mesothelioma	65-74	5	100947
1968-72	CT	Male	Mesothelioma	65-74	6	73863
1968-72	Stratford	Female	Mesothelioma	65-74	0	1713
1968-72	Stratford	Male	Mesothelioma	65-74	0	1290
1968-72	CT	Female	Mesothelioma	75+	2	71167
1968-72	CT	Male	Mesothelioma	75+	5	42931
1968-72	Stratford	Female	Mesothelioma	75+	0	1159
1968-72	Stratford	Male	Mesothelioma	75+	0	734
1973-77	CT	Female	Mesothelioma	0-44	2	1040203
1973-77	CT	Male	Mesothelioma	0-44	1	1033013
1973-77	Stratford	Female	Mesothelioma	0-44	0	15138
1973-77	Stratford	Male	Mesothelioma	0-44	0	14877
1973-77	CT	Female	Mesothelioma	45-64	15	348464
1973-77	CT	Male	Mesothelioma	45-64	24	321080
1973-77	Stratford	Female	Mesothelioma	45-64	1	7308
1973-77	Stratford	Male	Mesothelioma	45-64	1	6551
1973-77	CT	Female	Mesothelioma	65-74	2	112923
1973-77	CT	Male	Mesothelioma	65-74	10	84341
1973-77	Stratford	Female	Mesothelioma	65-74	0	2293
1973-77	Stratford	Male	Mesothelioma	65-74	0	1755
1973-77	CT	Female	Mesothelioma	75+	3	83809
1973-77	CT	Male	Mesothelioma	75+	14	45814
1973-77	Stratford	Female	Mesothelioma	75+	0	1442
1973-77	Stratford	Male	Mesothelioma	75+	0	797
1978-82	CT	Female	Mesothelioma	0-44	3	1036165
1978-82	CT	Male	Mesothelioma	0-44	1	1032264
1978-82	Stratford	Female	Mesothelioma	0-44	0	14584
1978-82	Stratford	Male	Mesothelioma	0-44	0	14306
1978-82	CT	Female	Mesothelioma	45-64	12	352057

1978-82	CT	Male	Mesothelioma	45-64	33	322226
1978-82	Stratford	Female	Mesothelioma	45-64	0	7450
1978-82	Stratford	Male	Mesothelioma	45-64	0	6526
1978-82	CT	Female	Mesothelioma	65-74	11	124899
1978-82	CT	Male	Mesothelioma	65-74	35	94818
1978-82	Stratford	Female	Mesothelioma	65-74	2	2873
1978-82	Stratford	Male	Mesothelioma	65-74	1	2219
1978-82	CT	Female	Mesothelioma	75+	5	96450
1978-82	CT	Male	Mesothelioma	75+	17	48697
1978-82	Stratford	Female	Mesothelioma	75+	0	1724
1978-82	Stratford	Male	Mesothelioma	75+	0	859
1983-87	CT	Female	Mesothelioma	0-44	1	1062099
1983-87	CT	Male	Mesothelioma	0-44	5	1067065
1983-87	Stratford	Female	Mesothelioma	0-44	0	14513
1983-87	Stratford	Male	Mesothelioma	0-44	0	14416
1983-87	CT	Female	Mesothelioma	45-64	14	344356
1983-87	CT	Male	Mesothelioma	45-64	45	318445
1983-87	Stratford	Female	Mesothelioma	45-64	0	6586
1983-87	Stratford	Male	Mesothelioma	45-64	1	5761
1983-87	CT	Female	Mesothelioma	65-74	12	134677
1983-87	CT	Male	Mesothelioma	65-74	28	103300
1983-87	Stratford	Female	Mesothelioma	65-74	0	3134
1983-87	Stratford	Male	Mesothelioma	65-74	1	2344
1983-87	CT	Female	Mesothelioma	75+	7	110778
1983-87	CT	Male	Mesothelioma	75+	23	56632
1983-87	Stratford	Female	Mesothelioma	75+	0	2087
1983-87	Stratford	Male	Mesothelioma	75+	2	1129
1988-91	CT	Female	Mesothelioma	0-44	4	1088030
1988-91	CT	Male	Mesothelioma	0-44	2	1101862
1988-91	Stratford	Female	Mesothelioma	0-44	0	14440
1988-91	Stratford	Male	Mesothelioma	0-44	0	14524
1988-91	CT	Female	Mesothelioma	45-64	12	336654
1988-91	CT	Male	Mesothelioma	45-64	21	314663
1988-91	Stratford	Female	Mesothelioma	45-64	0	5721
1988-91	Stratford	Male	Mesothelioma	45-64	0	4994
1988-91	CT	Female	Mesothelioma	65-74	10	144455
1988-91	CT	Male	Mesothelioma	65-74	37	111782
1988-91	Stratford	Female	Mesothelioma	65-74	0	3395
1988-91	Stratford	Male	Mesothelioma	65-74	1	2468
1988-91	CT	Female	Mesothelioma	75+	10	125104
1988-91	CT	Male	Mesothelioma	75+	24	64566
1988-91	Stratford	Female	Mesothelioma	75+	0	2449
1988-91	Stratford	Male	Mesothelioma	75+	1	1398
1958-62	CT	Male	Testicular	0-44	127	876418
1958-62	Stratford	Male	Testicular	0-44	0	15253
1958-62	CT	Male	Testicular	45-64	24	260601
1958-62	Stratford	Male	Testicular	45-64	0	5028
1958-62	CT	Male	Testicular	65-74	5	74564
1958-62	Stratford	Male	Testicular	65-74	0	1204
1958-62	CT	Male	Testicular	75+	3	32646
1958-62	Stratford	Male	Testicular	75+	0	516
1963-67	CT	Male	Testicular	0-44	133	955091
1963-67	Stratford	Male	Testicular	0-44	1	15351
1963-67	CT	Male	Testicular	45-64	38	290267

1963-67	Stratford	Male	Testicular	45-64	1	5802
1963-67	CT	Male	Testicular	65-74	4	74214
1963-67	Stratford	Male	Testicular	65-74	0	1247
1963-67	CT	Male	Testicular	75+	2	37789
1963-67	Stratford	Male	Testicular	75+	0	626
1968-72	CT	Male	Testicular	0-44	150	1033760
1968-72	Stratford	Male	Testicular	0-44	6	15446
1968-72	CT	Male	Testicular	45-64	39	319933
1968-72	Stratford	Male	Testicular	45-64	1	6575
1968-72	CT	Male	Testicular	65-74	8	73863
1968-72	Stratford	Male	Testicular	65-74	0	1290
1968-72	CT	Male	Testicular	75+	8	42931
1968-72	Stratford	Male	Testicular	75+	0	734
1973-77	CT	Male	Testicular	0-44	213	1033013
1973-77	Stratford	Male	Testicular	0-44	3	14877
1973-77	CT	Male	Testicular	45-64	47	321080
1973-77	Stratford	Male	Testicular	45-64	0	6551
1973-77	CT	Male	Testicular	65-74	6	84341
1973-77	Stratford	Male	Testicular	65-74	0	1755
1973-77	CT	Male	Testicular	75+	2	45814
1973-77	Stratford	Male	Testicular	75+	0	797
1978-82	CT	Male	Testicular	0-44	264	1032264
1978-82	Stratford	Male	Testicular	0-44	2	14306
1978-82	CT	Male	Testicular	45-64	49	322226
1978-82	Stratford	Male	Testicular	45-64	1	6526
1978-82	CT	Male	Testicular	65-74	6	94818
1978-82	Stratford	Male	Testicular	65-74	0	2219
1978-82	CT	Male	Testicular	75+	2	48697
1978-82	Stratford	Male	Testicular	75+	0	859
1983-87	CT	Male	Testicular	0-44	352	1067065
1983-87	Stratford	Male	Testicular	0-44	4	14416
1983-87	CT	Male	Testicular	45-64	49	318445
1983-87	Stratford	Male	Testicular	45-64	0	5761
1983-87	CT	Male	Testicular	65-74	7	103300
1983-87	Stratford	Male	Testicular	65-74	0	2344
1983-87	CT	Male	Testicular	75+	1	56632
1983-87	Stratford	Male	Testicular	75+	0	1129
1988-91	CT	Male	Testicular	0-44	309	1101862
1988-91	Stratford	Male	Testicular	0-44	4	14524
1988-91	CT	Male	Testicular	45-64	35	314663
1988-91	Stratford	Male	Testicular	45-64	0	4994
1988-91	CT	Male	Testicular	65-74	5	111782
1988-91	Stratford	Male	Testicular	65-74	0	2468
1988-91	CT	Male	Testicular	75+	2	64566
1988-91	Stratford	Male	Testicular	75+	0	1398

* Early Onset Cancers (EOC) - all tumors occurring to individuals under the age 25

Table 7 Test of Trend Over Time of the Comparative Morbidity Figures, 1958 - 1991, Stratford as Compared to Connecticut

Tumor Site	F value	Prob.>F
Mesothelioma	3.20	0.13
Bladder (Female & Male)	**12.82***	**0.02**
Bladder (Female)	5.04	0.08
Bladder (Male)	**19.63**	**0.01**
Testicular	0.00	0.96
Early Onset Cancers (Female & Male)	1.9	0.23
Early Onset Cancers (Female)	0.03	0.88
Early Onset Cancers (Male)	0.80	0.41

* Bold text indicates statistically significant finding

Table 8 Cancer Odds Ratios and 95% Confidence Intervals by Tumor, Exposure Model, and Controls From 1968 - 1991, Stratford, Connecticut

Tumor Site	Sample Size	Exposure Model	Odds Ratio Uncontrolled	95% CI*	Odds Ratio Controlled	95% CI*
Mesothelioma	12	Block Group	0.905	(0.109, 7.523)	-	-
		Distance to Nearest Site	1.063	(0.369, 3.061)	-	-
Bladder	307	Block Group	1.436	(0.996, 2.072)	1.417 †	(0.978, 2.051)
		Distance to Nearest Site	1.093	(0.882, 1.354)	1.222 †	(0.986, 1.514)
Testicular	21	Block Group	0.754	(0.137, 4.149)	-	-
		Distance to Nearest Site	1.403	(0.522, 3.769)	-	-
EOC‡	111	Block Group	1.315	(0.706, 2.449)	1.387 §	(0.739, 2.572)
		Distance to Nearest Site	1.465	(0.940, 2.285)	1.340 §	(0.845, 2.124)

* Bonferroni adjustment
† Control variables: age, gender
‡ Early Onset Cancers (EOC) - all tumors occuring to individuals under the age 25
§ Control variables: age

Table 9: Odds Ratios (OR) and 95% Confidence Intervals for Bladder Cancer by Gender and Exposure Models, 1968 - 1991, Stratford, Connecticut

Gender	Sample Size	Exposure Model*	OR (95% CI)
Female	81	Block group	1.960 (1.214-3.167)
		Distance from nearest waste site	1.611 (1.118-2.322)
Male	226	Block group	1.257 (0.913-1.731)
		Distance from nearest waste site	1.130 (0.953-1.339)

* All models control for age in four age groupings.

Table 10 Mean Birthweight, LBW, and VLBW According to Selected Risk Factors, 1982 - 1991, Stratford, Connecticut

Characteristic	N	Mean BW (g)	LBW (N)	LBW (%)	VLBW (N)	VLBW (%)
Gestational Age (weeks)						
< 37	292	2200	112	38.4	60	20.6
> 37	4465	3466	109	2.4	1	0.0
Maternal Age (years)						
< 20	337	3216	26	7.7	9	2.7
20 - 24	888	3352	40	4.5	19	2.1
25 - 29	2005	3408	83	4.1	16	0.8
30 - 34	1659	3407	76	4.6	17	1.0
35 +	538	3419	27	5.0	7	1.3
Marital Status						
Married	4546	3422	175	3.9	41	0.9
Other	871	3210	77	8.8	27	3.1
Child's Race						
White	4740	3414	201	4.2	44	0.9
Black	604	3198	47	7.8	22	3.6
Other	78	3240	4	5.1	2	2.6
Parity						
0	2264	3346	107	4.7	25	1.1
1	1588	3414	70	4.4	26	1.6
2	731	3442	34	4.7	7	1.0
3	188	3545	3	1.6	2	1.1
4 +	68	3234	6	8.8	4	5.9
Prenatal Care						
Adequate	3438	3406	150	4.4	42	1.2
Intermediate	1113	3375	50	4.5	12	1.1
Inadequate	253	3211	22	8.7	7	2.8
Interval Between Pregnancies (months)						
0 - 1	10	3080	2	20.0	0	0.0
2 - 5	117	3454	2	1.7	1	0.9
6 - 12	392	3461	13	3.3	1	0.2
13 - 24	652	3468	19	2.9	3	0.5
25 +	1521	3467	51	3.4	12	0.8
Maternal Education (grade level)						
< 9	46	3039	4	8.7	4	8.7
9 - 11	291	3166	30	10.3	8	2.8
12	1823	3370	90	4.9	25	1.4
13 +	2218	3436	89	4.0	18	0.8

Table 11 Distribution of Births and Mean Birthweight and 95% Confidence Intervals within Block Group Exposure Categories, 1982 - 1991, Stratford, Connecticut

Block Group Exposure Classification	Frequency	Percent	Mean Birthweight	95% Confidence Interval
No Waste	4247	80.6	3411.36	(3394.36, 3428.37)
With Waste	1028	19.4	3455.26	(3422.63, 3487.90)

Table 12 Birthweight Regression Analysis Results - Birthweight as a Continous Outcome, 1982 - 1991, Stratford, Connecticut

	Uncontrolled Model				Controlled Model*		
Exposure Model	**Single Variable Regression Model**	**F value**	**Prob.>F**	**R^2**	**Parameter Estimate of the Exposure Variable**	**T value**	**Prob. >|T|**
Block Group	Birthweight = 3411.36 + 43.9(exposure)	5.08	0.024	0.001	24.27	1.25	0.211
Distance to Nearest Site	Birthweight = 3405.27 + 4.8(distance)	0.19	0.660	0.000	10.5	0.97	.333
Cumulative Exposure	Birthweight = 3404.16 + 31.43(exposure)	1.38	0.239	0.000	44.44	1.68	0.09

* Control variables: Child's Race, Mother's Age, and Marital Status

Table 13 Birthweight Analysis Results - Birthweight as a categorical outcome (LBW-VLBW), 1982 - 1991, Stratford, Connecticut

Birthweight	Sample Size	Exposure Model	Odds Ratio Uncontrolled	95% CI	Odds Ratio Controlled *	95% CI
LBW†	233 (4.4%)	Block Group	0.80	(0.56, 1.14)	0.92	(0.64, 1.32)
		Distance to Nearest Site	1.01	(0.84, 1.22)	0.97	(0.78, 1.21)
		Cumulative Exposure	0.69	(0.44, 1.10)	0.62	(0.39, 0.99)
VLBW‡	41 (0.8%)	Block Group	0.58	(0.23, 1.47)	0.73	(0.29, 1.92)
		Distance to Nearest Site	1.27	(0.74, 2.16)	1.18	(0.68, 2.03)
		Cumulative Exposure	0.99	(0.34, 2.90)	0.79	(0.25, 2.46)

* Control variables: Child's Race, Mother's Age, and Marital Status
† Low Birthweight (<2500 grams)
‡ Very Low Birthweight (<1500 grams)

Table 14 Demographic and Reproductive Characteristics Among the Study Population in Stratford, Connecticut, 1982 - 1991

Characteristic	Distance to the nearest site: < 1/4 Mile N	< 1/4 Mile %	1/4 - 1 Mile N	1/4 - 1 Mile %	> 1 Mile N	> 1 Mile %	Total Pop. of Stratford N	Total Pop. of Stratford %
LBW	36	4.2	148	4.5	68	5.3	252	4.6
VLBW	8	0.9	51	1.6	9	0.7	68	1.3
Gestational Age (weeks)								
< 37	46	6.0	179	6.2	67	6.0	292	6.1
> 37	722	94.0	2701	95.8	1042	94.0	4465	93.9
Maternal Age (years)								
< 20	37	4.3	234	7.1	67	5.2	338	6.2
20 - 24	106	12.3	532	16.2	252	19.6	890	16.4
25 - 29	317	36.7	1203	36.5	489	38.1	2009	36.9
30 - 34	312	36.1	1003	30.4	350	27.3	1665	30.6
35 +	92	10.7	323	9.8	126	9.8	541	9.9
Marital Status								
Married	749	86.9	2728	82.9	1080	84.3	4557	83.9
Other	113	13.1	562	17.1	201	15.7	876	16.1
Mother's Race								
White	794	92.4	2819	86.0	1179	92.2	4792	88.5
Black	59	6.9	416	12.7	77	6.0	552	10.2
Other	6	0.7	45	1.3	27	1.8	73	1.4
Parity								
0	365	46.2	1338	45.7	561	50.2	2264	46.8
1	265	33.5	977	33.3	346	31.0	1588	32.8
2	118	14.9	444	15.2	169	15.1	731	15.1
3	30	3.8	127	4.3	31	2.8	188	3.9
4 +	12	1.5	45	1.5	11	1.0	68	1.4
Interval Between Pregnancies (months)								
0 - 1	1	0.2	6	0.4	3	0.5	10	0.4
2 - 5	16	3.7	73	4.4	28	4.7	117	4.4
6 - 12	67	15.4	240	14.5	85	14.2	392	14.6
13 - 24	115	26.5	379	22.9	158	26.4	652	24.2
25 +	235	54.2	961	57.9	325	54.3	1521	56.5
Maternal Education (grade level)								
< 9	5	0.7	28	1.1	13	1.3	46	1.1
9 - 11	46	6.4	166	6.3	80	7.8	292	6.7
12	256	35.7	1135	43.1	432	41.9	1823	41.6
13 +	410	57.2	1303	49.5	505	49.0	2218	50.7
Smoked Cigarettes								
No	29	3.4	90	2.7	50	3.9	169	3.1
Yes	230	26.6	821	24.9	324	6.0	1375	25.3
Missing	605	70.0	2384	72.3	910	70.9	3899	71.6
Prenatal Care								
Adequate	576	66.7	2046	62.1	816	63.6	3438	63.2
Intermediate	158	18.3	707	21.5	248	19.3	1113	20.5
Inadequate	43	5.0	154	4.7	56	4.4	253	4.7
Missing	87	10.1	388	11.8	164	12.8	639	11.7

IX. MAPS

FIGURE 1 MAP OF RAYMARK WASTE SITES LOCATED IN STRATFORD, CONNECTICUT, SEPTEMBER 1994 69
FIGURE 2 MAP OF EXPOSURE MODELS UTILIZED IN STRATFORD, CONNECTICUT, SEPTEMBER 1994 70
FIGURE 3 MAP OF MESOTHELIOMA AND TESTICULAR CANCER INCIDENCE FROM 1968 - 1991, STRATFORD, CONNECTICUT 71
FIGURE 4 MAP OF BLADDER CANCER INCIDENCE FROM 1968 - 1991, STRATFORD, CONNECTICUT 72
FIGURE 5 MAP OF EARLY ONSET CANCER INCIDENCE FROM 1968 - 1991, STRATFORD, CONNECTICUT 73
FIGURE 6 MAP OF FACTORS ASSOCIATED WITH BIRTHWEIGHT FROM 1982 - 1991, STRATFORD, CONNECTICUT 74
FIGURE 7 MAP OF BIRTHWEIGHT BY 1990 BLOCK GROUPS FROM 1982 - 1991, STRATFORD, CONNECTICUT 75

Figure 1: Map of Raymark Waste Sites Located in Stratford, Connecticut, September 1994

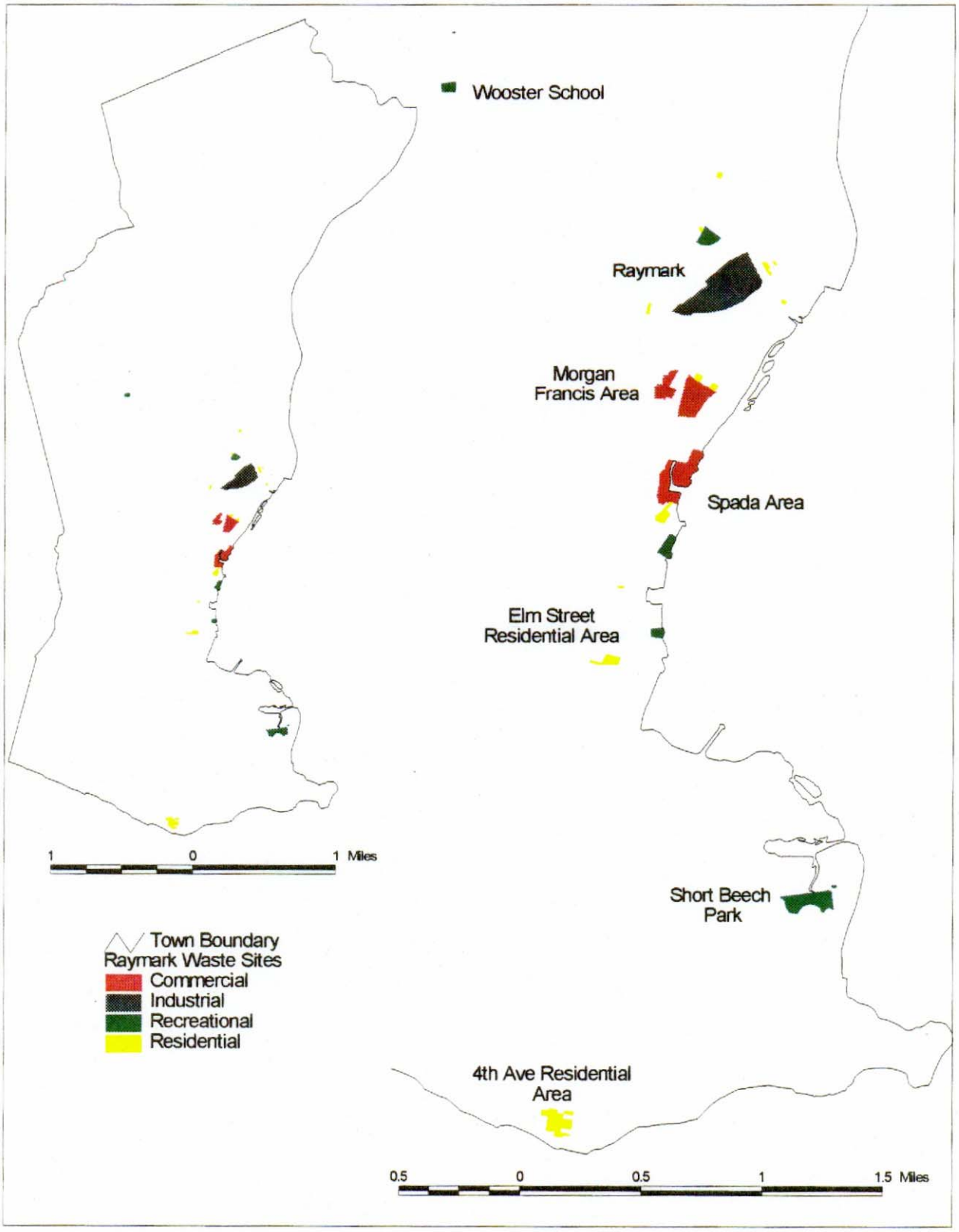


Figure 2: Map of Exposure Models Utilized in Stratford, Connecticut, September 1994

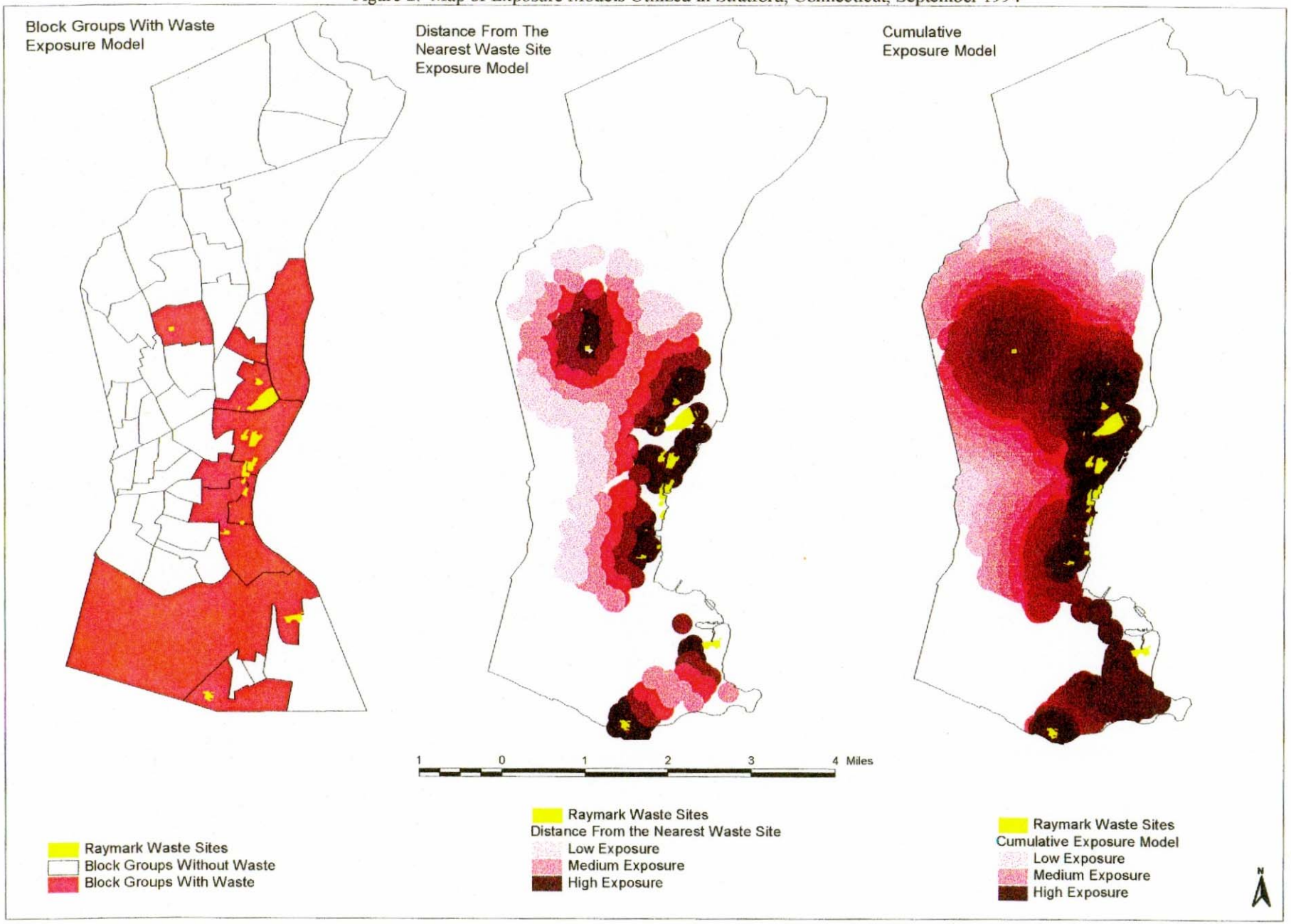


Figure 3: Map of Mesothelioma and Testicular Cancer Incidence per 1,000 From 1968 - 1991, Stratford, Connecticut

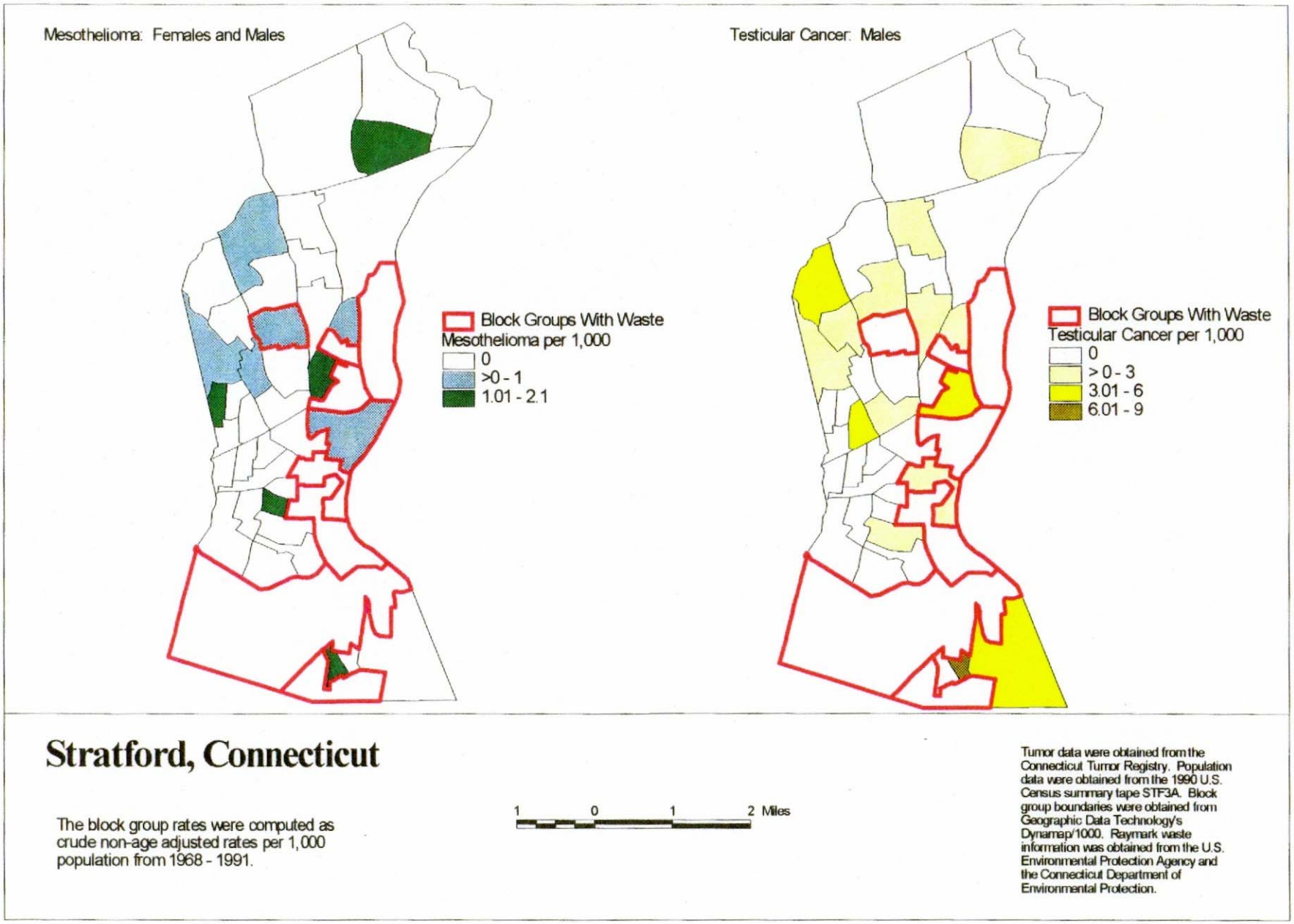


Figure 4: Map of Bladder Cancer Incidence per 1,000 From 1968 - 1991, Stratford, Connecticut

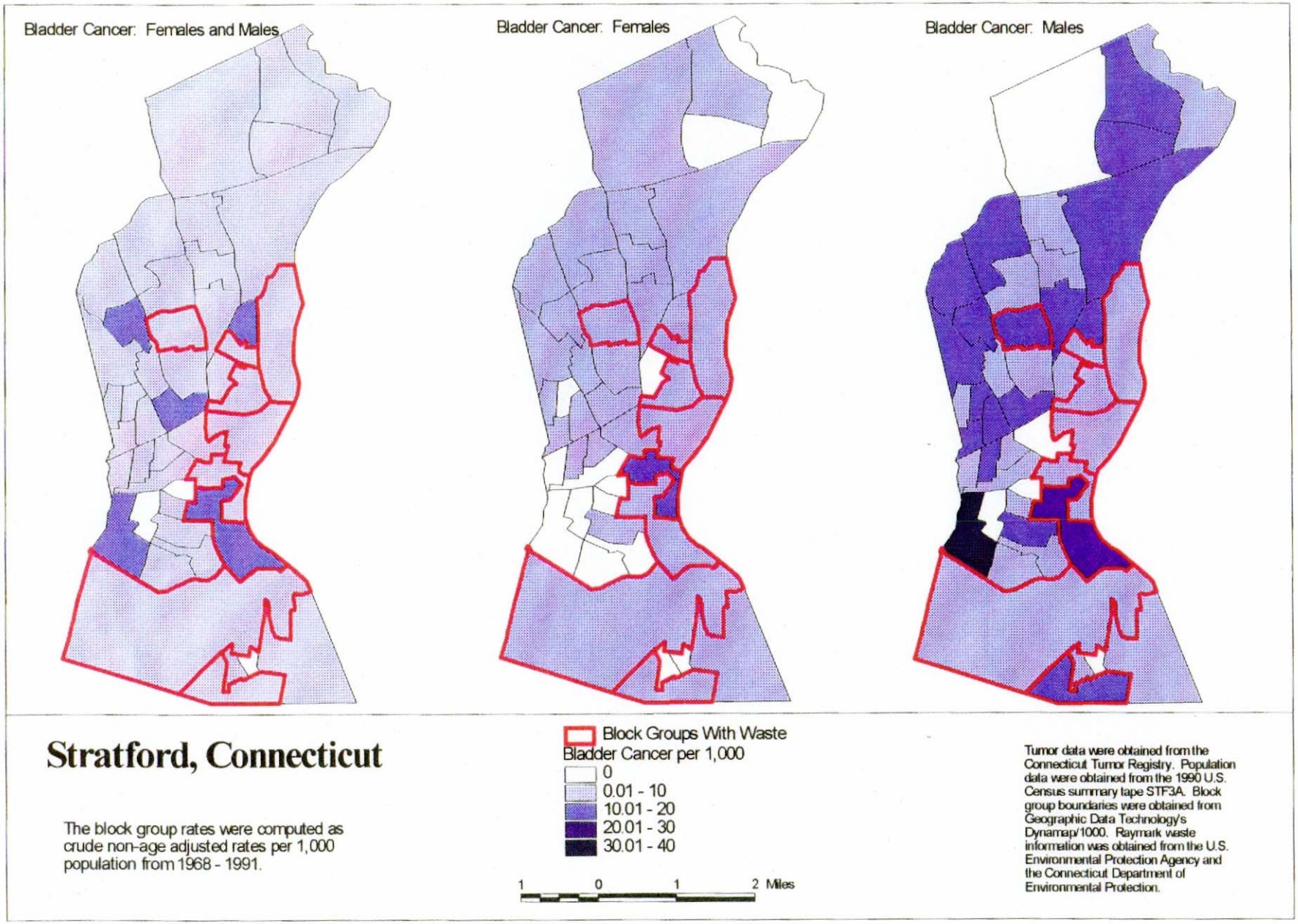


Figure 5: Map of Early Onset Cancer * Incidence per 1,000 From 1968 - 1991, Stratford, Connecticut

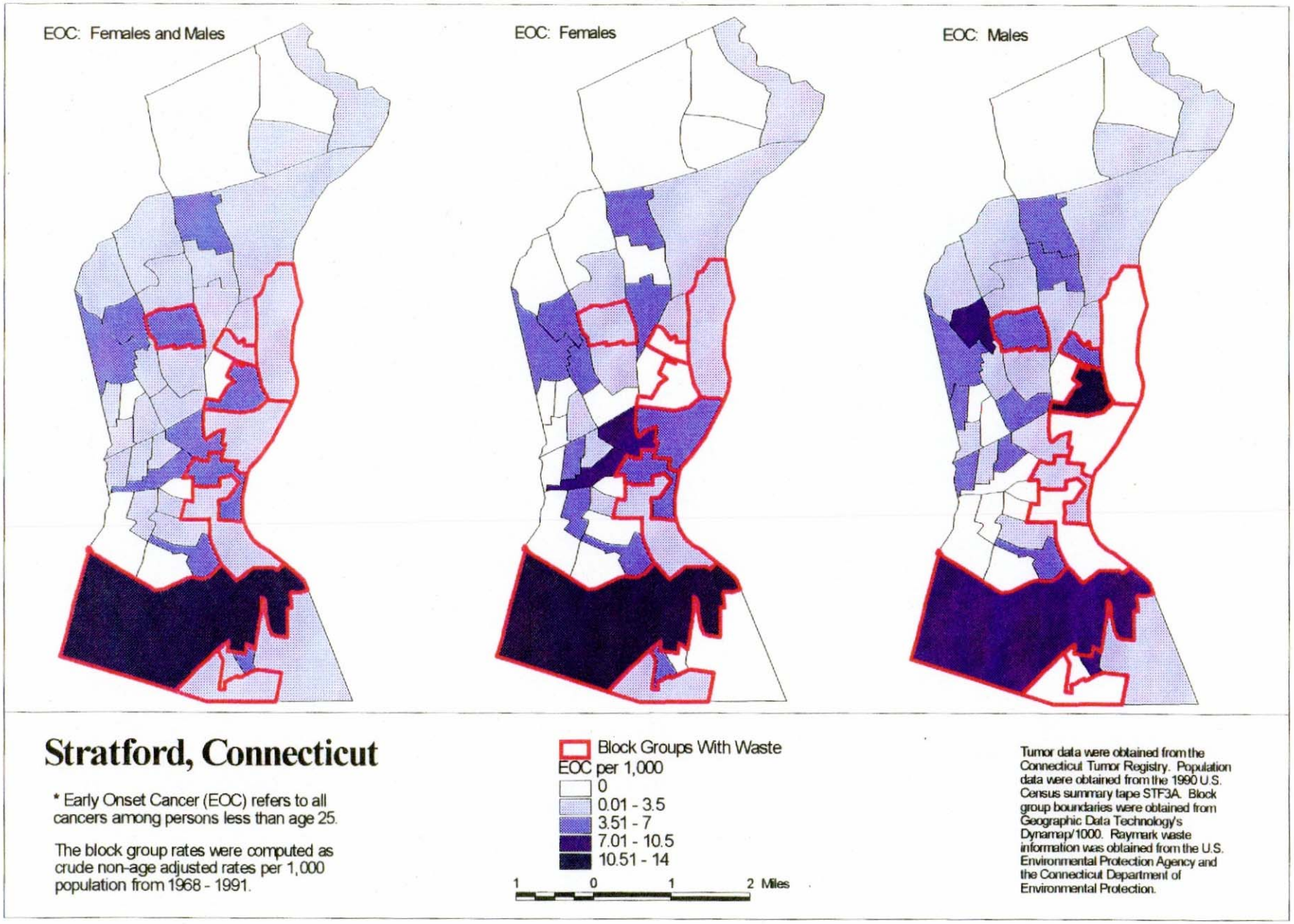


Figure 6: Map of Factors Associated With Birthweight From 1982 - 1991, Stratford, Connecticut

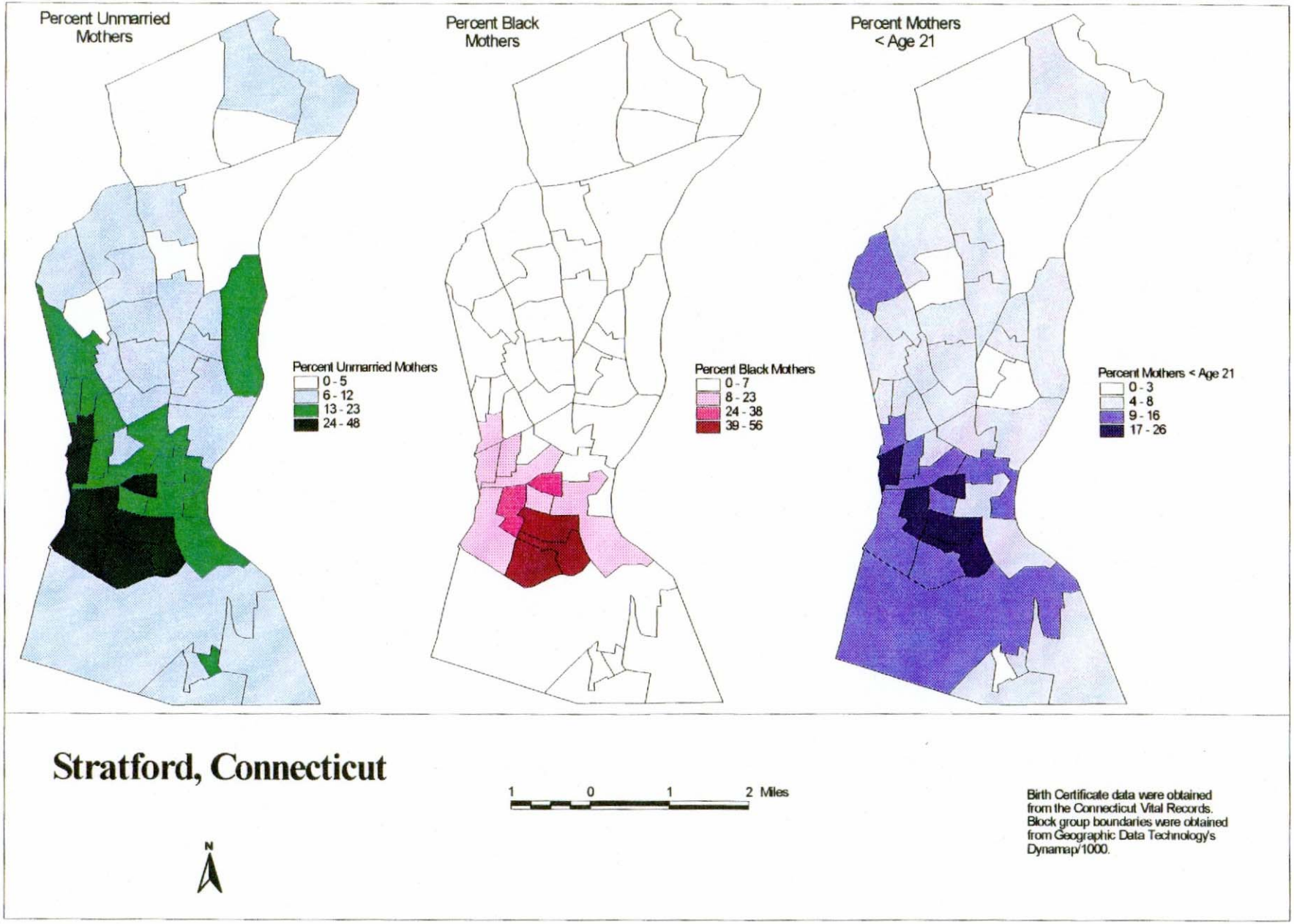
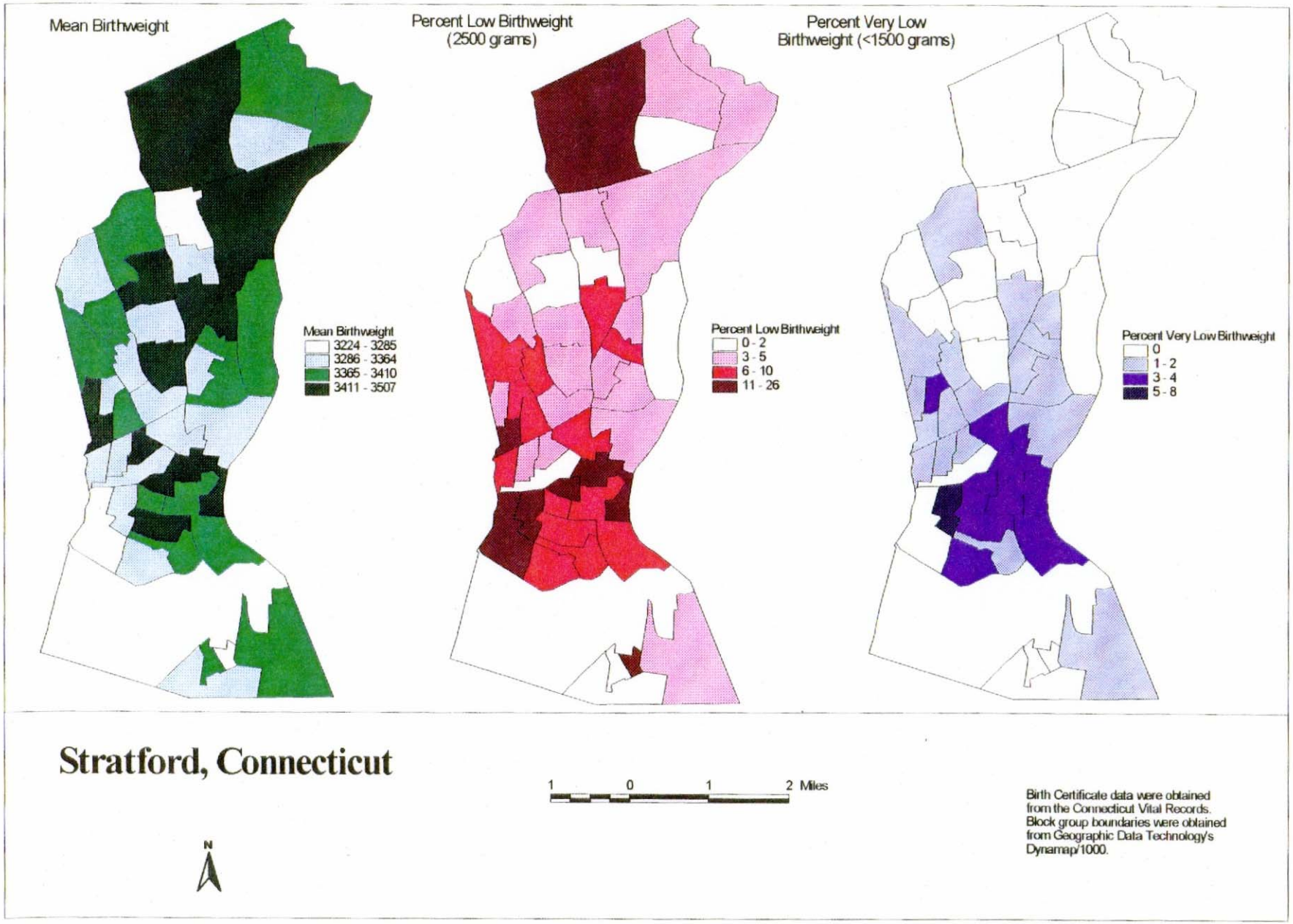


Figure 7: Map of Birthweight by 1990 Block Groups, From 1982 - 1991, Stratford, Connecticut



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XII. REFERENCES

[1] Sosniak WA, Kaye WE, Gomez TM. Data linkable to explore the risk of low birthweight associated with maternal proximity to hazardous waste sites from the National Priorities List. Arch Environ Health 1994;49:251-255.
[2] Geschwind S, Stolwijk, J, Bracken M, et.al. Risk of congenital malformations associated with proximity to hazardous waste sites. Am J Epidemiol 1992;135:1197-1207.
[3] ATSDR. Public health assessment for Raymark Industries Stratford, Farfield County, Connecticut. Atlanta, GA: US Dept. Health and Human Services, Agency for Toxic Substances and Disease Registry. 1996.
[4] Blot WJ, Fraumeni JF. Cancers of the lung and pleura. in Cancer Epidemiology and Prevention editors D Schottenfeld and JF Fraumeni. New York: Oxford University Press, 1996
[5] CDC. Occupational Respiratory Diseases. Atlanta, GA: US Dept Health and Human Services, National Institute for Occupational Safety and Health, September, 1986.
[6] Wyers H. Asbestosis. Postgrad Medical J 1949;25:631-638,.
[7] Wagner JC, Steggs CA, Marchand P. Diffuse Pleural Mesothelioma and asbestos exposure in north western Cape Province. Br J Ind Med 1960;17:260-271.
[8] Mossman BT, Bignon MC, Corn M, Seaton A, Gee BL. Asbestos: scientific developments and implications for public policy. Science 1990;247:294-301.
[9] Peto J, Seidman H, Selikoff IJ. Mesothelioma mortality in asbestos workers: implications for models of carcinogenesis and risk assessment. Br J Cancer 1982;45:124-135.
[10] Parker SL, Tong T, Bolden S, Wingo PA. Cancer Statistics, 1996.CA Cancer J Clin1996;46:5-27.
[11] Devesa SS, Silverman DT, Young JL, Pollack ES, Brown CC, Horm JW, Percy CL, Hyers MH, McKay Fw, Fraumeni JF. Cancer incidence and mortality trends among whites in the United States, 1947-84. JNCI 1987;79:701-75.
[12] Silverman DT, Hartge P, Morrison AS, Devesa SS. Epidemiology of bladder cancer. Hematology/Oncology Clin North America 1992;6:1-30.
[13] Silverman DT, Levin LI, Hoover RN, Hartge P. Occupational risks of bladder cancer in the United States: I white men. JNCI 1989;81:1472-1480.
[14] Silverman DT, Levin LI , Hoover RN. Occupational risks of bladder cancer in the United States: II nonwhite men. JNCI 1989;81:1480-83.
[15] Morrison AS, Ahlbom A, Verhoek WG, Aoki K Leck I, Ohno Y, Obata K. Occupation and bladder cancer in Boston USA, Manchester, UK, and Nagoya Japan. J Epid Comm Health 1985;39:294-300.
[16] Silverman DT, Levin LI, Hoover RN. Occupational risks of bladder cancer among white women in the United States. Am J Epidiol 1990;132:453-461.
[17] Meigs W, Marrett LD, Ulrich FU, Flannery JT. Bladder tumor incidence among workers exposed to benzidine: a thirty-year follow-up. JNCI 1986;76:1-8.
[18] Rench JD, Ouellet-Hellstrom R, Garner RP, Griffith MA, Norton JJ. Cancer incidence study of workers handling mono- and di-arylamines including dichlorobenzidine, ortho-tolidine and ortho-dianisidine. SRA Technologies, Inc. 1995; Falls Church, Va.
[19] Silverman DT, Morrison AS, Devesa SS. Bladder cancer. in Cancer Epidemiology and Prevention editors D Schottenfeld and JF Fraumeni. New York: Oxford University Press, 1996
[20] Page T, Harris RH, Epstein SS. Drinking water and cancer mortality in Louisiana. Science 1976;193:55-57.
[21] Cantor KP, Hoover R, Mason TJ, McCabe LJ. Associations of cancer mortality with halomethanes in drinking water. JNCI 1978;61:979-985.
[22] Cantor KP, Hoover R, Hartge P, Mason TJ, Silverman DT, Altman R, Austin DF, Child MA, Key CR, Marrett LD, Myers MH, Naryana AS, Levin LI, Sullivan JW, Swanson GM, Thomas DB, West DW. Bladder cancer, drinking water source, and tap water consumption: a case-control study. JNCI 1987;79:1269-1279.
[23] McGeehin MA, Reif JS, Becher JC, and Mangione EJ. Case-control study of bladder cancer and water disinfection methods in Colorado. Am J Epidemiol 1993;138:492-501.

[24] Morris RD, Audet AM, Angelillo IF, Chalmers TC, Mosteller F. Chlorination, chlorination by-products, and cancer: a meta-analysis. Am J Public Health 1992;82:955-963.
[25] Mallin K, Investigation of a bladder cancer cluster in northwestern Illinois. Am J Epidemiol 1990;132:s96-s106.
[26] Forman D, Moller H. Testicular cancer. Cancer Surveys 1994;19/20:323-341.
[27] Buetow SA. Epidemiology of Testicular Cancer.Epidemiologic Reviews 1995;17:433-449.
[28] Manson JM, Wise DL. Teratogens. In Casarett and Doul's Toxicology. Amdur MO, et al., eds.. New York: Pergamon Press, 1991:233.
[29] Karmaus W, Wolf N. Reduced birthweight and length in the offspring of females exposed to PCDFs, PCP and lindane. Environ Health Persp 1995;103:1120-1125.
[30] XU X, Ding H, Wang X. Acute effects of total suspended particulates and sulfur dioxides on preterm delivery: a community-based cohort study. Arch Environ Health 1995;50:407-415.
[31] Schardein, JL Chemically induced birth defects. New York: Marcel Dekker, Inc., 1993.
[32] Romw WN. Effects of lead on the female and reproduction: A review. Mt Sinai J Med 1976;43:542-552.
[33] Nordstrom S, Beckman L, Norensons I. Occupational and environmental risks in and around a smelter in Northern Sweeden. V. Spontaneous abortion among female employees and decreased birth weight in their offspring. Hereditas 1979;90:291-296.
[34] McMichael AJ, et al. The Port Pirie cohort study: Maternal blood lead and pregnance outcome. J Epidemiol Community Health 1986;40:18-25.
[35] Murphy MJ, et al. Past pregnancy outcomes among women living in the vicinity of a lead smelter in Kosovo, Yugoslavia. Am J Public Health 1990;80:33-35.
[36] Factor-Litvak P, Graziano JH, Kline JK, Popovak D, Mehmeti A Ahmed G et al., A prospective study of birthweight and length of gestation in a population surrounding a lead smelter in Kosovo, Yuglslavia. Int J Epidemiol 1991;20:772-728.
[37] Kuratsune M, et al. Past pregnancy outcomes among women living in the vicinity of a lead smelter in Kosovo, Yugoslavia. Int J Epidemiol 1991;20:772-728.
[38] Fein GG, Jacobson JL, Jacobson SW. Prenatal Exposure to Polychlorinated Biphenyls: Effects on Birth Size and Gestational Age. J Pediatrics 1984;105:315-320.
[39] Rogan WJ, Galden BC, McKinney JD, Polychlorinated Biphenyls (PCBs) and Dichlorodiphenyl Dichloroethane (DDE) in Human Milk: Effects on Growth, Morbidity, and Duration of Lactation. Am J Public Health 1987;77:1294-1297.
[40] Berkowitz GS, Lapinski RH, Wolff MS. The role of DDE and polychlorinated biphenyl levels in preterm birth. Arch Environ Contamination and Toxicology 1996;30:139-141.
[41] Spencer F. An Assessment of the Reproductive Toxic Potential of Aroclor 1254 in Female Sprague-Dawley Rats. Bull Environ Contamination and Toxicology 1982;28:290-297.
[42] ATSDR. Toxicological Profile for Polychlorinated Biphenyls, Atlanta, GA: US Dept. Health and Human Services, Agency for Toxic Substances and Disease Registry, 1993 pp. 53-56.
[43] National Research Council (U.S.) Committee on the Assessment of Polychlorinated Biphenyls in the Environment, Polychlorinated biphenyls: a report, Washington, D.C.: National Academy of Sciences, 1979.
[44] McCormick MC. The contribution of low birth weight to infant mortality and childhood morbidity. N Engl J Med 1985;312:82-90.
[45] Shapiro S, McCormick MC, Starfield BH, Krischer JP, Bross D, Relevance of correlates of infant deaths for significant morbidity at 1 year of age. Am J Obst Gyn 1980;136:363-373.
[46] Buehler JW, Strauss LT, Hogue CJ, Smith JC, Birth weight-specific causes of infant mortality, United States, 1980. Public Health Reports. 1987;102:162-71.
[47] CDC. From the Centers for Disease Control and Prevention., Infant mortality--United States, 1993. JAMA 1996;275:980.
[48] CDC. Increasing incidence of low birthweight--United States, 1981-1991. Morbidity and Mortality Weekly Report 1994;43:335-9.
[49] Christianson RE, Van den Berg BJ, Milkovich L, Oechsli FW. Incidence of congenital anomalies among white and black live births with long-term follow-up. Am J Public Health 1981;71:1333-1341.

[50] McCall MG, Acheson ED, Respiratory disease in infancy. J Chronic Disease 1968;21:349-359.
[51] Rawlings JS, Smith FR, Garcia J, Expected duration of hospital stay of low birth weight infants: graphic depiction in relation to birth weight and gestational age. J Pediatrics 1993;123:307-309.
[52] Yuksel B, Greenough A, Birth weight and hospital readmission of infants born prematurely. Arch Pediatric Adolescent Medicine 1994;148:384-388.
[53] McCormick MC. Workman-Daniels K, Brooks-Gunn J, Peckham GJ. Hospitalization of very low birth weight children at school age. J Pediatrics 1993;122:360-3655.
[54] Cronin CM, Shapiro CR, Casiro OG, Cheang MS, The impact of very low-birth-weight infants on the family is long lasting. A matched control study. Arch Pediatric and Adolescent Med 1995;149:151-158.
[55] ATSDR, Public Health Assessment Guidance Manual, Chelsea, MI: Lewis Publishers, 1992
[56] Linos A, Blair A, Gibson RW, Evertt G, Ban Lier S, Cantor KP, Schuman L, Burmeister L. Leukemia and non-Hodgkin's lymphoma and residential proximity to industrial plants. Arch Environ Health 1991;46:70-74.
[57] Najem G, Thind I, Lavenhar M, Louria D. Gastrointestinal cancer mortality in New Jersey counties, and their relationship with environmental variables. Int J Epidemiol 1983;12:276-289.
[58] Griffith J, Duncan R, Riggan W, Pellon A. Cancer mortality in U.S. counties with hazardous waste sites and groundwater pollution. Arch Environ Health 1989;44:69-74.
[59] Mallin K. Investigation of a bladder cancer cluster in northwestern Illinois. Am J Epidemiol 1990;132:s96-s106.
[60] Shaw GM, Schulman J, Frisch JD, Cummins SK, Harris JA. Congenital malformations and birthweight in areas with potential environmental contamination. Arch Environ Health 1992;47:147-154.
[61] Goldberg MS, Al-Homsi N, Goulet L, Riberdy H. Incidence of cancer among persons living near a municipal solid waste landfill site in Montreal, Quebec. Arch Environ Health 1995;50:416-424.
[62] Goldberg MS, Goulet L Riberdy H Vonvalot Y. Low birth weight and preterm birth among infants born to women living near a municipal solid waste landfill site in Montreal, Quebec. Environ Res 1995;69:37-50.
[63] Janerich DT, Burnett WS, Feck G, Hoff M, Nasca P, Polednak AP, Greenwald P, Vianna N. Cancer incidence in the Love Canal area. Science 1981;212:1404-1407.
[64] Janerich DT and Flannery J. Technical Proposal. Connecticut Tumor Registry. Contract No. N01-CN-55422, Hartford, CT: June 1987.
[65] Connecticut Tumor Registry (CTR). Tabular SEER computer printout. Hartford, CT: 1995
[66] Zippin C, and Lum D. Completeness of Case Reporting on 1987 Cancer Diagnoses Surveillance, Epidemiology and End Results (SEER) Program. San Francisco, CA: Unpublished. 1989.
[67] Flannery JT. Director, Connecticut Department of Public Health, Tumor Registy, Personal interview. 22 February 1995
[68] United States Department of Commerce (USDOC), United States Census Bureau. Geographic Areas Reference Manual. November, 1994
[69] Environmental Systems Research Institute, Inc. Understanding GIS. Redlands, CA: ESRI, 1990.
[70] Razavi, Amir H. ArcView: Developer's Guide. Santa Fe, New Mexico: On Word Press, 1995.
[71] Morrill RL., Kelly MB. The Simulation of Hospital Use and the Estimation of Location Efficiency, Geographical Analysis 283-300.
[72] Lowe J, Pederson E. Human Geography, An Integrated Approach. New York: John Wiley & Sons, 1983.
[73] Rothman KJ. Modern Epidemiology. Boston: Little, Brown and Company, 1986.
[74] Breslow NE,. Day NE. Statistical Methods in Cancer Research: Volume II - The Design and Analysis of Cohort Studies. Oxford, UK: Oxford University Press, 1987
[75] Mausner JS, Kramer S. Epidemiology - An Introductory Text. Philadelphia: W. B. Saunders Co., 1985.
[76] Daniel WW. Biostatistics: A foundation for anaylsis in the health sciences. New York: John Wiley & Sons, Inc., 1987.
[77] McClave JT, Dietrich FH. Statistics. San Francisco: Dellen Publishing Co.,1988.
[78] Stokes, ME Davis, CS, and Koch, GG, Categorical Data Analysis Using the SAS System, SAS Institute, Cary, NC, 1995.

[79] CDC. From the Centers for Disease Control and Prevention., Differences in infant mortality between blacks and whites--United States, 1980-1991. JAMA 1974;271:1570.

[80] Kempe A, Wise PH, Barkan SE, Sappenfield WM, Sachs B, Gortmaker SL, et al., Clinical determinants of the racial disparity in very low birth weight. New Engl J Med 1992;327(14):969-973.

[81] Starfield B, Shapiro S, Weiss J, Liang KY, Ra K, Paige D, et al., Race, family income, and low birth weight. Am J Epidemiol 1991;134:1167-1174.

[82] Schulman J, Selvin S, Shaw GM, Malcoe LH, Exposure misclassification due to residential mobility during pregnancy in epidemiologic investigations of congenital malformations. Arch Environ Health 1993;48:114-119.