LUNG CANCER INCIDENCE AND SOCIOECONOMIC STATUS IN PORTLAND, OREGON AND VANCOUVER, WASHINGTON

by

Charlene Bryan

A MASTER'S THESIS

Presented to the Department of Public Health
and Preventive Medicine and
Oregon Health Sciences University
School of Medicine
in partial fulfillment of the requirements
for the degree
of

Masters in Public Health

May 1999

School of Medicine
Oregon Health Sciences University

CERTIFICATE OF APPROVAL

This is to certify that the Master's thesis of

CHARLENE BRYAN

has been approved

[redacted]
Professor in charge of thesis

[redacted]
Member

[redacted]
Member

[redacted]
Member

[redacted]
Associate Dean for Graduate Studies

TABLE OF CONTENTS

TABLE OF FIGURES

LIST OF TABLES

LIST OF EQUATIONS

LIST OF APPENDICES

ACKNOWLEDGEMENTS

The author wishes to thank the members of her thesis committee: Dr. William Morton, Dr. Jodi Lapidus, Dr. Donald Freeborn and Dr. Kristine Wiren for their assistance in the development of this thesis. Thanks are also given to Dr. William Morton and Dr. David Phillips who performed the data collection. Several others have provided invaluable assistance with data management and statistical analysis, F. Keith Barker, Michael Lasarev, Jeffrey Capezzi, Michael Jordan, Dr. Joseph Istvan and Dr. Linda McCauley. Thanks are also given to her family and friends for their support.

ABSTRACT

The relationship between lung cancer incidence by histology and socioeconomic status was examined in men and women in the Portland, Oregon-Vancouver Washington SMSA between 1963 and 1977. Socioeconomic status was based on a scale that utilized education, income and housing density of census tract of residence as of the 1970 census. Because of its role as a possible confounder, occupation was not included in the assessment of socioeconomic status in this analysis. Lung cancer incidence was found to be higher in males. The three histologic groups, squamous cell, adenocarcinoma and undifferentiated small cell lung cancer were the most prevalent histologic types. The proportion of squamous cell lung cancer was highest in males at 35%. Adenocarcinoma was the most prevalent type of lung cancer among females, with 23% cases presenting. Lung cancer incidence was found to be inversely correlated with socioeconomic status in males, but did not vary by this index in females

INTRODUCTION

Lung cancer incidence and mortality have risen in an epidemic pattern in the United States and other developed countries since the 1940s and 1950s. At the beginning of the 20th century lung cancer was a relatively rare disease, with 7 deaths being reported by Oschner and DeBakey (1939) in New Orleans, Louisiana. As we approach the close of the 20th century, approximately 159,000 deaths are estimated to occur from lung cancer in 1999 in the United States alone (Cancer Statistics, 1999).

Eighty-three percent of all lung cancer deaths were directly attributed to smoking in 1990 (CDC, 1996; NCHS, 1993). Smoking is linked to all types of lung cancer (Wallace, 1998), but most strongly to squamous cell cancer and undifferentiated small cell cancer. Blot (1996) reported a dose-response relationship for adenocarcinoma and smoking. The CDC has shown that smoking prevalence decreased steadily at an average of 0.5% per year from 1964[1] to the late 1980s (Wallace, 1998). In the 1990s, smoking prevalence has remained essentially the same (Giovino et al., 1994; CDC, 1997). This is of some concern, especially as the 1995 National Health Interview Survey (NHIS) has indicated that approximately twenty-five percent of adults (>18 years of age) were reported to be current smokers and the latency period for lung cancer is between 20-30 years.

This stasis in the reduction of smoking prevalence is disturbing as it may be the beginning of an increase in the prevalence of smoking. It is important to

---

[1] In 1964, the first Surgeon General's Report summarizing the existing evidence on smoking and declaring cigarette smoking to be the major cause of lung cancer among American men.

examine the differences in the reduction of smoking prevalence by the epidemiological factors of age, sex, ethnic group, occupation, secular trends, i.e., year of diagnosis, socioeconomic status, and education (Wallace, 1998). These epidemiological factors have known impacts on the incidence of lung cancer, i.e., lung cancer incidence increases with age. They will also provide valuable background information into the design of intervention programs.

The epidemiological factors of age, sex, ethnic group, occupation and socioeconomic status all greatly impact the histological type of lung cancer. Among cell types different age and sex patterns of incidence may also point to differences in etiology. The incidence data on different histological types of lung cancer could impact the importance of a variety of epidemiological factors, for example, social class variations, secular trends and occupation. This was illustrated by Lubin and Blot in 1984 who found sex differences in histological cell type incidence that could not be explained by smoking history alone. Against this backdrop of increasing lung cancer incidence (SEER[2] data, 1973-1977) (Appendix A1) and the reduction of smoking prevalence (Appendix A2), this analysis examines the impact of socioeconomic status on lung cancer incidence by histology in the Portland, Oregon-Vancouver, Washington Standard Metropolitan Statistical Area between 1963 and 1977.

---

[2] Surveillance, Epidemiologic End Results -a program of the National Cancer Institute.

Lung cancer incidence

Lung cancer was a relatively rare cause of death from the 1920s to 1940s. In the United States during the 1950s, it became the leading cause of cancer mortality among Caucasian, middle-aged (45-54 years of age) males (Samet, 1992). In 1964, the observed male-to-female ratio of death rates was 7:1 (Samet, 1992). Lung cancer mortality has increased dramatically in women and in the late 1980s, lung cancer mortality surpassed that observed for breast cancer (Carney and Leij, 1988; Ianuzzi and Scoggin, 1986). The mortality rate ratio for males to females has fallen to 2:1 (SEER 1994 data), with lung cancer mortality for females approximating that observed for males approximately 30 years ago (Wallace, 1998).

In 1999, it has been estimated that about 1,221,800 cases of invasive cancer will be diagnosed in the United States alone, with approximately 14% of it being attributed to lung cancer (171,600 cases) (Cancer Statistics, 1999). SEER data indicated an observed incidence rate for lung cancer at 56 per 100,000[3] for 1990-1995 (Appendix A1). It is estimated that 158,900 deaths from lung cancer will also be observed in 1999. This will give rise to an observed mortality rate of 50 per 100,000 population[4].

Among males, the most commonly observed cancers are expected to be prostate, lung and bronchus, colon and rectum. Cancers of the breast, lung and bronchus and colon and rectum are expected to be the three most commonly observed cancers among women. These cancers in women are expected to

---

[3] All rates are age-adjusted to the 1970 United States population.
[4] Mortality data are from the Vital Statistics of the United States, 1998 for all states except Connecticut, Oklahoma, Louisiana and New Hampshire.

account for over 50% of the cancers observed in females in 1999.

The annual lung cancer incidence rate for men is beginning to decline from its peak at 87 per 100,000 in 1984 to 74 per 100,000 as observed in 1994 (SEER data) (Appendix A2). It was observed that during 1990-1995 incidence rates decreased significantly by 2.3% per year (Cancer Statistics, 1999). The incidence rate for females was observed to peak at 43 per 100,000 in 1991 and it appears that the rate of increase of incidence in the female cases is beginning to stabilize (Cancer Statistics, 1999). 1994 SEER data has also reported a lung cancer incidence rate of 43 per 100,000 in females (Appendix A4). Similar decreases in lung cancer mortality have been observed among males, with an average steady decrease of about 1.6% per year during 1990-1995, (Cancer Statistics, 1999). The mortality trend among females also mirrors the incidence trend, as rates have begun to slow and appear to be stabilizing (Appendix A4).

This stabilization and downturn in lung cancer rates can be largely attributed to the general decrease in smoking prevalence that has been observed nationally among adults (Appendix A2). Lung cancer has long been strongly associated with smoking (Engholm et al., 1996; Muscat and Wynder, 1995; Doll et al., 1980; Hammond, 1966; Doll and Hill, 1952; Doll and Hill, 1950; Wynder and Graham, 1950; Levin, 1950). It has also been associated with certain occupations, for example, the increased incidence of small cell cancer in uranium mining and plastics production (Sankila, 1990; Maher, 1987; Weiss, 1979; Figueosa, 1973). Early screening and improved treatment have also helped improve the relative 5-year survival rates from 12% (1974-1976) to 14%, (1989-1994), (Cancer Statistics, 1999). Despite the numerous surgical and therapeutic advances being made,

there has not been a significant impact on the survival rates, for the overall prognosis is still poor (Friedberg and Kaiser, 1997).

Ethnic and racial variations in lung cancer

Cancer incidence rates vary across different ethnic and racial groups. The highest incidence rates have been observed among African-Americans in the US (Appendix A5). The Third US National Cancer survey reported that mortality rates have been higher among African-American males than among Caucasian males since the 1970s (Appendix A5a, A5b and A5c). During 1973-1977, lung cancer mortality rates for African-American males increased from, 75.1 per 100,000 (1973) to 88 per 100,000 (1977). The observed mortality rates for Caucasian males ranged from 62 per 100,000 to 67 per 100,000 (1977).

The mortality rates observed for Caucasian and African-American females were similar during 1973-1977. African-American females experienced mortality rates ranging from 13.6 per 100,000 in 1973 to 17.3 per 100,000 in 1977 (NCI, 1994). Caucasian females also experienced similar mortality rates between 1973 and 1977 (NCI, 1994) (Appendix A5a, A5b, A5c & A6). African-American and Caucasian women have also continued to experience similar incidence and mortality rates (Cancer Statistics, 1999).

SEER incidence data (1990-1995) has indicated a lung cancer rate of 78 per 100,000 for African-Americans (Appendix A7). They are twice as likely to develop lung cancer as are Asian/Pacific Islanders (36 per 100,000) or Hispanics (28 per 100,000). They are also approximately four times as likely to develop lung cancer as are American Indians (19 per 100,000). Native Americans have traditionally had a lower incidence of lung cancer than the rest of the United

States (Blot, 1996). Age adjusted rates for lung cancer mortality for Native Americans in New Mexico showed a steady increase between 1963 and 1977 (Samet, 1988). The following rates were observed: 1) 4.7 per 100,000 (1963-1967), 2) 9.0 per 100,000 (1968-1972) and 7.7 per 100,000 (1973-1977).

During 1990-1995, the incidence rates for lung cancer decreased slightly among Hispanics and Caucasians. Lung cancer incidence rates remained stable among African-Americans (Cancer Statistics, 1999). This has also been observed among Asian/Pacific Islanders. Lung cancer incidence appears to be increasing slightly among Native Americans (Cancer Statistics, 1999). African-American men have been observed to have the highest incidence rates of lung and bronchus cancers (Appendix A7).

Mortality data for 1990-1995 has reported a rate of 61 per 100,000 for African-Americans. This exceeds that reported for Caucasians at 49 per 100,000. The African-American mortality rate is more than double the rate observed for Asian/Pacific Islanders at 24 per 100,000, for American Indians at 28.5 per 100,000, and for Hispanics at 20 per 100,000, (Cancer Statistics, 1999).

Lung cancer and histology

The term "lung cancer" should strictly refer to all malignancies that arise in the lung, but in practice it usually refers to the "bronchogenic" carcinomas. This term is inaccurate but it is useful in denoting the four major histological types of lung cancer. These four histopathological types are squamous cell carcinoma, adenocarcinoma, undifferentiated small cell cancer and undifferentiated large cell cancer. These cancer types are extremely common and generally associated with cigarette smoking (Kreyberg, 1962). Ninety percent of

all malignant lung tumors belong to these four major cell types (Wallace, 1998).

Kreyberg, in 1962, proposed a classification of lung tumors into 2 groups determined by morphological characteristics of the different cell types. Group I tumors were squamous and undifferentiated small cell carcinomas located centrally in the respiratory tract and Group II tumors were located in the periphery of the lung and were undifferentiated large cell carcinomas and adenocarcinomas. In 1971, Kreyberg proposed that Group I tumors were related to smoking, and exposure to ionizing radiation and nickel (Kreyberg, 1978).

In 1972 and 1977, Weiss et al., refuted Kreyberg's assumption of little or no causal relationship between smoking and adenocarcinomas. They concluded that adenocarcinomas developed among smokers in a dose-related fashion. This was also shown by Brownson et al., in 1987. They conducted a case control study in Denver, Colorado during 1979-1982. From this they were able to determine determined that prior cigarette use was the most significant predictor of adenocarcinoma risk among both males and females.

Undifferentiated small cell carcinoma and adenocarcinoma incidence have been steadily increasing, whilst squamous cell carcinoma has been on the decrease between 1969 and 1987 (Appendix A8). These three histological cell types have emerged as the most prevalent.

The 1973-1977 SEER data showed that in males there were twice as many cases of squamous cell carcinomas as adenocarcinomas among males. Adenocarcinoma was observed to be slightly higher for females when compared to males (Young, 1981). This is phenomenon that has been shown repeatedly by epidemiologists in the US and also internationally. Shimizu et al., in 1982 studied the epidemiology of adenocarcinoma in Los Angeles County, California

during 1972-1976. They observed that cigarette smoking was responsible for 90% of squamous cell cancer incidence in both sexes, 79% of male and 46% of female adenocarcinoma. Wu et al., in 1986 reported on secular trends in histologic types of lung cancer in Los Angeles County, California during 1972-1981. There was an average annual increase of 2.9% in the incidence rate of adenocarcinoma in males in Los Angeles County, California, and an annual 5.5% increase in the incidence rate of undifferentiated small cell cancer in females. Modan (1978), in a nationwide study of lung cancer during 1968-1970 in Israel, observed that adenocarcinoma was the most prevalent histologic cell type in women at 1.6 per 100,000. Squamous cell lung cancer had the highest incidence in men at 4.0 per 100,000.

A French case-control study (Benhamou et al, 1987) among French female smokers examined the effects of different smoking habits on lung cancer cell type. Fifty-two percent of all the cancer found was in non-smokers and approximately seventy-one percent of the adenocarcinoma cases were found in non-smokers. Dodds et al., (1986) reviewed SEER data (1974-1981) for western Washington state. They showed that incidence of adenocarcinoma has increased significantly within each sex since 1974. The incidence rate of adenocarcinoma has increased by 86% in females and by 54% in males during 1974-1981.

A comparison of the SEER data from (1983-1987) and the 1969-1971 Third National Cancer Survey (Samet, 1994) (Appendix A8), has shown an increase in the incidence of small cell cancer in all females from 14.5 % to 20.2%. Squamous cell carcinoma has decreased from 21.5% to 18.9 % of observed female cases. Adenocarcinoma has increased substantially from 23.1% to 29.4% in females. A similar pattern was observed in males (Samet, 1994) (Appendix A8).

All histological types of lung cancer in men appear to peak at approximately 70-74 years of age. In women, adenocarcinoma has been reported to peak at an earlier age, (50-59yr) followed by undifferentiated small cell cancer at 60-69 years, then squamous carcinoma at 70-74 years of age (Greenberg et al., 1984).

Lung cancer and socioeconomic status

One of the most telling patterns of disease is that individuals in the lowest socioeconomic groups often have the highest morbidity and mortality. Socioeconomic characteristics are powerful determinants of disease incidence across all populations (CDC, 1998; Williams and Collins, 1995). Antonovsky (1967) noted that this pattern remains consistent globally, regardless of 1) the infectious or non-infectious nature of causal agents of disease and 2) the specific methods of ascertaining socioeconomic status, (Wallace, 1998). Socioeconomic status (SES) has the effect of grouping individuals and of nesting them within geographic, social and occupational environments.

Socioeconomic status has long been an important variable in a variety of health-related studies. Liberatos et al., in 1988, determined that almost forty percent of seventy-six studies on chronic diseases reported in the American Journal of Epidemiology utilized some measure of socioeconomic status. SES has been viewed as a potential confounder, risk factor and as a descriptive variable in epidemiological studies.

Lung cancer in men has been shown to be inversely correlated with socioeconomic status since the 1950s. Brown et al., (1975) examined mortality rates from Buffalo, New York among Caucasian males and females, 1959-1961.

Here lung cancer in men was shown to be inversely associated with economic class. For women, there was higher mortality in the upper economic classes, but the trend was not statistically significant. A series of subsequent studies have confirmed these findings (van Loon et al., 1995; Månsson, 1995; Hein, 1992; Samet 1992; Baquet et al., 1991; Vågeröet al, 1986; Teppo et al., 1984; Simpson and Comstock, 1983; Cuello et al., 1982; Williams and Horn, 1977; Wynder and Stellman, 1977).

The effect of socioeconomic status on lung cancer in women has been less clear cut and appears to be the result of an interaction of a variety of etiological factors (Samet, 1994), for example occupation or hormonal factors. Women also appear to have different susceptibilities to certain histologies of lung cancer than do men.

The association between smoking and socioeconomic status is different among men and women in the United States. In 1984, Rosenwaike reported on the changing patterns of lung cancer among socio-cultural groups in New York City. Socioeconomic status was ascertained utilizing level of education as the main indicator and study subjects were grouped by their religious affiliation. Rosenwaike utilized the 1963-1964 study conducted in New York City on educational attainment. Here both male and female Jews were found to have most likely attended or completed college, when compared to Catholics and Protestants. He showed that there were relatively low lung cancer rates among Jewish men, compared to Catholic and Protestant male groups. Jewish males were also found to smoke 40% less than their Protestant or Catholic counterparts. In contrast, Jewish women, experienced a higher lung cancer mortality rate than their Catholic and Protestant counterparts. This indicated that other factors

besides education were contributing to smoking habits in women.

Despite the fact that socioeconomic status has been shown to be associated with lung cancer, it is not thought to be a direct risk factor. SES is commonly regarded as a proxy for a variety of lifestyle variables, which have been identified as possible risk factors for lung cancer. These include smoking, dietary factors and occupational exposure to carcinogens. For example, Levi et al in 1988, proposed that differences in smoking prevalence were largely responsible for the observed differences in lung cancer risk found between different SES groups. Smoking in the lower SES groups has long been believed to be higher than that observed in the higher SES groups in the developed world (van Loon et al., 1995; Hein et al., 1992). The assumption can be made that persons of high SE status are more educated about the hazards of smoking and have more to lose from premature death.

Based on this medical literature review, the following points have emerged: 1) there appears to be consensus in the medical literature that socioeconomic status is inversely related to lung cancer in males and 2) the relationship between SES and lung cancer incidence in females appears to be less clear. Given that SES is regarded as a proxy for lifestyle variables that are known risk factors for lung cancer incidence, we propose to utilize a new composite measure of socioeconomic status. This measure excludes occupation because of its role as a confounder. The following research hypotheses were proposed and tested:

1. There was no relationship between SES and mean annual age-standardized total lung cancer incidence ratio (SIR) in the Portland-Vancouver metropolitan area during 1963-1977.

2. There was no relationship between SES and mean annual age-standardized lung cancer cell type-specific incidence ratios (SIR) in the Portland-Vancouver metropolitan area during 1963-1977.

METHODS

Data collection

Cases of primary pulmonary and pleural neoplasms were identified by searches of the medical records of the 24 hospitals within the Portland-Vancouver Standard Metropolitan Statistical Area (SMSA). The tertiary medical care center for the state of Oregon is located in Portland. Cases were referred from the Vancouver metropolitan area of Washington State and from the entire state of Oregon to Portland. It was extremely rare for cases to go elsewhere for diagnosis and treatment.

All patients, who received a diagnosis of lung cancer and satisfied residency requirements in the Portland-Vancouver SMSA at the time of diagnosis, during the period 1963-1977, were included in the study. Case ascertainment was analagous to that of the National Cancer Institute SEER program (Percy and Sobin, 1983). The primary data sources for this study were:

1. A medical chart review of lung cancer diagnoses in the 24 hospitals in the Portland, OR, and Vancouver, WA metropolitan area.
2. A review of lung cancer death certificates for residents of the Portland, OR, and Vancouver, WA metropolitan area.
3. Socioeconomic status (SES) as determined from a summary score for census tract of residence indirectly using four census tract characteristics, according to the patient's address at the time of diagnosis.
4. Census tract population and socioeconomic characteristic informationobtained from the 1970 US Census.

All diagnoses of lung cancer during 1963-1977 for residents of the Portland-Vancouver metropolitan area were included in the study. The records were reviewed by a team of medical students under the direct supervision of Dr. William Morton, the principal investigator. In each hospital, the principal investigator compiled a list of all the cases of lung cancer to be investigated.

Tumor registries, hospital discharge diagnoses and tissue pathological report files were utilized wherever available. For each case found and identified by the above methods, the hospital medical records were found and reviewed extensively. Histological slides were accepted as listed on the chart or pathology report and classified according to the WHO classification system suggested by Kreyberg, 1967.

A total of seventy-two histological codes were utilized to characterize the observed lung cancer cell types and their combinations (Appendix B1). No independent slide reviews were conducted. This meant that the data would include some degree of histological inconsistencies and error, but it is representative of the information available to the health care practitioners within the community at that time (Morton and Treyve, 1982).

Cases which manifested more than one primary lung neoplasm cell type were counted in the rates for each cell type, but only once in the rates for all lung cancer. This phenomenon would tend to minimize observed differences among cell type rates. Twenty-two major histogroups (Appendix B2) (including an overall observation of lung cancer incidence) were developed utilizing the WHO classification from the individual histocodes. These groups were not mutually exclusive.

Investigators were only allowed access to hospital records on the condition that the patients or families were not to be contacted. Thus case records were abstracted, alphabetized and collated into a master file to avoid duplication, as individuals who were seen at more than one hospital were collated into a single record (Morton and Phillips, 1983). In order to enhance the completeness of the lung cancer data from the hospital records, death certificates

were obtained from the Oregon State Health Division—for the residents of the three counties that comprise the Portland metropolitan area, i.e., Multnomah, Clackamas and Washington—and from the Washington State Health Division, for the residents of the single county that comprises the Vancouver metropolitan area, i.e., Clark county. An additional 10% of cases were discovered by the use of death certificates. Hospital records were then abstracted for these cases. Death certificates contributed the following information to the lung cancer study, i.e., the cause of death (Morton and Phillips, 1983).

The census tract information was required for coding census tract of residence at time of diagnosis. The census tract code used the 1970 US census tract numbers and boundaries, and their identification utilized the metropolitan census tract street index, telephone books, city and county directories, maps and postal route maps (Morton and Phillips, 1983).

Quality control

The medical abstracts were checked for completeness by the principal investigator. Incomplete abstracts tended to cluster by hospital. They were compiled, and the hospital charts were reviewed again. Incomplete abstracts were usually the result of missing information in the hospital charts, or the abstractors had overlooked information. Funding was unavailable to conduct a systematic verification of a sample of apparently complete abstracts (Morton and Phillips, 1983).

The coding was checked by different individuals than the original coders. Coded data was punched into IBM cards, and the punching was verified, and tabulations were done with an IBM card sorter. Initial tabulations of

identification number, age-groups, and census tract groups provided any additional opportunity for verification of data accuracy. An exact estimate of the ultimate error rate for the tabulated data was unavailable, but was believed to be extremely small, (Morton and Phillips, 1983).

Data management

Data was transferred from the IBM punch cards to an electronic form for data manipulation. Data management was handled in the Microsoft Excel™ 1998 program. Descriptive frequencies were performed in SPSS™ in order to identify any possible outliers, miscoding errors and errors due to the data transfer. Cross-tabulations were performed in Access™ in order to stratify the data by sex, age, diagnosis year and census tract. The data (all observed cases) was then organized into 22 histologic groups in preparation for the calculation of the dependent variable.

The unit of analysis

The unit of analysis for the study was the 1970 geographic, residential census tract, as this was the midpoint of the study timeline. The SMSA contained 265 census tracts, 35 of which were in Clark county, Washington (Appendix B3). The remaining 230 census tracts were divided between the three counties which comprise the Portland, Oregon SMSA, Multnomah (150), Clackamas (43) and Washington (37). Of the 265 census tracts, 45 were designated rural and 220 were urban.

The independent variable

The main independent variable for this study was socioeconomic status (as determined by the SES score. The SES score was determined for an individual census tract. The risk of lung cancer has been believed to be influenced by the SES of the host/subject (van Loon et al., 1995; Månsson, 1995; Hein, 1992; Samet 1992; Baquet et al., 1991; Vågerö et al., 1986; Teppo et al, 1984; Williams and Horm, 1977; Wynder and Stellman, 1977; Wynder and Doll, 1950; Doll and Hill, 1950). The SES of the case was estimated indirectly, based on the socioeconomic characteristics of the census tract of residence at the time of diagnosis. This assumed that persons of similar SES lived near one another.

SES was estimated by stratifications of a continuously distributed numerical score which was the sum of scores of the four census tract characteristics: 1) median family income, 2) percent of families below poverty level, 3) percent of high school graduates among persons aged 25 or older, and 4) percent of occupied housing units containing 1.01 or more persons per room.

The SES ranged from 20 to 130, where a higher score indicated a lower socioeconomic status. There was a smaller range of socioeconomic scores within the rural tracts, where scores ranged form 30 to 89. Urban tracts (n=220) had a range of SES from 20 to 130.

A secondary independent variable, Urbanicity, was utilized in order to describe the census tracts as either urban (coded as 1) or rural (coded as 0). This was necessary for the implementation of the multiple regression analysis, where the relationship between lung cancer incidence and socioeconomic status in the urban and rural areas is examined separately.

The dependent variable

The summary dependent/outcome variable was the mean annual age-standardized incidence ratio (SIR) of lung cancer (all cell types) within the individual census tract and within designated census tract strata in the Portland-Vancouver metropolitan area. Secondary outcome variables were the mean annual age-standardized incidence ratio for each designated lung cancer cell type, within the individual census tract and within designated census tract strata in the Portland-Vancouver metropolitan area. The dependent variables were also calculated for separately for males and females.

This study was a total community risk estimation. The primary estimate of cancer risk was the Standardized Incidence Ratio (SIR). The lung cancer data extended over a 15-year period, 1963-1977, so the total number of cases or deaths during this time period was transformed into mean annual numbers. The age-standardized incidence ratios (SIRs) for different lung cancer cell types—including individuals with multiple histologic cell types)—was determined for each census tract stratum and each individual census tract. Mean annual age-standardized incidence ratios (SIRs) were on the 1970 population (US Census, 1970); as 1970 was the mid-year of 1963-1977.

The standardized incidence ratio for total lung cancer and lung cancer by histologic group was calculated by the indirect standardization method, using the age distribution of the exposed (at risk) population within the Portland, Oregon and Vancouver, Washington Standard Statistical Metropolitan Area during 1970.

Calculation of the dependent variable —SIR

The SIR for a given census tract was calculated in Excel™ spreadsheets as follows:

(a) The 1970 Census divided the Portland-Vancouver population into eight age groups, i.e., 0-14, 15-24, 25-34, 35-44, 45-54, 55-64, 65-74, 75 and older. These age groups were used to stratify the observed cases. For a given census tract, an age-specific rate was developed for each age group for all 22 histogroups.

Equation 1. Calculation of age-specific lung cancer rates.

$$\text{Age specific rate for a given age group} = \frac{\text{Observed cases in age group}}{\text{Total population within the age group}}$$

(b) The age specific rate was then applied to each age group of the exposed population at risk within the 1970 SMSA. This produced an expected number of cases within each age group of the exposed population for every census tract.

Equation 2. Calculation of expected numbers of cases of lung cancer in each age group for a given 1970 US census tract in the Portland-Vancouver SMSA.

$$\begin{array}{l}\text{Expected number of cases} \\ \text{for an age group of the} \\ \text{exposed population}\end{array} = \begin{array}{l}\text{Age specific rate for} \\ \text{a given age group}\end{array} \times \begin{array}{l}\text{Exposed population at} \\ \text{risk within a given age} \\ \text{group}\end{array}$$

(c) For each census tract, the expected number of cases for each age group was summed to come up with a total expected number of cases across all age groups, for that census tract (E_T)

(d) For a given census tract:

Equation 3. Calculation of the standardized incidence ratio (SIR) for each 1970 US census tract in the Portland-Vancouver SMSA.

$$\text{SIR} = \frac{\text{Total observed cases in the census tract } (O_T)}{\text{Total expected cases in census tract } (E_T)}$$

(e) This process was repeated for each of the 265 census tracts and for each of the 22 histogroups.

Data analysis

The main objective of the statistical analysis was to determine the strength of the association between the mean annual standardized age-adjusted incidence ratios (SIRs) for total lung cancer (all types) by census tracts and different histologic types of lung cancers by census tracts and SES for each gender. Linear regression was the statistical method chosen to determine the strength of the association between the SIR and SES. In order to facilitate this, the statistical software package SPSS™ was used to perform all of the linear regression analyses.

Simple and multiple regression models were implemented in order to test the following null hypotheses:

1. There was no relationship between SES and mean annual age-standardized total lung cancer incidence ratio (SIR) in the Portland-Vancouver metropolitan area during 1963-1977.
2. There was no relationship between SES and mean annual age-standardized lung cancer cell type-specific incidence ratio (SIR) in the Portland-Vancouver metropolitan area during 1963-1977.

Before beginning the statistical analysis, the dependent variable or the Standardized Incidence Ratio (SIR) was first transformed utilizing the square root transformation. Skewness and kurtosis were checked for both male and female frequency distributions of total lung cancer both pre and post transformation to ensure a more nearly normal distribution (Appendix B3 and B4). The dependent variable was then referred to as the **Transformed SIR** and was utilized in this form in all subsequent statistical analyses.

The simple regression model (Equation 4) was utilized in order to determine the strength of the association between the lung cancer incidence and SES.

Equation 4. Simple regression model utilized in the analysis.

$$\text{Transformed SIR} = a + \beta(\text{SES Score})$$

The multiple regression model (Equation 5) was utilized in order to examine the effects of urbanicity and the interaction of urbanicity and SES (as represented by the SES Score) upon the relationship between lung cancer incidence and SES. Urbanicity was utilized as a secondary independent variable because of disparity between the number of urban (n=220) and rural (n=45) 1970 census tracts. This disparity may have obscured the contribution of the rural tracts to the regression analysis when the simple model was utilized.

Equation 5. Multiple regression model utilized for the data analysis.

$$\text{Transformed SIR} = a + \beta_1(\text{SES Score}) + \beta_2(\text{Urbanicity}) + \beta_3(\text{SES Score} * \text{Urbanicity})$$

Trends Analysis

The effect of time on the incidence of lung cancer was explored. The fifteen-year period was divided into three five year periods, 1963-1967, 1968-1972 and1963-1977. The midpoints of the first and third five year periods were 1965 and 1975 respectively. These were years during which US Census population estimates for the Portland SMSA were available. They were unavailable for Vancouver, Washington at those two time points. The 1970 US Census without Vancouver, Washington was utilized for the 1968-1972 period. SIRs were only calculated for the 230 US census tracts that comprised the Portland SMSA. All observed cases in Vancouver, Washington were excluded from this calculation.

SIRs were calculated for each of the 230 US census tracts during each of the three five year periods. A SIR was also calculated for each of the 22

histogroups. The General Linear Model for Repeated ANOVA[5] algorithm in SPSS™ was utilized. For this analysis, Time was the factor for the Repeated ANOVA analysis with three levels, 1,2, and 3. Level 1 referred to the first five year period, 1963-1967, level 2 referred to the second five year period, 1968-1972, etc. SES (as represented by SES Score) was evaluated as a covariate and Urbanicity was the "Between subjects" or grouping variable. The interaction term "SES Score*Urbanicity" was also evaluated in this model.

Data selection for reporting purposes

Three histologic groups of lung cancer were selected for reporting purposes from the 21 histologic groups along with the overall observation of total lung cancer incidence was also included. They were squamous cell carcinoma, adenocarcinoma[6] and undifferentiated small cell cancer. These three groups were selected because of support from the medical literature that indicated their emergence as the most prevalent histologic cell types of lung cancer observed (SEER Cancer Statistics Review, 1973-1994 data).

Simple and multiple regressions were performed for all 22 histologic groups. The trends analysis was only performed on for the overall observation of lung cancer and the squamous, adenocarcinoma and undifferentiated small cell histogroups.

---

[5] Analysis of Variance (ANOVA)

[6] The adenocarcinoma histologroup in this analysis does not include clear cell and alveolar cell lung cancer.

RESULTS

Demographic data

A total of 7087 cases of lung cancer were diagnosed between 1963-1977 in the 24 hospitals located in the Portland, Oregon and the Vancouver Washington Standard Metropolitan Statistical Area (SMSA). Of the 7087 diagnosed cases of lung cancer, 5485 were observed to be male and the remaining 1602 cases were females. A 3.4:1 ratio of all diagnosed male to female cases was observed to exist. There was a significant difference between the number of observed cases in the urban and rural census tracts. Overall 87% of all the lung cancer cases occurred in the urban census tracts. Approximately 15% of all the female cases were observed in the rural census tracts. Thirteen percent of the male cases occurred in the rural areas (Table 1).

Table 1. Distribution of lung cancer cases by sex and urbanicity in the Portland-Vancouver SMSA, 1963-1977.

Urbanicity	Female		Male		Total
	n	%	n	%	
Urban	1392	87	4762	85	6154
Rural	210	13	723	15	**933**
Total	**1602**	**100**	**5485**	**100**	**7087**

There is a general increase in the number of observed cases in both sexes from 1963-1977, (Figure 1). In 1977, the total number of observed cases was approximately twice the number diagnosed in 1963 (Table 2). Female lung cancer cases in 1977, were almost five times larger the number observed in 1963. The number of male cases increased by almost twice the number observed in 1963.

Table 2. Lung cancer cases by gender and year of diagnosis in the Portland –Vancouver SMSA, 1963-1977.

Diagnosis year	Female	Male	Total Cases
1963	43	292	335
1964	56	276	332
1965	60	308	368
1966	59	301	360
1967	81	309	390
1968	88	280	368
1969	105	370	475
1970	87	358	445
1971	104	379	483
1972	112	388	500
1973	120	395	515
1974	139	437	576
1975	172	453	625
1976	181	467	648
1977	195	472	667
Total	**1602**	**5485**	**7087**

Figure 1. Male and female cases of lung cancer in the Portland, Oregon-Vancouver, Washington, 1963-1977.

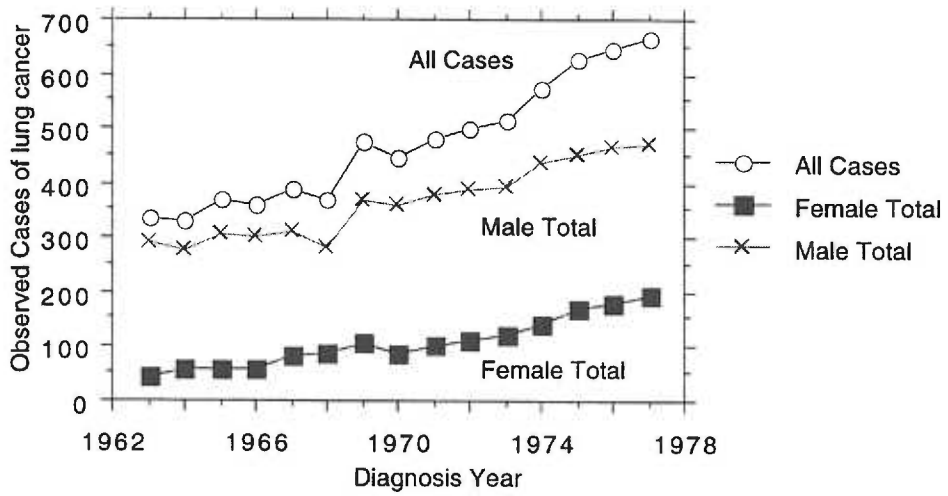


Table 3 depicts the observed cases of lung cancer stratified by diagnosis year and urbanicity. Once more, the general upward trend was maintained by both genders (Figure 2). This representation of the data clearly shows that the majority of the cases are derived from the male urban census tracts. Female rural cases constitute the smallest fraction of observed cases over the fifteen-year period in the Portland, Oregon-Vancouver, Washington SMSA (Appendix C1a and C1b).

Table 3. Lung cancer cases by gender, diagnosis year and urbanicity in the Portland-Vancouver SMSA, 1963-1977.

	Female			Male		
	Total cases	**Urban**	**Rural**	**Total cases**	**Urban**	**Rural**
1963	**43**	40	3	**292**	258	34
1964	**56**	47	9	**276**	256	20
1965	**60**	58	2	**308**	277	31
1966	**59**	53	6	**301**	267	34
1967	**81**	70	11	**309**	278	31
1968	**88**	72	16	**280**	243	37
1969	**105**	94	11	**370**	326	44
1970	**87**	76	11	**358**	315	43
1971	**104**	96	8	**379**	332	47
1972	**112**	97	15	**388**	334	54
1973	**120**	105	15	**395**	319	76
1974	**139**	120	19	**437**	371	66
1975	**172**	147	25	**453**	381	72
1976	**181**	151	30	**467**	403	64
1977	**195**	166	29	**472**	402	70
Total	**1602**	**1392**	**210**	**5485**	**4762**	**723**

The observed lung cancer cases were stratified by sex and ethnic group for the fifteen-year period (Table 4). African American males had the highest observed incidence rate of lung cancer for the fifteen-year period at 90.90 per 100,000 population. The incidence rate for Caucasian males (also included

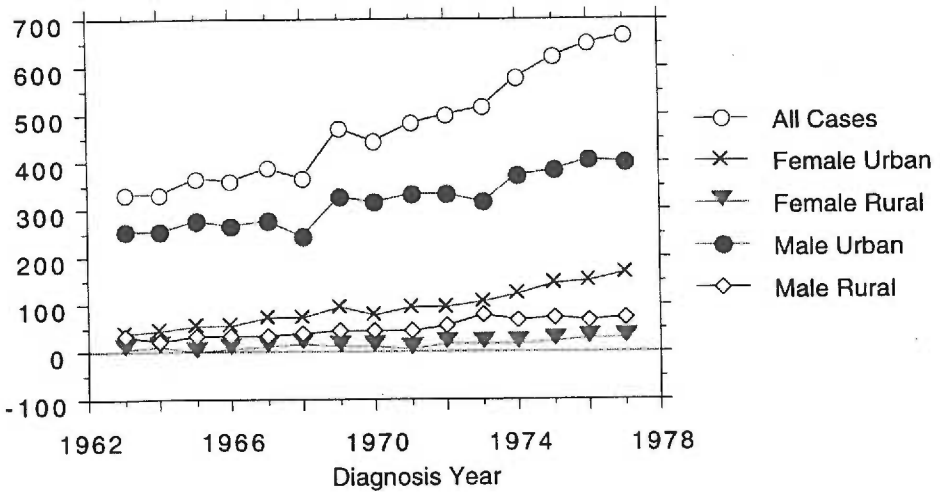
Hispanic males) was approximately twice that observed for males belonging to the Other ethnic group category (included Asian-Americans and Native Americans) at 76.08 per 100,000 population. The incidence rate observed for Caucasian females (also included Hispanic females) (18.35 per 100,000) was slightly less than that observed for African–American females (21.83 per 100,000). The incidence rate observed for females in the Other ethnic group category was 14.08 per 100,000 population. These relationships are in close agreements with those reported by other investigators. This was less than the rates observed for Caucasian and African-American females. Subsequent analyses will not include ethnic variations because of the population data were not available.

Table 4. Age-adjusted rates per 100,000 population for in males and females stratified by gender and ethnic group in the Portland-Oregon-Vancouver, Washington SMSA, 1963-1977.

	Female	Male
All ethnic groups‡	18.42	76.22
Caucasians•	18.35	76.08
African-Americans	21.83	90.91
Other Ethnic Groups◊	14.08	36.79

‡ Cases without an ethnic group code were excluded form these calculations, males (n= 9)
• The 1970 US Census grouped Hispanic Americans with Caucasians
◊ Other ethnic groups include Native Americans and Asian Americans

Figure 2. Increasing incidence of lung cancer cases over time in males and females in the Portland, Oregon-Vancouver, Washington SMSA, 1963-1977.



Lung cancer incidence by cell type

Table 5 depicts the observed cases of these three histologic cell types of lung cancer over the fifteen-year period the Portland-Vancouver SMSA. Table 6 depicts the observed cases of lung cancer stratified by gender, histologic type and urbanicity. Adenocarcinoma (23%) was the predominant lung cancer cell type observed in females in both the urban and rural census tracts. Squamous cell lung cancer (38%) had the highest incidence among males in both urban and rural tracts of the SMSA. There were more observed cases of squamous cell lung cancer for both genders combined than there were for any of the other histologic types of lung cancer.

The age-adjusted rate for lung cancer for males was 76.22 per 100,000 population. This exceeded that observed for females (18.42 per 100,000 population) over the fifteen-year period (Table 7, Column 1 and 2). Adenocarcinoma incidence at 4.34 per 100,000 population had the highest incidence among females over the fifteen-year period (Table 7, Columns. 2). Squamous cell lung cancer had the highest incidence of the three histologic groups reported for males at 29.22 per 100,000 population over the fifteen-year period (Table 7, Column 1).

During the three five year periods, 1963-1967, 1968-1972 and 1973-1977, there was a uniformly, increasing trend in incidence across the three histologic types of lung cancer and also for total lung cancer in both genders (Table 7, Columns 3-8). Adenocarcinoma incidence was the lung cancer cell type that showed the highest incidence in females over the fifteen-year period. This trend was maintained consistently over each of the three five year periods. An

increasing trend of incidence with age was observed for both sexes in the three different lung cancer histologic types (Figure 3 and Figure 4).

Table 5. Observed number of lung cancer cases stratified by gender and histologic cell type for the Portland, Oregon-Vancouver, Washington SMSA, 1963-1977.

Cancer Type	Female n (%)	Male n (%)
Total lung cancer	1602 (100)	5485 (100)
Squamous	335 (21)	2105 (38)
Adenocarcinoma	374 (23)	962 (18)
Undifferentiated Small Cell	258 (16)	777 (14)

Table 6. Observed number of lung cancer cases stratified by gender and histologic cell type for the Portland, Oregon-Vancouver, Washington SMSA, 1963-1977.

Cancer Type	Female (n=1602)		Male (n=5485)	
	Urban	Rural	Urban	Rural
Total lung cancer	1392	210	4762	723
Squamous	295	40	1824	281
Adenocarcinoma	316	58	834	128
Undifferentiated Small Cell	229	29	676	101

Table 7. Age adjusted rates per 100,000 population for different histologic types of lung cancer in the Portland, Oregon-Vancouver, Washington SMSA between 1963-1977 and for the three five year periods.

CANCER TYPE	1963-1977		1963-1967		1968-1972		1973-1977	
	Male	Female	Male	Female	Male	Female	Male	Female
TOTAL	76.22	18.42	69.69	11.2	74.29	17.01	85.92	25.36
SQUAMOUS CELL	29.22	3.92	26.27	2.36	28.63	3.25	34.21	5.98
ADENOCARCINOMA	13.33	4.34	10.95	2.55	11.6	3.59	16.84	6.14
UNDIFF SMALL CELL	10.77	3.00	8.94	1.31	9.63	2.51	13.14	5.04

† The Vancouver, Washington area is not included in the calculation of the three five year period rates (See Methods).

Figure 3. Increasing lung cancer age-specific rates per 100,000 by histology in males for the Portland, Oregon-Vancouver, Washington SMSA, 1963-1977.

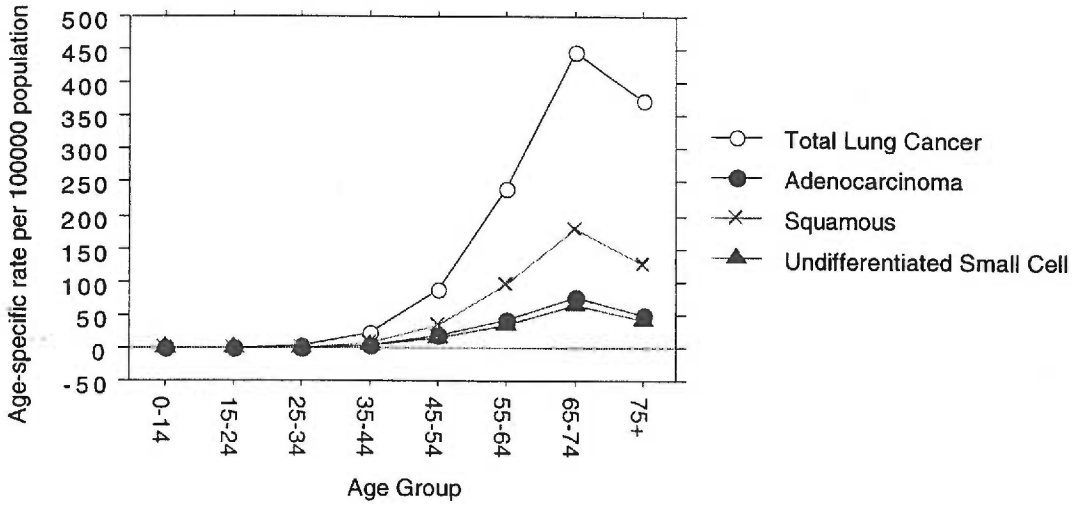
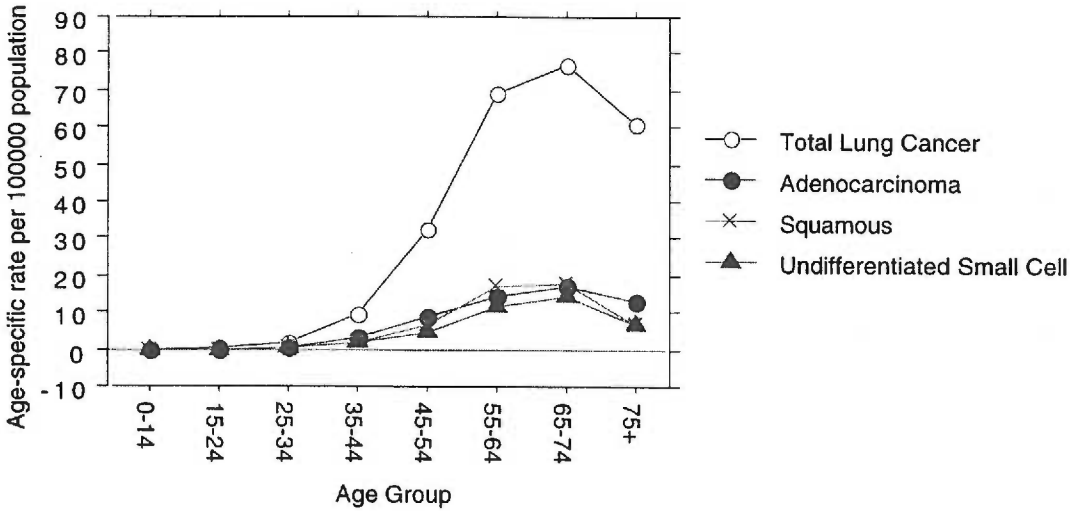


Figure 4. Increasing lung cancer age-specific rates per 100,000 by histology in females for the Portland, Oregon-Vancouver, Washington SMSA, 1963-1977.



Statistical analysis:

Simple regressions

Proposed model: Transformed SIR[7] = a + β(SES Score)

Table 8[8] shows that for total lung cancer incidence in the Portland-Vancouver area between 1963 and 1977, a statistically significant ($p<0.05$), positive relationship with SES score was observed to exist for males. The model accounted for 10.3% of the observed variance. For the male individual histologic group incidences, squamous lung cancer, and undifferentiated small cell lung cancer all showed a statistically significant ($p<0.05$), relationship between lung cancer incidence and socioeconomic status (as represented by the SES Score). The simple regression model accounted for 11.5% of the variance observed for squamous cell lung cancer. A positive, but statistically non-significant relationship ($p=0.29$) was observed for the incidence of adenocarcinoma among males and SES Score, $R^2=0.004$. For undifferentiated small cell lung cancer, a positive, statistically significant relationship, ($p<0.05$) was observed, despite that fact that the model only accounted for approximately 3% of the observed variance. Overall for males, a higher incidence of lung cancer was observed in the census tracts with a lower SES.

A positive, but statistically non-significant relationship ($p=0.09$) was observed for total lung cancer incidence among females in the SMSA, between

---

[7] Transformed SIR is the square root transformation of the Dependent variable-the standardized incidence ratio or the (SIR) (See Methods).

[8] Table 7 only shows the β values (± standard error) for single regression models for total, squamous, adenocarcinoma and undifferentiated small cell. See **Appendix C2** for the list of B-values for the remaining histogroups.

1963-1977 (Figure 5). The simple regression model accounted for 1.1% of the observed variance.

Table 8. β values (± standard error) for single regression models describing census tract correlations for male and female cancer incidence in the Portland, Oregon-Vancouver, Washington SMSA, 1963-1977.

Cancer Incidence§ (Y)	Environmental Variable SES Score			
	Male		Female	
	B	R^2	B	R^2
Total Lung Cancer	0.043±0.008*	0.103	0.022±0.013	0.011
Squamous	0.066±0.011*	0.115	0.024±0.025	0.003
Adenocarcinoma	0.017±0.016	0.004	-0.022±0.024	0.003
Undifferentiated Small Cell	0.046±0.017*	0.026	0.035±0.030	0.005

*p<0.05

§ Lung cancer incidence is expressed as the unitless transformed standardized incidence ratio (SIR).

Figure 5. Simple regression model showing the relationship between total lung cancer incidence (as shown by the transformed variable for total lung cancer, MSIRtot) and SES Score for males in the Portland, Oregon-Vancouver, Washington SMSA, 1963-1977.

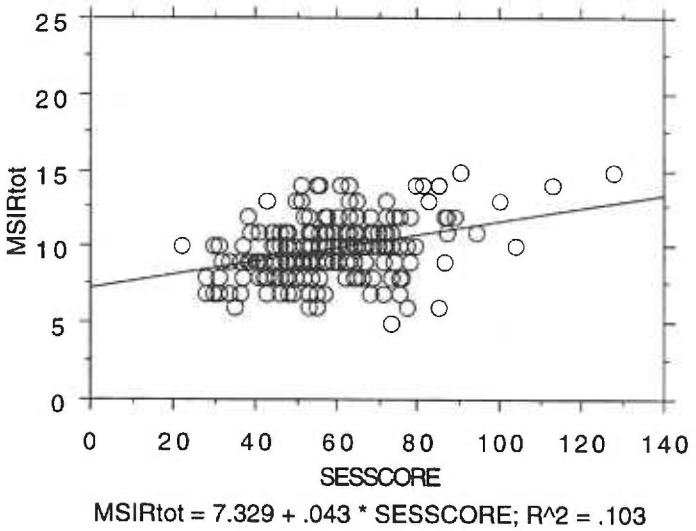


Figure 6. Simple regression model showing the relationship between total lung cancer incidence (as shown by the transformed variable for total lung cancer incidence, FSIRtot) and SES Score for females in the Portland, Oregon-Vancouver, Washington SMSA, 1963-1977.

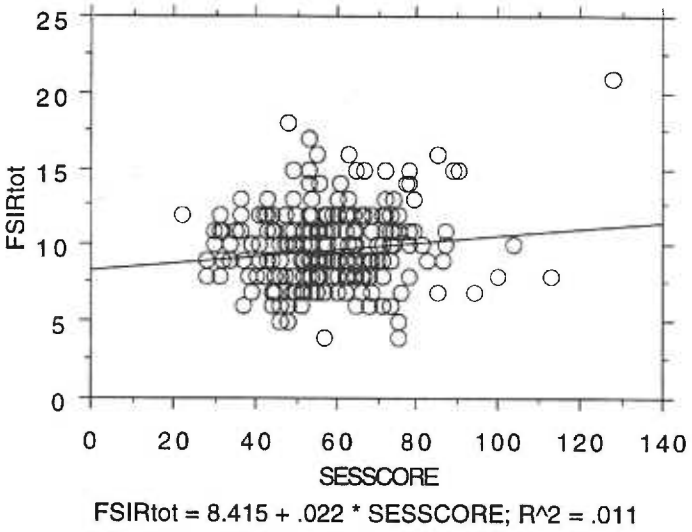
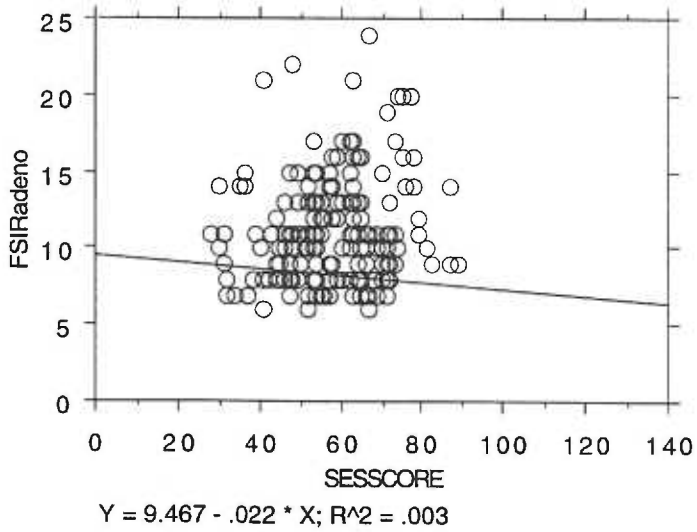


Figure 7. Simple regression model showing the relationship between adenocarcinoma incidence (as shown by the transformed variable for total lung cancer incidence, FSIRadeno) and SES Score for females in the Portland, Oregon-Vancouver, Washington SMSA, 1963-1977.



Multiple regressions

Proposed model:

Transformed SIR = $a+\beta_1$(SES Score)+ β_2(Urbanicity)+ β_3(SES Score*Urbanicity)

Within this model, a positive, statistically significant ($p<0.05$) correlation between total lung cancer incidence and the three independent variables, SES Score, Urbanicity and the interaction term SES Score*Urbanicity in males was observed (Table 9). The inclusion of Urbanicity and the interaction term affected the relationship between SES Score and total lung cancer incidence in males. There was no longer a positive, statistically significant relationship observed between SES Score and total lung cancer incidence in males. In fact, there was a negative, statistically non-significant ($p=0.69$), relationship between total lung cancer and SES Score (Table 9).

The multiple variable regression accounted for approximately 14% of the observed variance for total lung cancer incidence and for squamous cell lung cancer incidence. This was an improvement over the simple regression model, where $R^2 = 0.103$ for total lung cancer incidence and $R^2= 0.11$ for squamous cell (Table 8). For adenocarcinoma and undifferentiated small cell lung cancer in males, the multiple regression model accounted for less than 5% of the variance.

The multivariate regression model for females (Table 10) depicts an overall, statistically non-significant ($p=0.31$), negative correlation between total lung cancer incidence and the SES score. This model only accounted for 1.4% of the observed variance. There was little improvement observed when compared to the simple regression model, where $R^2=0.011$ (Table 8).

Urban total lung cancer incidence was observed to be lower than rural lung cancer rates for the census tract (Figure 8). The statistically significant interaction term indicated the presence of a statistically significant difference between the total lung cancer incidence observed in urban areas when compared to that observed for the rural areas. Socioeconomic status appeared to have no effect on total lung cancer incidence in females in both the urban and rural census tracts (Figure 9). There did not appear to be any statistically significant trend ($p>0.05$) in total lung cancer incidence that was associated with urbanicity. The incidences for rural tracts also appeared to cluster around the middle of the SES range. There was a wider dispersion of the urban total lung cancer incidences over the SES range.

For squamous cell (Figure 10), adenocarcinoma (Figure 12) and undifferentiated small cell (Figure 14) lung cancer incidence in males, (when each was examined separately), a negative, statistically non-significant relationship was observed to exist between these the individual incidences of each type of lung cancer and SES Score. Urbanicity was significant for squamous cell cancer (Table 9), in that there was a statistically significant difference ($p=0.02$) between the slopes of the regression lines for the urban and rural census tracts.

For males, squamous cell lung cancer incidence (Figure 10) in rural census tracts with high SES status also exceeded that of the more affluent urban census tracts. The interaction between SES and urbanicity was also significant indicating that there was also a statistically significant difference in the incidence of squamous cell lung cancer in the poorer, urban census tracts when compared to the poorer rural census tracts. Squamous cell cancer incidence in females

appeared to increase at approximately the same rate with increasing socioeconomic scores in both the urban and rural census tracts (Figure 11).

For males, a negative correlation was observed for adenocarcinoma incidence and SES Score in the multivariate model for the rural census tracts (Figure 12). This relationship was not statistically significant, p=0.12. Rural census tracts experienced a decreasing incidence with increasing socioeconomic score. The interaction between SES Score and urbanicity was not statistically significant (p=0.06). For females, adenocarcinoma incidence (Figure 13) appeared to decrease with increasing SES score in the urban tracts. SES did not appear to affect adenocarcinoma incidence in rural females.

For males in the rural census tracts, there was a negative relationship between undifferentiated small cell cancer incidence and SES in the rural tracts (Figure 14). This was statistically significant, p=0.02. Undifferentiated small cancer lung incidence increased with increasing SES score in the urban tracts. The effects of urbanicity (p=0.20) and the interaction term SES Score*Urbanicity (p=0.14) did not have a statistically significant effect on undifferentiated small cell cancer incidence in males.

Undifferentiated small cell cancer incidence increased with increasing SES in the urban areas for females (Figure 15). The converse was observed for the rural areas. This pattern of decreasing incidence with increasing SES Score in the rural tracts was also observed for adenocarcinoma in males. There were no statistically significant relationships observed for this multivariate model, R^2=1.6%, (p=0.27).

Table 9. β values (± standard error) for multiple regression models describing census tract correlations for male cancer incidence in the Portland, Oregon-Vancouver, Washington SMSA, 1963-1977.

Cancer Incidence§ (Y)	Environmental Variables				
	SES Score	Urbanicity	Ses Score*Urbanicity	R^2	p◊
Total Lung Cancer	-0.001±0.026	-3.237±1.766	0.062±0.027*	0.137	0.00
Squamous	-0.013±0.011	-5.112±2.582*	0.090±0.040*	0.139	0.00
Adenocarcinoma	-0.076±0.053	-6.081±3.591	0.105±0.055	0.022	0.12
Undifferentiated Small Cell	-0.032±0.059	-5.092±3.979	0.090±0.061	0.038	0.02

§ Lung cancer incidence is expressed as the unit-less transformed standardized incidence ratio (SIR).
*p<0.05
◊ p value for the entire multiple regression model.

Table 10. β values (± standard error) for multiple regression models describing census tract correlations for female cancer incidence in the Portland, Oregon-Vancouver, Washington SMSA, 1963-1977.

Cancer Incidence§ (Y)	Environmental Variables				
	SES Score	Urbanicity	Ses.Score*Urbanicity	R^2	p◊
Total Lung Cancer	-0.014±0.044	-2.573±2.971	0.038±0.046	0.014	0.31
Squamous	0.026±0.086	0.792±5.853	0.002±0.090	0.007	0.62
Adenocarcinoma	-0.007±0.081	1.242±5.529	0.004±0.085	0.009	0.48
Undifferentiated Small Cell	-0.062±0.101	-5.747±6.846	0.114±0.106	0.016	0.27

§ Lung cancer incidence is expressed as the unitless transformed standardized incidence ratio.
*p<0.05
◊ p value for the entire multiple regression model.

N.B. Table 9 and Table 10 only show the β values (± standard error) for the three histogroups selected for reporting purposes. Please see Appendices C3 and C4 for the other histogroups.

Figure 8. The relationship between total lung cancer (as shown by the transformed SIR variable, MSIRtot) and SES Score in males, split by urbanicity for the Portland, Oregon-Vancouver, Washington SMSA, 1963-1977.

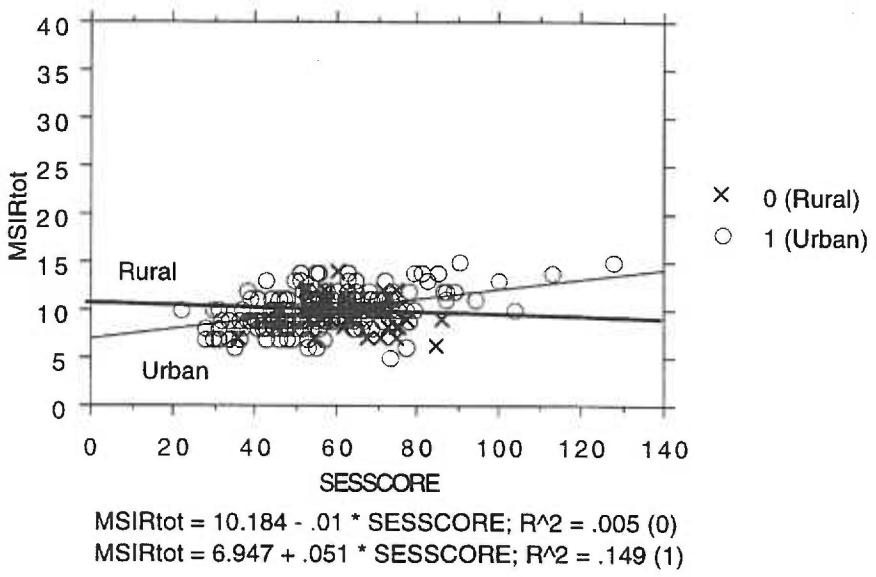


Figure 9. The relationship between total lung cancer (as shown by the transformed SIR variable, FSIRtot) and SES Score in females, split by urbanicity for the Portland, Oregon-Vancouver, Washington SMSA, 1963-1977.

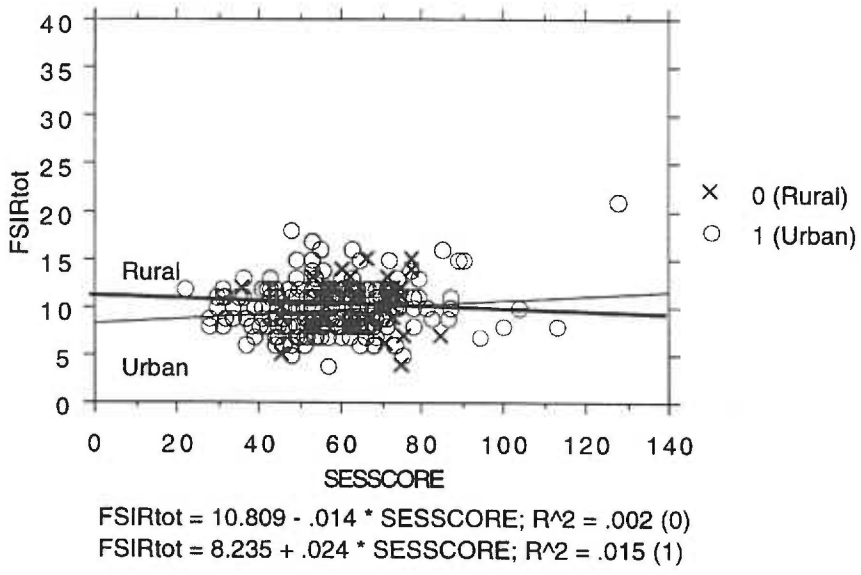


Figure 10. The relationship between squamous cell lung cancer (as shown by the transformed SIR variable, MSIRsq) and SES Score in males, split by urbanicity for males in the Portland, Oregon-Vancouver, Washington, 1963-1977.

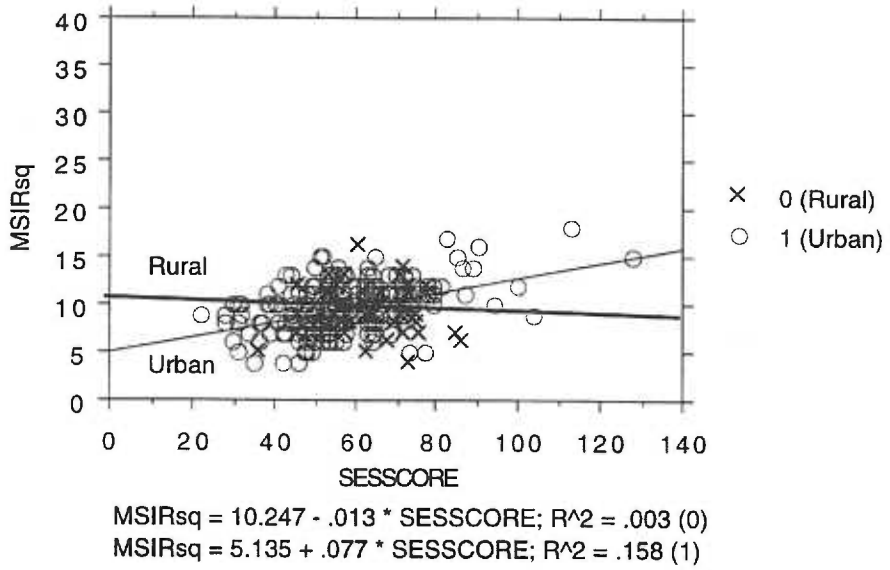


Figure 11. The relationship between squamous lung cell cancer (as shown by the transformed SIR variable, FSIRsq) and SES Score in females, split by urbanicity for the Portland-Vancouver SMSA, 1963-1977.

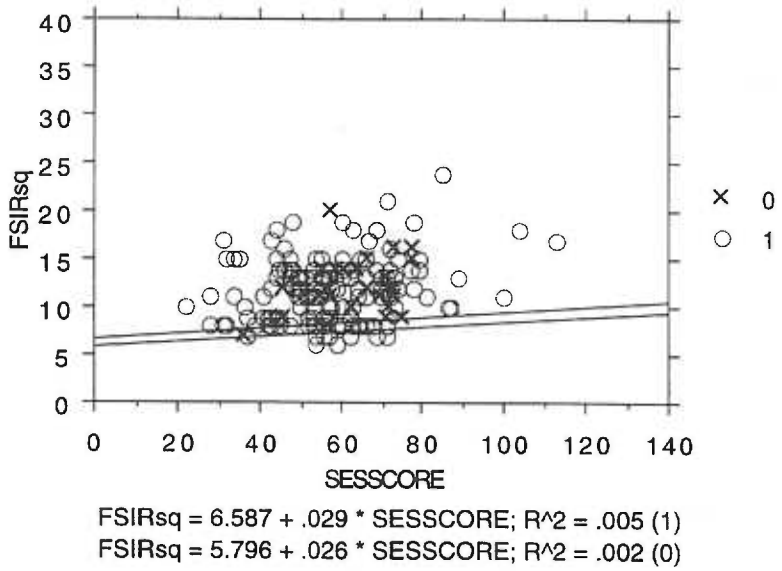
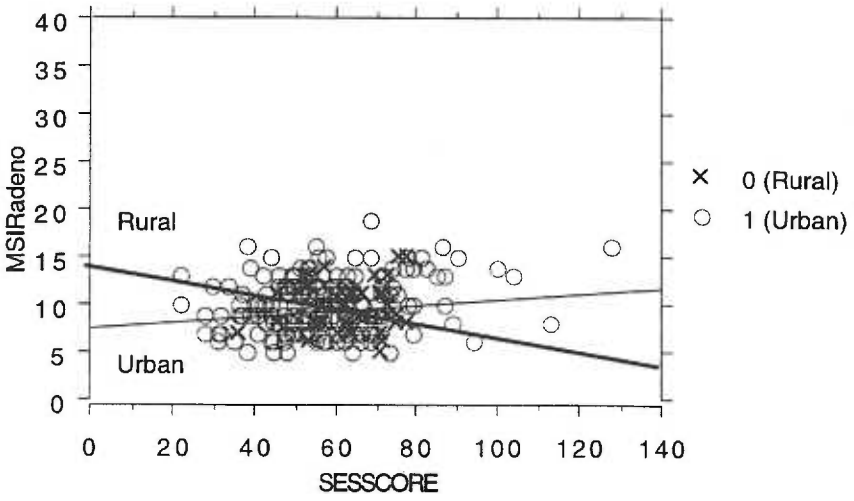
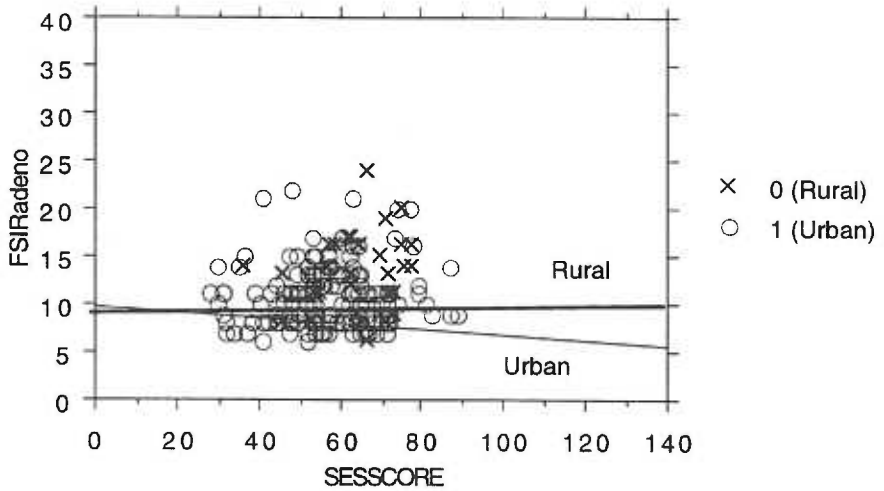


Figure 12. The relationship between adenocarcinoma lung cancer (as shown by the transformed SIR variable, MSIRadeno) and SES Score in males, split by urbanicity for males for the Portland, Oregon-Vancouver, Washington SMSA, 1963-1977.



MSIRadeno = 13.678 - .076 * SESSCORE; R^2 = .044 (0)
MSIRadeno = 7.597 + .029 * SESSCORE; R^2 = .014 (1)

Figure 13. The relationship between adenocarcinoma lung cell cancer (as shown by the transformed SIR variable, FSIRadeno) and SES Score in females, split by urbanicity for the Portland-Vancouver SMSA, 1963-1977.



FSIRadeno = 8.528 + .007 * SESSCORE; R^2 = 1.188E-4 (0)
FSIRadeno = 9.77 - .03 * SESSCORE; R^2 = .007 (1)

Figure 14. The relationship between undifferentiated small cell lung cancer (as shown by the transformed SIR variable, MSIRunsmall) and SES Score in males, split by urbanicity for the Portland, Oregon-Vancouver, Washington SMSA, 1963-1977.

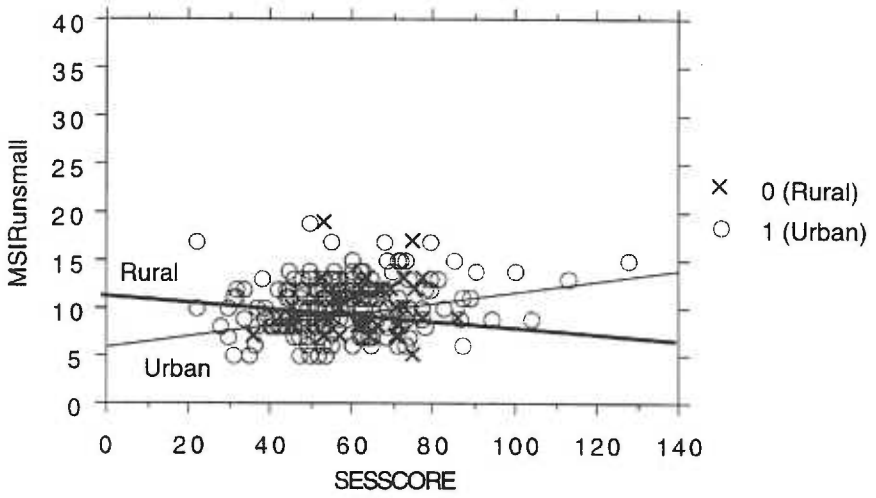
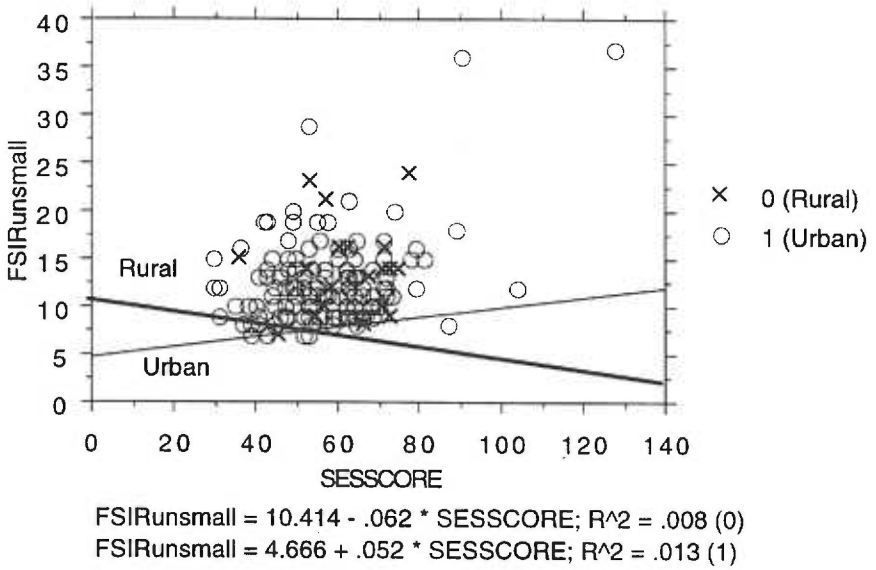


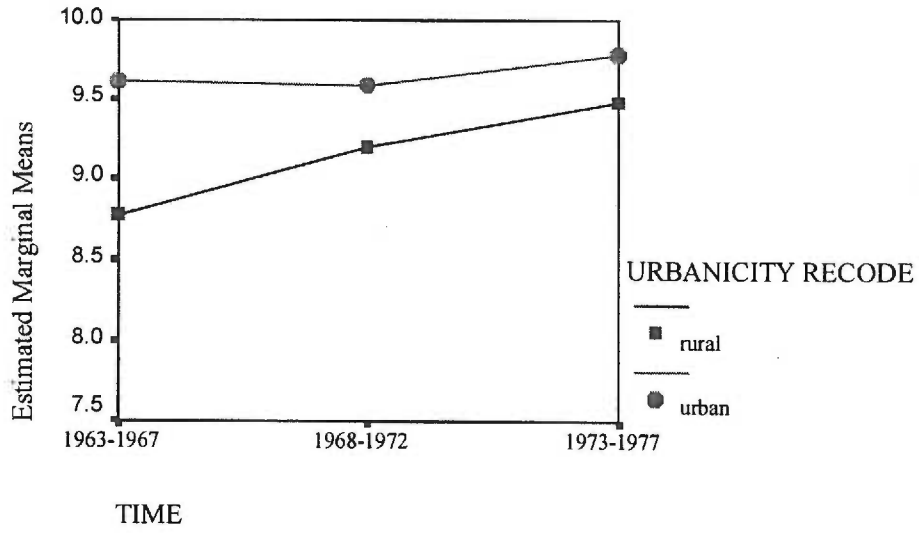
Figure 15. The relationship between undifferentiated small cell lung cancer (as shown by the transformed SIR variable, FSIRunsmall) and SES Score in females, split by urbanicity for the Portland, Oregon-Vancouver, Washington SMSA, 1963-1977.



Trends analysis

The effect of time on total lung cancer incidence and the three histologic groups selected for reporting purposes[9] for males and females over the three five year periods was examined. For males, the urban and rural trends were both upward, drawing closer together over time (Figure 16). For females time was found not to have a significant effect on the incidence of total lung cancer (Figure 17). The overall effect of time in the model approached significance for adenocarcinoma in males (Figure 18).

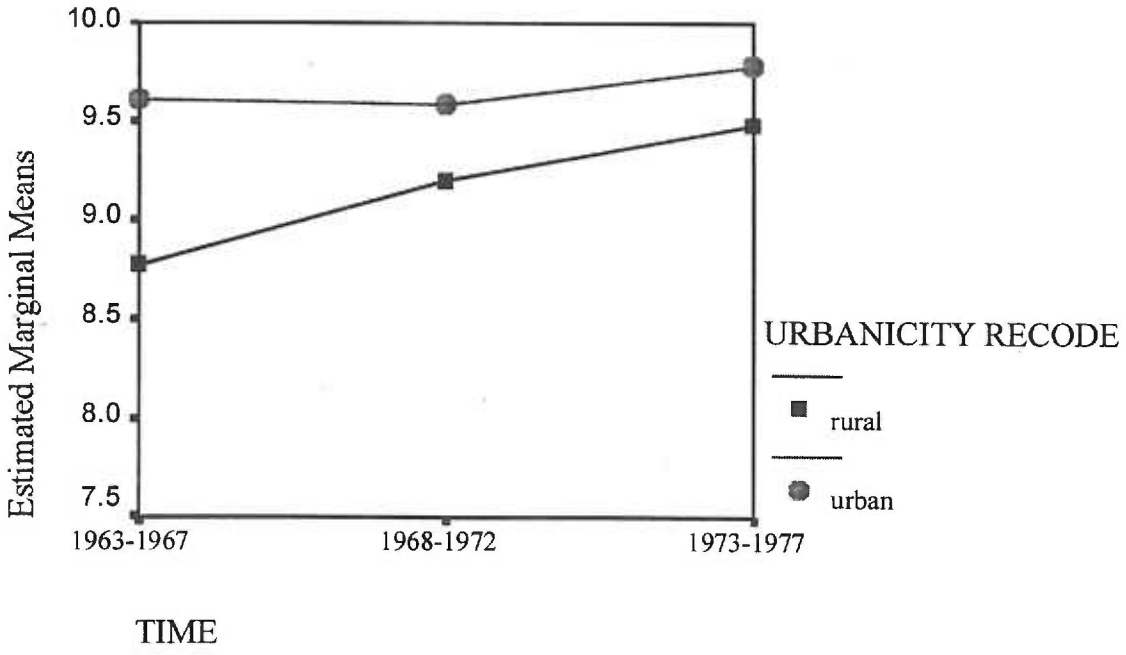
Figure 16. The estimated marginal means of total lung cancer incidence (SIR) for the three five year periods for males in the Portland, Oregon-Vancouver, Washington SMSA.



Time F=1.396, p=0.249
Time*Urbanicity F=0.706, p=0.494 (SES score was controlled for this model).

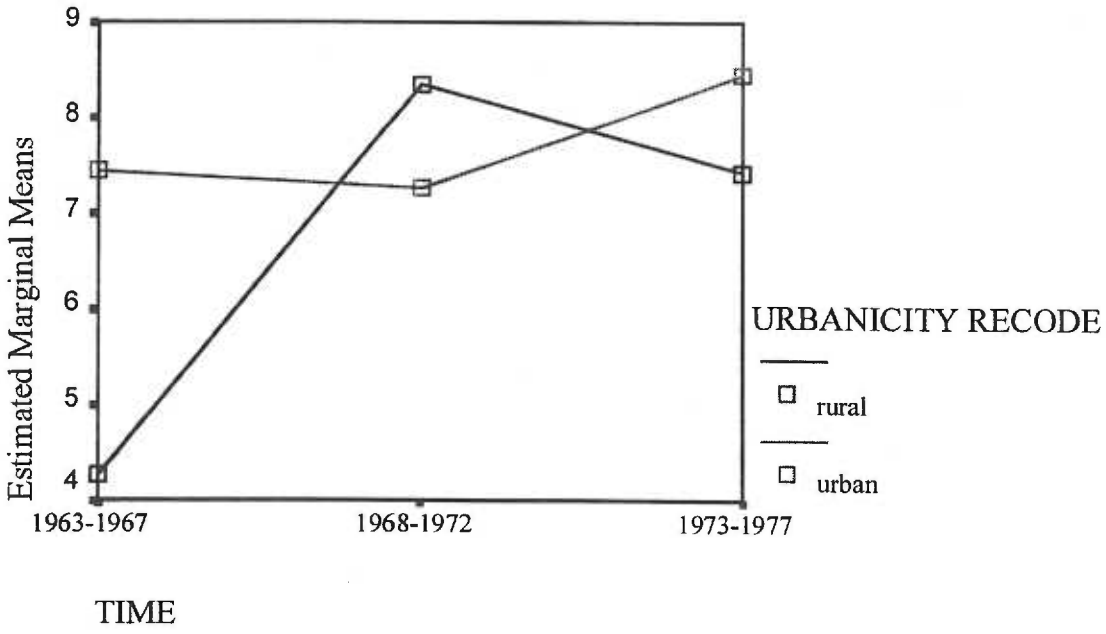
[9] See Appendices C5-C9.

Figure 17. The estimated marginal means of total lung cancer incidence (SIR) for the three five year periods for females in the Portland, Oregon-Vancouver, Washington SMSA.



Time F=2.987, p=0.051
Time*Urbanicity F=0.851, p=0.428
SES score was controlled for this model.

Figure 18. The estimated marginal means of adenocarcinoma (SIR) for the three five year periods for males in the Portland, Oregon-Vancouver, Washington SMSA.



Time F=0.287, p=0.751
Time*Urbanicity F=2.694, p=0.069
SES score was controlled for this model.

DISCUSSION

This study sought to examine the relationship between lung cancer incidence by histology and socioeconomic status in the Portland, Oregon-Vancouver, Washington SMSA. This was an ecologic study, and it should be noted that any associations between lung cancer incidence by histology and socioeconomic status might not necessarily represent an association at the individual level or even a causal relationship. Any associations shown here may lead to further investigation at an individual level.

The strengths of the ecologic study are as follows:

1) Low cost and convenience, due to its use of secondary data sources that can be linked at the aggregate level (Hulley and Cummings, 1988; Rothman and Greenland, 1998).
2) Measurement limitations of individual-level studies, sometimes in environmental epidemiology studies relevant environmental exposures cannot be measured accurately. Individual level exposures cannot be measured accurately due to within-person variability. Ecological studies accurately affect group averages (Rothman and Greenland, 1998).
3) There may be little variation of the exposure within the study area. Thus utilizing an individual-level study design may not be practical for assessing exposure effects. Utilizing the ecologic study design enables the coverage of a wider study area. This allows for the achievement of some variation among the mean exposures across groups (Rothman and Greenland, 1998).
4) There may be an interest in establishing an ecologic effect. Ecologic effects are especially relevant when assessing the impacts of social programs or interventions. They may also be valuable in indicating target populations for intervention programs (Rothman and Greenland, 1998).
5) Ecologic studies have an inherent simplicity of analysis and presentation. This allows the manipulation and analysis of data gathered form large, periodic studies (Rothman and Greenland, 1998). For example, the National Health Interview Survey.

The major limitation to the use of the ecological study design is the problem of ecologic bias. This is characterized by the failure of expected ecologic effects to accurately estimate the biologic effect at an individual level (Rothman and Greenland; 1998). Because of the aggregate nature of the ecologic study

design, the heterogeneity of the biologic effect is lost or is not fully captured. This is realized in the reduction of the variance. Small variance values are the norm for ecologic studies, for example, $R^2=0.10$, $p<0.05$, was observed for males in the simple regression analysis (See Results). It should be understood that each aggregate observation (the SIR, or dependent variable) is a mean measure of the group of individual lung cancer cases within the unit of analysis. This will cause the range of observed values for an ecologic study to be less than that observed for an individual-design study. A corresponding reduction in the variance estimate usually accompanies a reduction in the range of values (Connor and Gillings, 1984).

Greenland and Morgenstern (1989) characterized the problems that can emerge from the use of simple linear regressions to estimate the crude exposure effect. It is important to realize that "within-group" bias may emerge from bias within groups that is due to confounding, selection methods or misclassification. In this study we have attempted to address "within-groups" bias by the following:

1) The selection of a measure of socioeconomic status that did not utilize the confounding variable, occupation and
2) All diagnoses of lung cancer during 1963-1977 for residents of the Portland-Vancouver metropolitan area were included in the study and death certificates were reviewed to locate additional cases.

Misclassification bias may have occurred during the data collection process. There were no independent slide reviews conducted when the data was originally collected. This meant that the data would include some degree of histological inconsistencies and error (Feinstein et al., 1970). Another of the strategies for reducing ecologic bias is the use of smaller units in an ecologic

study, i.e., counties instead of states (Rothman and Greenland, 1998). By utilizing the 1970 US census tracts as the unit of analysis, we attempted to make the groups more homogenous with respect to exposure.

In the Portland-Vancouver SMSA, lung cancer incidence for males was approximately four times that of females over the fifteen-year period, 1963-1977 (Table 6). A steady increasing trend was observed which mirrored that observed nationally. The number of female cases in the Portland-Vancouver SMSA was observed to double between 1963 and 1977 (Table 2). Lung cancer incidence was negatively correlated with socioeconomic status in males. The correlation was statistically significantly ($p<0.05$). This also mirrored the trend that had been observed nationally and globally (van Loon et al., 1995; Månsson et al., 1995; Hein et al., 1992; Samet 1992).

The association between lung cancer incidence and smoking has been well established through many epidemiological and experimental studies. There have been few epidemiological studies that have attempted to (1) examine lung cancer incidence by histologic type and (2) characterize the effect of socioeconomic status on lung cancer incidence by histology. Despite this, there appears to be general consensus that the different histologic types of lung cancer have a multi-factorial etiology which includes, occupational exposures, cigarette smoking and other environmental exposures (Brownson, 1987).

The three main indicators of socioeconomic status used by others have been occupation, education and income. The majority of SES rankings that have been developed by sociologists are based on occupation because many sociologists believe that occupation is a single reliable indicator of relative standing in developed societies (Liberatos et al., 1988). Poor measurement of

socioeconomic status can lead to random misclassification that can in turn dilute the effect of any actual bivariate associations. Liberatos et al., in 1988 illustrated this with the example of the controversy that surrounded the finding that there was an inverse relationship between schizophrenia and social class. They cited the conclusions reached by Hollingshead and Redlich in their 1958 publication "Social Class and mental illness: a community study".

Hollingshead and Redlich utilized a composite index that was heavily influenced by education as an indicator of social class. They found that there was no evidence that schizophrenia was inversely related to social class. Education proved to be a misleading indicator because the onset of schizophrenia does not occur until late adolescence or early adulthood. This is the period by which education is usually completed. Hollingshead and Redlich produced a bias towards finding that an inverse relationship between social class and schizophrenia did not exist. This was later refuted by several studies that utilized occupation-based measures of social class. Here it was shown that an inverse relationship existed between social class and schizophrenia by the time of first admission to a mental hospital. Schizophrenics were unlikely to be steadily employed due to the natural course of their disease, but would have completed at least a high school education.

Conversely the inclusion of a potentially confounding factor into the assessment of SES can lead to the misinterpretation of any bivariate association. The utilization of occupation as a means of assessing socioeconomic status can possibly lead to the inflation of observed lung cancer incidence, especially as different occupational exposures have been shown to be powerful risk factors. In the case of small cell lung cancer, occupational exposures are powerful, potential

risk factors for cancer incidence. This was shown by Figueosa, et al., in 1973 when the relationship between small cell lung cancer and exposure to chloromethyl ethers in plastics production was discovered. Sankila et al., (1990) also showed the relationship between occupation and lung cancer as analyzed by age and histologic type. Sankila conducted a population-based survey on males utilizing the records from the Finnish Cancer Registry. Here mining and quarrying carried a high SIR of 238, $p< 0.05$ for small cell lung cancer, when compared to those of other economically similar males.

It is important to understand that an individual's occupational status can vary over a lifetime. This can also further confound the issue when examining the role of possible exposures and their potential biological outcomes, i.e., cancer incidence. The recognition of possible role of occupational exposures as confounders has led to the avoidance of any possible upward bias in lung cancer incidence, i.e., an increase in the estimate of lung cancer risk.

This study calculated socioeconomic status based on a socioeconomic score. This score was derived from the social indicators—education, median income and household density at the time of diagnosis (See Methods). This is a composite measure that is appropriate given the multi-faceted nature of social class. Haer (1957) found the greatest increase in predictive utility using a composite measure of social class. The results of this presentation showed that lung cancer incidence increased with decreasing socioeconomic status in males. This finding echoes what had been reported by the medical literature (See Introduction) for males and strengthens the validity of the social indicators used in the determination of socioeconomic status-the Socioeconomic Score.

A negatively correlated, but statistically non-significant (p= 0.09) relationship for lung cancer incidence and socioeconomic status was observed in females in the Portland-Vancouver SMSA. This was not unexpected, for previous evidence in the medical literature (van Loon 1995; Faggiano et al., 1994,) had showed that consensus had not been reached for lung cancer incidence by SES in females.

Conflicting observations have been made within the same study. Faggiano conducted a cancer incidence follow-up study in Turin in relation to the socioeconomic characteristics of education, housing tenure and occupation. Professional women were found to have half the incidence of lung cancer than manual workers (OR=0.45 (95%CI: 0.22-0.91)). The same risk (adjusted by education) was found to be 44% higher (p=0.09 for X_2) for female tenants (OR=1.44 (95% CI: 0.99-2.10) than for house owners. This inverse trend in ORs in women reflects the social distribution of smoking in Italy, which ranges from 14.2% for the lowest educational level to 23.3% for the highest educational level. These findings suggest that assessing lung cancer incidence by SES in females may not reveal any socioeconomic differences.

The above study illustrates the importance of the different meanings of social indicators. Low income (as represented by housing tenure) appears to confer a protective effect on females. Education is assumed to confer an emancipative effect, i.e., women have followed the smoking trends of men (Faggiano et al., 1994).

Another unexpected finding of this present investigation was the apparent negative correlation between socioeconomic status and adenocarcinoma incidence in males in the rural areas over the fifteen-year period.

Adenocarcinoma incidence decreased with increasing socioeconomic score in the rural census tracts. This was a statistically non-significant relationship ($p=0.12$), but it was consistently different from the urban pattern for males. The same relationship was observed for females in the urban census tracts. These were interesting findings that pointed to the possibility that different etiologic factors may be at work influencing the incidence of adenocarcinoma in rural males and urban females.

Existing medical literature has indicated a shift in the histologic pattern of lung cancer. The incidence of adenocarcinoma has appeared to be increasing overall and within each gender (Dodds, 1986; Wu, 1986). Similar patterns were not observed for squamous cell and undifferentiated small cell lung cancer incidence in the literature.

The smaller number of cases observed in the smaller number of rural census tracts and the smaller range of socioeconomic scores assigned to these tracts made it difficult to directly compare urban and rural cancer risks. Rural census tracts were often geographically larger, less populated and more diverse. This could lead to an over-representation of the cancer risk estimate. It should also be understood that the geographical location of the census tract within the SMSA is also of some importance. For example, urban tracts for central Washington county, Oregon were surrounded by a primarily agricultural community. This would provide a host of different environmental exposures, than what would be observed for urban Multnomah county (location of the city of Portland).

The increasing trends in lung cancer incidence observed in the Portland, Oregon-Vancouver, Washington SMSA could be attributed to improving

diagnostic techniques and a change in pathological classifications. This would not change total lung cancer but it could impact the incidence of observed individual histologic types.

This study utilized a composite measure of socioeconomic status, which did not include occupation. This utility of this measure was validated in males, as it provided results of what had been observed elsewhere. For females, this measure had no effect. One future direction may be to examine the possibility of the predictive utility in reproducing the statistical analyses using the socioeconomic indices of education, household density index and income as separate indicator variables. It may be that using an aggregate index may obscure differences in the individual relationships between lung cancer incidence in females and each of the separate indicator variables, for example, income and education.

CONCLUSIONS

Lung cancer incidence in males was shown to be positively correlated with socioeconomic status (as represented by the composite index, SES Score). The utility of the SES Score was not realized in females, for the model accounted for approximately 1% of the observed variance. Adenocarcinoma in rural males and urban females decreased with increasing SES score. Undifferentiated small cell cancer incidence was observed to decrease with increasing SES score males and females in rural. These two phenomena underscored the fact that different etiologic factors irrespective of socioeconomic status are impacting both genders differently in the urban and rural census tracts.

REFERENCES

1. Antonovsky A. Social class, life expectancy and overall mortality. *Millbank Memorial Fund Quarterly*. 1967;45:31-73.
2. Baquet CR, Horm JW, Gibbs T, Greenwald P. Socioeconmic factors and cancer incidence among blacks and whites. *Journal of the National Cancer Institute*. 1991;83(8):551-556.
3. Beard CM, Annegers JF, Woolner LB, Kurland LT. Bronchogenic carcinoma in Olmsted County, 1935-1979. *Cancer*. 1985;55(9):2026-2030.
4. Beard MC, Jedd MB, Woolner LB, Richardson RL, Bergstralh EJ, Melton JL. Fifty-year trend in incidence rates of bronchogenic carcinoma by cell type in Olmsted county, Minnesota. *Journal of the National Cancer Institute*. 1988;80(17):1404-1407.
5. Benhamou E, Benhamou S, R F. Lung cancer and women: results of a French case-control study. *British Journal of Cancer*. 1987;55(1):91-95.
6. Blot W, Fraumeni J. Cancers of the lung and pleura. In: Schotenfeld D, Fraumeni J, eds. *Cancer Epidemioloogy and Prevention*. New York: Oxford University Press; 1996.
7. Brown SM, Selvin S, WInkelstein W. The association of economic status with the occurrence of lung cancer. *Cancer*. 1975;36(5):1903-1911.
8. Brownson RC, Reif JS, Keefe TJ, Ferguson SW, Pritzl JA. Risk factors for adenocarcinoma of the lung. *American Journal of Epidemiology*. 1987;125(1):25-34.
9. Carney D, Leij L. Lung cancer biology. *Seminars in Oncology*. 1988;15(3):199-214.
10. CDC. Cigarette smoking among adults-United States, 1990-1994. *Morbidity and Mortality Reports*. 1995;46(51):1217-1220.
11. CDC. Projected smoking-related deaths among youth-United States. *Morbidity and Mortality Reports*. 1996;45(44):971-974.
12. CDC. Tobacco use among US racial/ethnic minority groups, African-Americans, American Indians and Alaska Natives, Asian Americans and Pacific Islanders, Hispanics: A report of the Surgeon General (Executive Summary). *Morbidity and Mortality Reports*. 1998;47(RR-18):1-16.
13. Connor MJ, Gillings D. An empiric study of ecological interference. *American Journal of Public Health*. 1984;74(5):555-559.
14. Cuello C, Correa P, Haenszel W. Socio-economic class differences in cancer incidence in Cali, Columbia. *International Journal of Cancer*. 1982;29:637-643.
15. Devesa SS, Diamond EL. Socioeconomic and racial differences in lung cancer incidence. *American Journal of Epidemiology*. 1983;118(6):818-830.
16. Dodds L, Davis S, Polissar L. A population-based of lung cancer incidence trends by histologic type, 1974-1981. *Journal of the National Cancer Institute*. 1986;76(1):21-29.
17. Doll R, AB H. A study of the aetiology of carcinoma of the lung. *British Medical Journal*. 1950;2:740-748.
18. Doll R, Hill AB. A study of the aetiology of carcinoma of the lung. *British Medical Journal*. 1952;2:1271-1286.
19. Doll R, Gray R, Hafner B, Peto R. Mortality in relation to smoking: 22 years' observation on female British doctors. *British Medical Journal*. 1980;2(967-971).

20. Engholm G, Palmgren F, Lygne E. Lung cacner, smoking and environment: a cohort study of the Danish population. *British Medical Journal.* 1996;312:1259-1263.
21. Faggiano F, Zanetti R, Costa G. Cancer risk and social inequalities in Italy. *Journal of Epidemiology and Community Health.* 1994;48:447-452.
22. Faggiano F, Lemma P, Costa G, Gnavi R, Pagnanelli F. Cancer mortality by educational level in Italy. *Cancer Cases and Control.* 1995;6:311-320.
23. Feinstein AR, Gelfman NA, Yesner R. Observer variability in the histopathologic diagnosis of lung cancer. *American Review of Respiratory Disease.* 1970;101(5):671-684.
24. Friedberg J, Kaiser L. Epidemiology of lung cancer. *Seminars in Thoracic and Cardiovascular Surgery.* 1997;9(1):56-59.
25. Giovino G, Schooley M, Zhu B. Surveillance for selected tobacco-use behaviors-United States, 1990-1994. *Morbidity and Mortality Reports.* 1994;43(SS-3):1-43.
26. Greenberg ER, Korson R, Baker J, Barrett J, Baron JA, Yates J. Incidence of lung cancer by cell type: a population-based study in New Hampshire and Vermont. *Journal of the National Cancer Institute.* 1984;72(3):599-603.
27. Greenland S, Morgenstern H. Ecologic bias, confounding and effect modification. *International Journal of Epidemiology.* 1989;18(1):269-274.
28. Haer J. Predictive utility of the five indices of social stratification. *American Social Review.* 1957;22:541-546.
29. Hammond E. Smoking in relation to the death rates of one million men and women. *national Cancer Institute Monograph.* 1966;19:127-204.
30. Hearst N, Hulley SB. Using secondary data. In: Hulley SB, Cummings SR, eds. *Designing Clinical Research.* Baltimore: Williams and Wilkins; 1988.
31. Hein HO, Suadicani P, Gyntelberg F. Lung cancer risk and social class. *Danish Medical Bulletin.* 1992;39:173-176.
32. Ianuzzi M, Scoggin C. Small cell lung cancer. *American Review of Respiratory Diseases.* 1986;134:593-608.
33. Ives JC, Buffler PA, Greenberg SD. Environmental associations and histopathological patterns of carcinoma of the lung: the challenge and dilemma in epidemiologic studies. *American Review of Respiratory Disease.* 1983;128:195-209.
34. Kreyberg L. Histological lung cancer types: A morphological and biological correlation. *Acta Pathology Microbiology Immunology Scandanavia.* 1962;157(S):11-90.
35. Kreyberg L, Liebow A, Uehlinger E. Histological typing of lung tumors. Geneva: World Health Organization; 1967.
36. Kreyberg L. Comments in the histolgical typing of lung tumors. *Acta Pathology Microbiology Scandanavia.* 1971;79:409-422.
37. Kreyberg L. Lung cancer in workers in a nickel refinery. *British Journal of Industrial Medicine.* 1978;35:109-116.
38. Landis SH, Murray T, Bolden S, Wingo PA. Cancer Statistics, 1999. *CA-A Cancer Journal for Clinicians.* 1999;49(1):8-31.
39. Levi F, Negri E, La Vecchia C, Te Vc. Socioeconomic groups and cancer risk at death in the Swiss Canton of Vaud. *International Journal of Epidemiology.* 1988;17(4):711-717.
40. Levin ML, Goldstein H, Gerhardt PR. Cancer and tobacco smoking. *The Journal of the American Medical Association.* 1950;113(4):336-338.

41. Liberatos P, Link BG, Kelsey JL. The measurement of social class in epidemiology. *Epidemiologic Reviews*. 1988;10:87-121.
42. Lubin JH, Blot WJ. Assessment of lung cancer risk factors by histologic category. *Journal of the National Cancer Institute*. 1984;73:383-389.
43. Maher K, Defonso L. Respiratory cancer among chloromethyl ether workers. *Journal of the National Cancer Institute*. 1987;78:839-843.
44. Månsson J, Marklund B, Bengtsson C, Odén A. Differences in cancer incidence between parts with different socio-economic structure within a Swedish big-city area. *Neoplasma*. 1995;42(4):149-153.
45. Minowa M, Stone BJ, Blot WJ. Geographic pattern of lung cancer in Japan and its environmental correlations. *Japanese Journal of Cancer Research*. 1988;79:1017-1023.
46. Modan B. Population distribution of histological types of lung cancer. *Israel Journal of Medical Science*. 1978;14(7):771-784.
47. Morton W, Treyve EL. Histologic differences in occupational risks of lung cancer incidence. *American Journal of Industrial Medicine*. 1982;3:441-457.
48. Morton W, Phillips D. Radioemission density and cancer epidemiology in the Portland metropolitan area. Research Triangle Park, N.C.: Health Effects Research Laboratory, US EPA; 1983.
49. Muscat J, Wynder E. Lung cancer pathology in smokers, ex-smokers and never smokers. *Cancer letters*. 1995;88:1-5.
50. NCHS. Advance report of final monthly statistics. Rockwille, MD: Centers for Disease Control and Prevention; 1993.
51. NCI. SEER Cancer Statistics Review, 1973-1994; 1996.
52. Ochsner A, DeBakey M. Symposium on lung cancer: primary pulmonary malignancy, treatment by total pneumonectomy, analysis of 79 collected cases and presentations of 7 personal cases. *Surgery, Gynecology and Obstetrics*. 1939;68:435-451.
53. Rosenwaite I. Changing patterns of lung cancer among socio-cultural groups in New York City. *Public Health Briefs*. 1984;74(8):839-840.
54. Rothman KJ, Greenland S. *Modern Epidemiology*. Philadelphia: Lippincott-Raven; 1998.
55. Samet JM, Wiggins CL, Key CR, Becker TM. Mortality from lung cancer and chronic obstructive pulmonary disease in New Mexico, 1958-82. *American Journal of Public Health*. 1988;78(9):1182-1186.
56. Samet JM. Epidemiology of lung cancer. *Chest*. 1992;103(1):20S-29S.
57. Samet JM. Epidemiology of lung cancer. In: Lenfant C, ed. *Lung biology in health and disease*. New York: Marcel Dekker, Inc.; 1994.
58. Sankila Rj, Karjalainen ES, Oksanen HM, Hakulinen TR, Teppo LHI. Relationship between occupations and lung cancer as analyzed by age and histologic type. *Cancer*. 1990;65(1651-1656).
59. Schimizu H, Preston-Martin S, Casagrande JT, Ross RK, Henderson BE. Epidemiological characteristics of adenocarcinoma of the lung in Los Angeles county. *National Cancer Institute Monograph*. 1982;62:161-164.
60. Schottenfeld D, Fraumeni Jr JF. Cancer Epidemiology and Prevention. New York: Oxford University Press; 1996.
61. Schrijvers CTM, Mackenbach JP. Cancer patient survival by socioeconomic status in The Netherlands: a review for six common cancer sites. *Journal of Epidemiology and Community Health*. 1994;48:441-446.

62. Schrijvers CTM, Coebergh J-WW, van der Heijden LH, Mackenbach JP. Socioeconomic variation in cancer survival in the Southeastern Netherlands, 1980-1989. *Cancer*. 1995;75(12):2946-2953.
63. Shimizu H, Preston-Martin S, Casagrande JT, Ross RK, Henderson bE, Pike MC. Epidemiological characteristics of adenocarcinoma of the lung in Los Angeles County. *National Cancer Institutre Monograph*. 1982;62:161-164.
64. Siemiatycki J, Wacholder S, Dewar R, Cardis E, Greenwood C, Richardson L. Degree of confounding bias related to smoking, ethnic group, and socioeconomic status in estimates of the associations between occupation and cancer. *Journal of Occupational Medicine*. 1988;30(8):617-624.
65. Simpson SG, Comstock GW. Lung cancer and housing characteristics. *Archives of Environmental Health*. 1983;38(4):248-251.
66. Stayner LT, Wegman DH. Smoking, occupation and histopathology of lung cancer: a case-control study with the use of the Third National Cancer Survey. *Journal of the National Cancer Institute*. 1983;70(3):421-425.
67. Teppo L. Cancer incidence by living area, social class and occupation. *Scandanavian Journal of Work and Environmental Health*. 1984;10:361-366.
68. Valatis J, Warren S, Gamble D. Increasing incidence of adenocarcinoma of the lung. *Cancer*. 1981;47:1042-1046.
69. van Loon AJM, Goldbohm RA, van den Brandt PA. Lung cancer: is there and association with socioeconomic status in the Netherlands? *Journal of Epidemiology and Community Health*. 1995;49:65-69.
70. van Loon AJM, Burg J, Goldbohm RA, van den Brandt P. Differences in cancer incidence and mortality among socio-economic groups. *Scandanavian Journal of Social Medicine*. 1995;23(2):110-120.
71. Vågerö D, Persson G. Occurence of cancer in socioeconomic groups in Sweden. *Scandanavian Journal of Social Medicine*. 1986;14(3):151-160.
72. Wallace RB, Doebbling BN, Last JM. Public Health and Preventive Medicine. Stamford: Appleton and Lange; 1998.
73. Weiss W, Boucot K, Seidman H, Carnaham W. Risk of lung cancer according to histologic type and cigarette dosage. *The Journal of the American Medical Association*. 1972;222(7):799-801.
74. Weiss W, Altan S, Rozenzweig M, Weiss WA. Lung cancer type in relation to cigarette dosage. *Cancer*. 1977;39(6):2568-2572.
75. Weiss W, Moser R, Auerbach O. Lung cancer in chloromethyl ether workers. *American Review of Respiratory Diseases*. 1979;120:1031-1037.
76. Williams RR, Horm JW. Association of cancer sites with tobacco and alcohol consumotion and socioeconomic status of patients: interview study from the Third National Cancer Survey. *Journal of the National Cancer Institute*. 1977;58(3):525-547.
77. Williams D, Collins C. US socioeconomic and racial differences in health: patterns and explanantions. *Annual Review of Sociology*. 1995;21:349-386.
78. Wu AH, Henderson BE, Thomas DC, Mack TM. Secular trends in histologic types of lung cancer. *Journal of the National Cancer Institute*. 1986;77(1):53-55.
79. Wynder El, Graham EA. Tobacco smoking as a possible etiologic factor in bronchogenic carcinoma. *The Journal of the American Medical Association*. 1950;113(4):329-336.

80. Young L. Cancer incidence and mortality in the United States 1973-1977. *National Cancer Institute Monograph.* 1981;57:1-1082.

APPENDICES

APPENDIX A1

LUNG AND BRONCHUS CANCER (Invasive)

INCIDENCE, MORTALITY, AND SURVIVAL RATES

By Year of Diagnosis/Death

All Races, Males and Females

Year of Diagnosis/Death

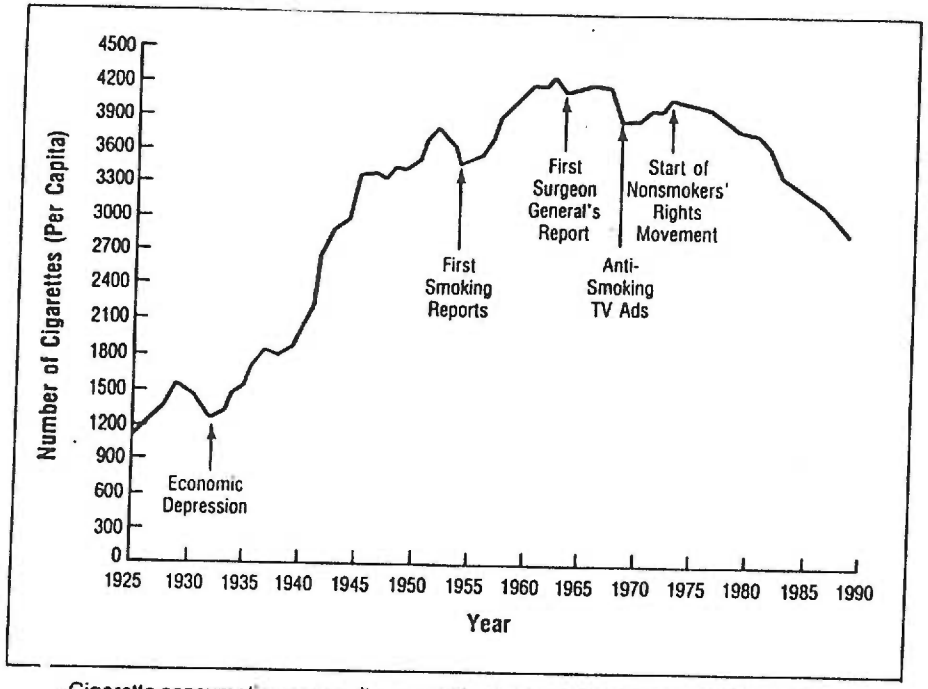
	1973	1974	1975	1976	1977	1978	1979	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990	1991	1992	1993	1994
Incidence																						
SEER	42.4	43.9	45.3	47.8	48.9	50.2	50.9	52.3	53.9	54.9	55.0	56.8	56.2	56.9	58.5	58.7	58.0	58.6	59.5	59.6	57.6	55.9
Mortality																						
U.S.	34.8	36.0	36.8	38.1	39.2	40.4	41.1	42.5	43.0	44.2	44.9	45.8	46.6	47.1	48.1	48.6	49.3	50.1	50.0	50.0	50.1	49.6
SEER	34.3	34.7	35.5	37.2	37.4	38.1	39.4	40.0	40.2	41.5	42.5	42.0	43.4	42.4	43.7	44.5	44.8	44.2	45.8	44.9	44.5	44.4
Relative Survival Rates (SEER)																						
1-year	31.5	34.3	34.1	36.1	37.0	38.5	37.4	38.2	37.7	39.1	39.6	39.0	39.7	40.5	39.9	40.4	40.6	40.6	41.3	41.0	40.3	
2-year	18.2	20.2	20.3	21.4	21.9	22.6	22.1	22.2	22.2	22.9	23.5	22.5	23.1	23.4	23.0	23.5	23.7	23.8	24.0	24.4		
3-year	13.6	15.5	15.4	16.8	16.7	17.4	17.1	17.0	16.9	17.9	18.1	17.5	17.9	17.9	17.2	17.8	17.8	18.2	18.7			
4-year	12.2	13.3	13.3	14.5	14.4	15.0	14.8	14.4	14.7	15.6	15.6	14.8	15.3	15.0	15.0	15.3	15.4	15.4				
5-year	10.9	12.1	12.0	13.0	13.1	13.6	13.5	12.9	13.2	13.9	14.2	13.2	13.8	13.4	13.5	13.6	14.1					
6-year	10.2	11.2	10.9	12.0	12.4	12.5	12.5	12.0	12.1	12.6	13.1	12.2	12.9	12.4	12.4	12.3						
7-year	9.5	10.7	10.3	11.5	11.6	11.7	11.9	11.2	11.1	12.0	12.1	11.3	11.9	11.6	11.3							
8-year	9.1	10.3	10.1	10.7	11.0	11.1	11.1	10.4	10.4	11.2	11.4	10.5	11.1	10.8								
9-year	8.7	9.7	9.8	10.2	10.5	10.5	10.4	9.8	9.7	10.6	10.6	9.8	10.5									
10-year	8.1	9.1	9.3	9.7	9.9	10.0	9.7	9.0	9.2	10.1	10.0	9.4										
11-year	7.7	9.0	9.1	9.1	9.5	9.6	9.3	8.5	8.9	9.7	9.6											
12-year	7.5	8.8	8.8	8.7	9.0	9.2	9.0	8.1	8.6	9.1												
13-year	7.1	8.5	8.4	8.3	8.7	9.0	8.6	7.6	8.1													
14-year	6.8	8.3	8.2	7.9	8.5	8.5	8.4	7.2														
15-year	6.4	7.9	7.9	7.5	8.1	8.1	7.8															
16-year	6.4	7.9	7.7	7.4	7.8	8.1																
17-year	6.4	7.6	7.2	7.2	7.7																	
18-year	6.0	7.6	7.0	6.9																		
19-year	5.5	7.6	7.0																			
20-year	5.3	7.3																				
21-year	5.1																					

Note: Incidence and mortality rates are per 100,000 and are age-adjusted to the 1970 U.S. standard population.

Survival rates are relative rates expressed as percents.

-SEER CANCER STATISTICS REVIEW 1973-1994

APPENDIX A2



Cigarette consumption per capita, age 18 and older, United States, 1925 to 1989.

-Public Health and Preventive Medicine, 1998

APPENDIX A3

LUNG AND BRONCHUS CANCER (Invasive)

INCIDENCE, MORTALITY, AND SURVIVAL RATES

By Year of Diagnosis/Death

All Races, Males

Year of Diagnosis/Death

	1973	1974	1975	1976	1977	1978	1979	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990	1991	1992	1993	1994
Incidence																						
SEER	73.2	74.5	76.2	79.4	80.7	82.0	81.5	84.4	84.5	85.1	84.1	86.5	83.9	83.6	85.2	83.3	82.1	81.6	81.8	81.7	78.3	73.9
Mortality																						
U.S.	62.4	64.1	65.1	66.7	68.2	69.5	70.1	71.5	71.7	72.9	72.9	73.9	74.1	74.3	75.1	74.8	74.7	75.3	74.5	72.9	72.6	70.9
SEER	61.4	60.4	61.9	63.7	63.1	64.2	65.5	65.9	65.3	65.6	67.0	65.3	66.5	64.8	65.5	65.8	65.4	63.9	64.5	63.2	62.1	61.6
Relative Survival Rates (SEER)																						
1-year	29.5	32.7	32.9	34.1	35.1	36.2	35.2	36.6	35.7	37.2	37.6	37.2	37.1	38.5	38.5	38.4	38.3	38.3	39.1	39.2	38.0	
2-year	16.7	18.9	19.1	19.8	20.4	20.7	20.4	20.9	20.3	21.2	21.4	20.9	21.2	21.3	22.0	22.1	22.3	21.6	22.0	23.2		
3-year	12.2	14.1	14.2	15.3	15.4	15.5	15.4	15.9	15.0	16.3	16.0	15.7	15.9	16.3	16.1	16.7	16.5	16.3	17.1			
4-year	11.0	12.0	12.2	13.2	12.9	13.1	13.3	13.5	13.0	13.9	13.7	13.1	13.2	13.4	14.0	13.9	14.2	13.7				
5-year	9.6	10.7	10.8	11.8	11.9	11.6	11.9	12.1	11.7	12.4	12.5	11.5	11.9	11.9	12.6	12.3	13.0					
6-year	8.9	9.8	9.7	10.7	11.3	10.6	11.0	11.3	10.7	11.2	11.4	10.6	11.0	11.0	11.6	11.0						
7-year	8.2	9.2	9.2	10.2	10.6	9.9	10.5	10.5	9.8	10.5	10.6	9.7	10.2	10.3	10.7							
8-year	7.8	8.9	9.0	9.5	10.1	9.4	9.7	9.6	8.9	9.8	9.9	8.9	9.5	9.4								
9-year	7.3	8.3	8.8	9.0	9.5	8.8	9.2	9.0	8.4	9.1	9.0	8.4	9.0									
10-year	6.7	7.8	8.4	8.6	8.9	8.2	8.7	8.3	7.9	8.7	8.5	8.0										
11-year	6.3	7.8	8.1	8.1	8.4	7.9	8.2	7.9	7.7	8.5	8.1											
12-year	6.1	7.7	7.8	7.8	7.8	7.6	7.9	7.3	7.4	8.1												
13-year	5.5	7.4	7.4	7.3	7.6	7.3	7.5	7.1	6.8													
14-year	5.2	7.0	7.2	7.0	7.4	6.9	7.5	6.8														
15-year	4.7	6.5	6.8	6.5	7.1	6.3	7.0															
16-year	4.7	6.5	6.7	6.3	6.7	6.3																
17-year	4.7	6.2	6.4	6.2	6.7																	
18-year	4.4	6.1	6.0	5.9																		
19-year	4.0	6.0	6.0																			
20-year	3.7	5.7																				
21-year	3.5																					

Note: Incidence and mortality rates are per 100,000 and are age-adjusted to the 1970 U.S. standard population.

Survival rates are relative rates expressed as percents.

APPENDIX A4

LUNG AND BRONCHUS CANCER (Invasive)

INCIDENCE, MORTALITY, AND SURVIVAL RATES

By Year of Diagnosis/Death

All Races, Females

Year of Diagnosis/Death

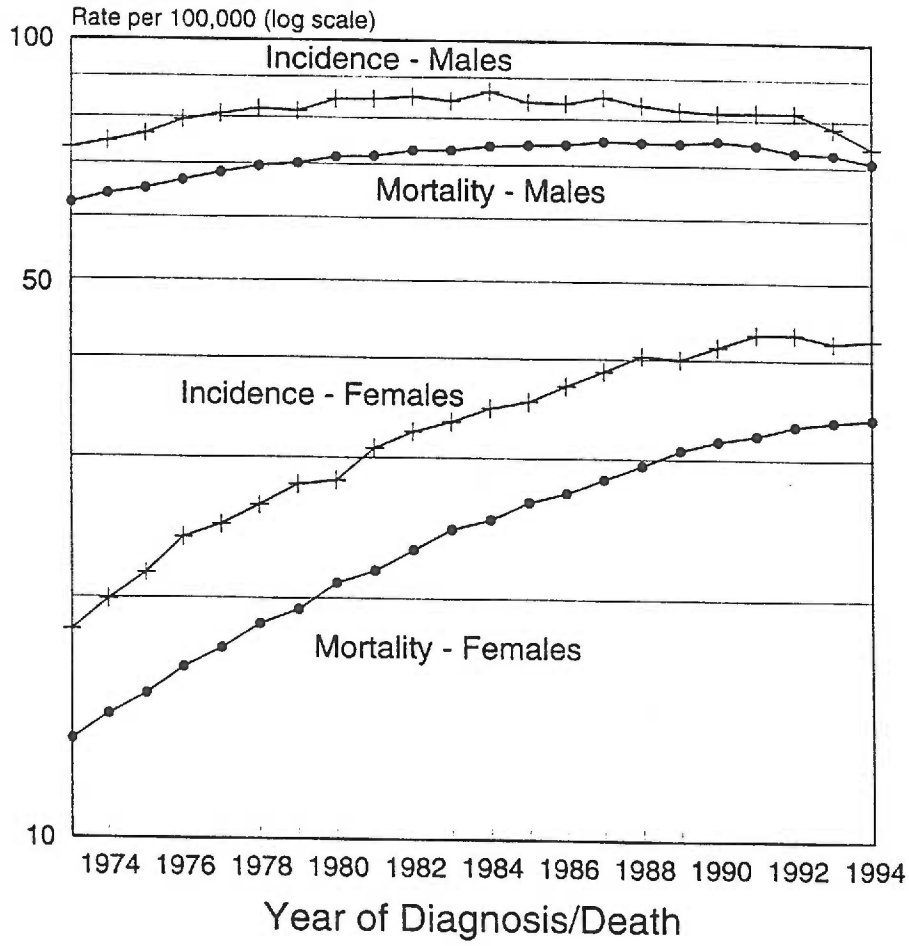
	1973	1974	1975	1976	1977	1978	1979	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990	1991	1992	1993	1994
Incidence																						
SEER	18.2	19.9	21.5	23.8	24.7	26.2	27.8	28.1	30.8	32.3	33.3	34.6	35.3	37.0	38.6	40.4	40.0	41.5	43.1	43.1	42.1	42.4
Mortality																						
U.S.	13.3	14.3	15.2	16.4	17.3	18.6	19.4	20.9	21.7	23.0	24.4	25.1	26.4	27.2	28.3	29.4	30.8	31.6	32.2	33.1	33.5	33.8
SEER	13.1	14.8	15.2	17.2	18.1	18.5	19.9	20.6	21.6	23.7	24.5	24.9	26.4	25.9	27.5	28.9	29.7	29.9	32.3	31.4	31.4	31.8
Relative Survival Rates (SEER)																						
1-year	38.3	39.4	37.4	41.4	41.9	44.4	42.7	42.2	42.2	43.2	43.6	42.6	44.8	44.0	42.5	43.6	44.3	44.1	44.8	43.8	43.7	
2-year	23.1	24.0	23.7	25.6	25.9	27.4	26.2	25.1	26.1	26.5	27.9	25.7	26.8	27.0	24.7	25.7	25.8	27.2	26.9	26.3		
3-year	17.9	19.5	18.9	20.8	20.1	22.1	21.0	19.5	20.8	21.2	22.4	20.9	21.6	20.6	19.1	19.6	19.8	21.1	21.2			
4-year	16.0	17.1	16.5	17.9	18.1	19.6	18.3	16.5	18.1	18.8	19.1	18.0	19.2	17.9	16.7	17.4	17.3	18.0				
5-year	14.8	16.0	15.0	16.1	16.0	18.3	17.1	14.8	16.4	16.9	17.4	16.3	17.3	15.9	14.9	15.6	15.7					
6-year	13.9	15.4	14.1	15.3	14.9	16.7	15.7	13.7	15.0	15.1	16.2	15.0	16.2	14.7	13.6	14.3						
7-year	13.3	14.7	13.2	14.5	14.2	15.7	14.9	12.7	13.8	14.8	14.8	14.2	14.8	13.8	12.2							
8-year	12.7	14.2	12.8	13.7	13.1	14.9	14.0	12.0	13.1	13.7	14.0	13.3	14.0	12.9								
9-year	12.5	13.3	12.2	12.9	12.7	14.3	13.0	11.5	12.2	12.9	13.2	12.1	13.1									
10-year	11.8	12.6	11.5	12.1	12.2	13.8	11.7	10.4	11.5	12.3	12.5	11.6										
11-year	11.4	11.9	11.4	11.2	11.9	13.1	11.5	9.7	11.2	11.8	12.0											
12-year	10.9	11.3	11.3	10.6	11.7	12.5	11.0	9.5	10.8	10.7												
13-year	10.8	11.3	10.9	10.4	11.1	12.4	10.7	8.6	10.2													
14-year	10.7	11.3	10.5	9.7	10.8	11.5	10.0	7.8														
15-year	10.4	11.1	10.3	9.6	9.9	11.4	9.1															
16-year	10.2	10.9	9.7	9.6	9.9	11.0																
17-year	10.1	10.7	8.8	9.2	9.4																	
18-year	9.7	10.7	8.8	8.9																		
19-year	8.6	10.7	8.6																			
20-year	8.5	10.5																				
21-year	8.3																					

Note: Incidence and mortality rates are per 100,000 and are age-adjusted to the 1970 U.S. standard population.

Survival rates are relative rates expressed as percents.

APPENDIX A5a

Cancer of the Lung & Bronchus
SEER Incidence & U.S. Mortality Rates, 1973-94
By Sex, All Races



Age-adjusted to 1970 Standard

APPENDIX A5b

LUNG AND BRONCHUS CANCER (Invasive)

U.S. MORTALITY RATES, AGE-ADJUSTED AND AGE-SPECIFIC RATES§, BY RACE AND SEX

	All Races			Whites			Blacks		
	Total	Males	Females	Total	Males	Females	Total	Males	Females
AGE-ADJUSTED RATES									
YEAR OF DEATH:									
1973	34.8	62.4	11.3	34.3	61.6	11.1	40.8	75.1	13.6
1974	36.0	64.1	14.3	35.5	63.2	14.3	42.5	78.5	14.2
1975	36.8	65.1	15.2	36.4	64.2	15.3	43.3	79.7	14.9
1976	38.1	66.7	16.4	37.7	65.7	16.5	44.7	82.0	15.9
1977	39.2	68.2	17.3	38.6	66.8	17.4	47.8	87.8	17.3
1978	40.4	69.5	18.6	39.9	68.2	18.7	48.5	88.8	17.9
1979	41.1	70.1	19.4	40.5	68.8	19.5	49.4	89.7	19.2
1980	42.5	71.5	20.9	41.9	70.0	21.0	51.8	92.7	21.4
1981	43.0	71.7	21.7	42.3	70.0	21.8	53.1	95.5	21.9
1982	44.2	72.9	23.0	43.6	71.3	23.2	54.6	97.9	23.0
1983	44.9	72.9	24.4	44.3	71.3	24.6	55.7	98.0	25.0
1984	45.8	73.9	25.1	45.1	72.0	25.4	56.9	102.1	24.5
1985	46.6	74.1	26.4	46.0	72.3	26.8	57.5	101.6	26.0
1986	47.1	74.3	27.2	46.4	72.4	27.5	58.3	102.6	27.0
1987	48.1	75.1	28.3	47.4	73.3	28.5	59.8	104.6	28.4
1988	48.6	74.8	29.4	48.0	73.0	29.8	59.9	104.1	29.3
1989	49.3	74.7	30.8	48.7	72.6	31.2	61.6	106.7	30.3
1990	50.1	75.3	31.6	49.6	73.2	32.0	62.6	107.1	31.8
1991	50.0	74.5	32.2	49.5	72.5	32.6	61.7	104.8	32.0
1992	50.0	72.9	33.1	49.4	71.0	33.5	61.6	102.5	33.4
1993	50.1	72.6	33.5	49.7	70.7	34.1	60.9	102.6	32.4
1994	49.6	70.9	33.8	49.2	69.2	34.4	60.1	99.4	33.2
AGE ADJUSTED RATES, 1990-94									
AGE AT DEATH:									
All ages	49.9	73.2	32.8	49.4	71.3	33.3	61.3	103.2	32.6
Under 65	21.9	29.2	15.3	21.4	27.8	15.4	31.1	48.8	17.2
65 and over	305.5	474.2	192.8	305.8	468.6	196.8	337.2	599.5	171.2
All ages (world std.)φ	39.0	56.0	25.9	38.5	54.4	26.2	49.1	81.2	26.3
AGE-SPECIFIC RATES, 1990-94									
AGE AT DEATH:									
0-4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0
5-9	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
10-14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
15-19	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.1	0.1
20-24	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
25-29	0.2	0.3	0.2	0.2	0.2	0.2	0.3	0.4	0.3
30-34	1.0	1.2	0.9	0.9	1.0	0.8	1.8	2.4	1.2
35-39	3.5	4.1	2.9	3.2	3.7	2.7	6.1	8.3	4.2
40-44	9.7	12.1	7.5	8.9	10.7	7.2	17.7	25.9	10.7
45-49	27.3	33.9	20.9	25.7	30.7	20.7	47.0	71.2	26.8
50-54	61.7	79.4	45.0	59.2	73.8	45.2	96.1	148.6	53.1
55-59	117.6	156.8	81.7	114.7	148.9	82.6	166.3	261.3	90.0
60-64	192.1	265.8	127.2	190.4	258.3	129.5	243.0	390.3	131.7
65-69	266.6	382.5	172.2	265.8	374.8	175.9	315.9	521.2	165.6
70-74	323.4	475.9	208.0	323.1	467.9	212.4	371.4	642.3	190.3
75-79	350.5	548.5	219.4	353.0	545.3	224.8	351.2	649.5	178.0
80-84	338.5	594.2	200.1	339.8	591.3	204.0	344.1	696.0	163.2
85+	257.3	522.8	155.3	257.0	524.3	155.7	261.6	534.1	149.2

§ NCHS public use tape. Rates are per 100,000 and are age-adjusted to the 1970 U.S. standard population, except where noted.

φ NCHS public use tape. Rates are per 100,000 and are age-adjusted to the world standard population.

APPENDIX A5c

LUNG AND BRONCHUS CANCER (Invasive)

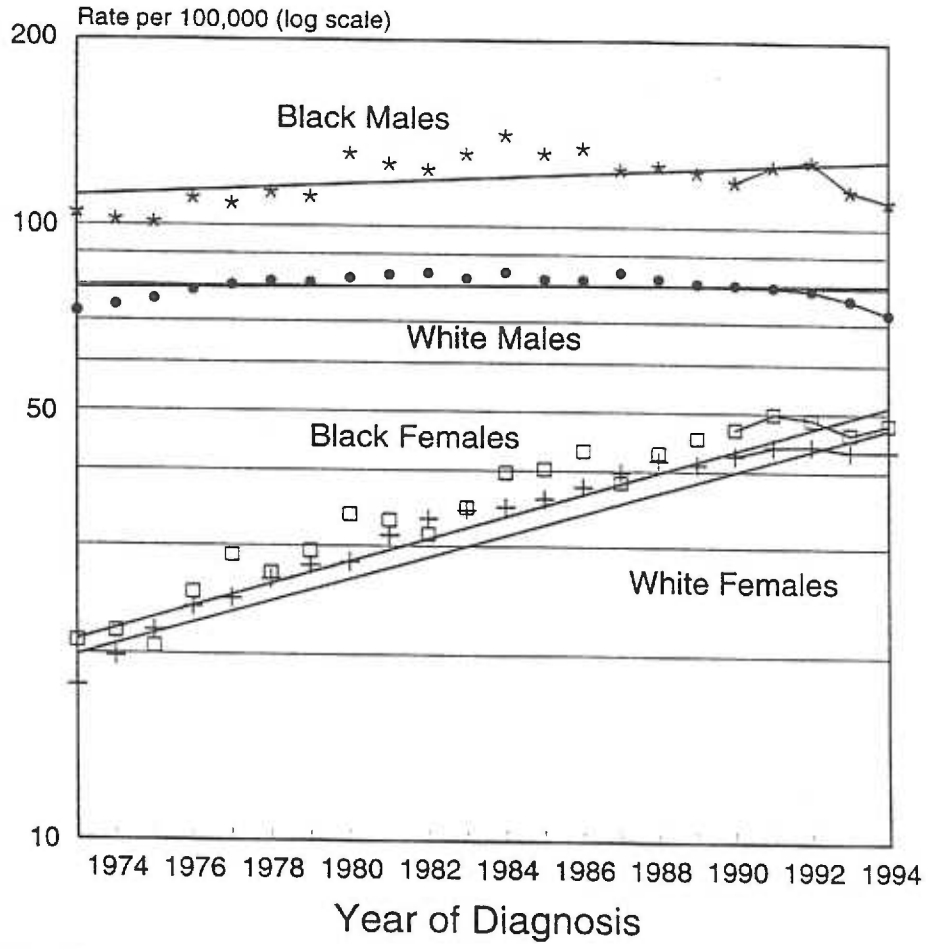
SEER INCIDENCE RATES, AGE-ADJUSTED AND AGE-SPECIFIC RATES§, BY RACE AND SEX

	All Races			Whites			Blacks		
	Total	Males	Females	Total	Males	Females	Total	Males	Females
AGE-ADJUSTED RATES									
YEAR OF DIAGNOSIS:									
1973	42.4	73.2	18.2	41.6	72.4	17.8	58.8	104.8	20.9
1974	43.9	74.5	19.9	43.4	74.1	19.8	58.9	102.0	21.7
1975	45.3	76.2	21.5	45.1	75.9	21.8	56.6	101.2	20.6
1976	47.8	79.4	23.8	47.2	78.4	23.8	62.6	110.9	25.2
1977	48.9	80.7	24.7	48.3	80.0	24.6	64.2	108.7	28.9
1978	50.2	82.0	26.2	49.7	81.1	26.5	65.0	113.3	27.1
1979	50.9	81.5	27.8	50.4	80.7	27.9	65.4	111.6	29.4
1980	52.3	84.4	28.1	51.2	82.2	28.2	76.0	131.0	33.8
1981	53.9	84.5	30.8	53.4	83.3	31.3	73.1	126.1	33.1
1982	54.9	85.1	32.3	54.6	83.8	33.3	71.1	123.5	31.4
1983	55.0	84.1	33.3	54.5	82.2	34.4	75.9	130.9	34.7
1984	56.8	86.5	34.6	55.7	84.2	34.8	82.8	140.6	39.7
1985	56.2	83.9	35.3	55.6	82.1	35.9	79.0	131.3	40.2
1986	56.9	81.6	37.0	56.3	81.9	37.6	81.4	134.1	43.2
1987	58.5	85.2	38.6	58.6	84.3	39.8	74.4	124.1	38.3
1988	58.7	83.3	40.4	58.8	82.2	41.7	77.8	125.7	42.9
1989	58.0	82.1	40.0	57.9	81.0	41.0	78.0	123.0	45.4
1990	58.6	81.6	41.5	58.7	80.7	42.5	76.7	118.7	47.0
1991	59.5	81.8	43.1	59.2	80.1	44.0	81.9	126.0	49.8
1992	59.6	81.7	43.1	59.0	79.2	44.2	82.2	128.4	48.8
1993	57.6	78.3	42.1	57.4	76.5	43.3	75.2	115.2	46.3
1994	55.9	73.9	42.4	55.7	72.6	43.3	74.4	110.6	48.0
AGE-ADJUSTED RATES, 1990-94									
AGE AT DIAGNOSIS:									
All ages	58.2	79.4	42.4	58.0	77.8	43.5	78.0	119.6	48.0
Under 65	26.7	32.7	21.2	26.1	30.9	21.5	42.0	60.5	26.8
65 and over	345.9	505.3	236.8	349.1	505.6	243.5	407.1	658.9	241.5
All ages (world std.)λ	45.9	61.4	33.9	45.6	59.9	34.7	63.5	95.8	39.5
AGE-SPECIFIC RATES, 1990-94									
AGE AT DIAGNOSIS:									
0-4	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0
5-9	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
10-14	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0
15-19	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.0	0.2
20-24	0.3	0.3	0.3	0.3	0.2	0.5	0.4	0.9	0.0
25-29	0.6	0.6	0.6	0.6	0.5	0.6	1.1	1.2	0.9
30-34	1.9	2.1	1.7	1.7	2.0	1.4	3.6	3.8	3.4
35-39	5.1	6.0	4.3	4.4	5.0	3.8	11.0	14.5	8.0
40-44	13.0	15.0	11.1	11.6	12.7	10.5	27.8	38.3	18.7
45-49	34.5	39.5	29.6	32.2	35.2	29.1	64.8	86.1	46.2
50-54	77.7	90.5	65.3	73.9	82.2	65.7	136.9	196.4	86.1
55-59	141.0	170.7	113.3	138.2	160.9	116.4	216.7	320.0	133.4
60-64	226.7	292.7	167.6	220.1	287.2	173.9	313.7	461.4	196.8
65-69	321.5	434.7	227.9	324.0	430.9	234.6	415.5	613.1	264.7
70-74	375.2	523.0	261.7	377.1	515.5	270.8	481.0	781.7	272.7
75-79	391.1	579.2	264.0	400.1	589.6	273.2	380.3	648.7	215.3
80-84	349.1	580.5	221.2	352.9	590.1	225.2	368.9	679.2	205.1
85+	233.4	467.2	142.6	229.6	473.5	138.7	227.6	424.1	143.3

§ SEER Program. Rates are per 100,000 and are age-adjusted to the 1970 U.S. standard population, except where noted.
λ SEER Program. Rates are per 100,000 and are age-adjusted to the world standard population.

APPENDIX A6

Cancer of the Lung & Bronchus
SEER Incidence, 1973-94
By Sex and Race



Age-adjusted to 1970 Standard
Recent trend, 1990-94, highlighted

APPENDIX A7

LUNG AND BRONCHUS CANCER (Invasive)

AGE-ADJUSTED SEER INCIDENCE RATES§, SEER MORTALITY RATES§, AND 5-YEAR RELATIVE SURVIVAL RATES¶
By Registry, Race and Sex

	All Races			Whites			Blacks		
	Total	Males	Females	Total	Males	Females	Total	Males	Females
SEER INCIDENCE RATES§, 1990-94									
REGISTRY									
Standard 9 registries	58.2	79.4	42.4	58.0	77.8	43.5	78.0	119.6	48.0
San Francisco-Oakland	57.2	73.2	45.2	58.4	71.7	48.6	80.8	115.0	55.1
Connecticut	59.0	78.8	45.2	58.6	77.5	45.6	67.6	104.4	41.6
Metropolitan Detroit	72.9	102.2	52.0	70.3	96.4	52.1	83.2	126.9	51.8
Hawaii	46.7	63.8	31.4	65.1	80.0	51.1	-	-	-
Iowa	57.2	83.6	37.6	56.8	82.9	37.4	-	-	-
New Mexico	40.5	55.2	29.0	42.2	57.3	30.4	-	-	-
Seattle-Puget Sound	62.6	81.0	48.6	62.9	80.6	49.4	78.3	117.8	41.2
Utah	27.5	40.0	17.5	27.4	39.5	17.6	-	-	-
Metropolitan Atlanta	65.4	96.4	44.1	63.9	90.3	45.8	69.6	118.2	38.9
San Jose-Monterey	51.0	66.5	39.7	53.4	68.3	42.8	59.5	83.9	38.2
Los Angeles	51.8	69.0	39.2	51.7	66.4	41.1	69.4	103.9	44.9
SEER MORTALITY RATES§, 1990-94									
REGISTRY									
Standard 9 registries	44.8	63.0	31.3	44.7	62.0	32.1	61.3	97.3	35.7
San Francisco-Oakland	44.5	57.6	35.0	45.9	56.8	38.0	62.8	92.3	41.3
Connecticut	44.0	61.3	32.1	43.7	60.2	32.4	51.9	85.6	28.7
Metropolitan Detroit	53.3	77.9	36.0	50.8	73.2	35.4	63.7	99.0	38.3
Hawaii	34.1	47.1	22.6	47.6	58.8	37.1	-	-	-
Iowa	45.0	67.8	28.3	44.7	67.3	28.2	-	-	-
New Mexico	35.2	49.4	24.1	36.6	51.4	24.9	-	-	-
Seattle-Puget Sound	48.7	65.1	36.5	49.4	65.3	37.5	56.1	89.7	25.8
Utah	22.0	32.5	13.7	21.9	32.1	13.7	-	-	-
Metropolitan Atlanta	52.4	81.0	33.2	50.6	74.7	34.4	60.8	110.0	30.3
San Jose-Monterey	39.2	51.7	28.8	41.2	55.4	31.2	50.8	76.7	32.0
Los Angeles	41.2	55.9	30.5	41.2	53.9	32.0	57.9	89.3	35.9
SURVIVAL RATES¶, 1986-91									
REGISTRY									
Standard 9 registries	13.8	12.5	15.7	14.1	12.7	16.1	11.1	10.5	12.2
San Francisco-Oakland	13.7	12.6	15.2	14.0	12.9	15.3	13.1	12.3	14.6
Connecticut	14.0	12.9	15.7	14.2	13.1	15.8	10.9	9.6	11.6
Metropolitan Detroit	14.3	12.9	16.4	15.4	13.8	17.8	10.7	10.5	10.9
Hawaii	15.4	14.6	16.7	16.7	15.3	18.6	-	-	-
Iowa	12.0	11.1	13.7	12.1	11.1	13.8	-	-	-
New Mexico	10.4	8.6	13.3	10.4	8.7	13.0	-	-	-
Seattle-Puget Sound	14.7	13.6	16.3	14.8	13.6	16.4	14.4	15.1	11.5
Utah	12.4	10.4	16.0	12.5	10.5	16.1	-	-	-
Metropolitan Atlanta	14.8	12.8	18.2	16.3	14.2	19.5	9.8	8.6	12.6

Note: San Jose-Monterey and Los Angeles are new SEER areas as of 1992 and their data are only used in this table and not other tables in this section.

§ SEER Program. Incidence rates are per 100,000 and are age-adjusted to the 1970 U.S. standard population.

δ NCHS public use tape. Mortality rates are per 100,000 and are age-adjusted to the 1970 U.S. standard population.

¶ Survival rates are relative rates expressed as percents and are based on follow-up of patients through 1994.

† The standard error of the survival rate is between 5 and 10 percentage points.

‡ The standard error of the survival rate is greater than 10 percentage points.

- Statistic could not be calculated or populations are inadequate.

APPENDIX A8

Percent distribution by major histologic type for cancer of the lung and bronchus.

Histology	1969–1971[a] All races (*n* = 4077)	White (*n* = 3695)	Black (*n* = 340)	1983–1987[b] All races (*n* = 20867)	White (*n* = 18303)	Black (*n* = 1795)
Females						
Small-cell carcinoma	14.5	14.6	13.5	20.2	21.0	16.5
Squamous cell, other and unspecified	21.5	21.1	26.7	18.9	18.6	23.2
Adenocarcinoma, NOS	23.1	22.8	23.5	29.7	29.4	28.6
	All races (*n* = 16078)	White (*n* = 14199)	Black (*n* = 1735)	All races (*n* = 39653)	White (*n* = 33316)	Black (*n* = 4588)
Males						
Small-cell carcinoma	13.4	13.9	9.7	16.6	17.3	11.6
Squamous cell, other and unspecified	38.3	37.7	43.5	31.3	30.8	35.9
Adenocarcinoma, NOS	12.9	12.9	13.5	22.7	22.5	22.7

[a] Third National Cancer Survey data, 1969-1971

[b] Cancer Statistics Review, 1973-1988

APPENDIX B1

HISTOLOGICAL CODES UTILIZED TO CHARACTERIZE THE OBSERVED LUNG CANCER CELL TYPES AND THAT OF COMBINATIONS.

00	unknown or clinical diagnosis only
05	hamartoma alone
10	carcinoma only, NOS
11	squam. cell ca., undiff. intermediate cell ca.
12	alveolar cell ca., undiff. large cell ca.
13	alveolar cell ca., undiff. small cell ca.
14	alveolar cell ca., undiff. unspec. ca.
15	squam. cell ca., undiff. large cell ca., undiff. small cell ca.
16	adenoca., undiff. large cell ca., alveolar cell ca.
17	adenoca., undiff. large cell ca., clear cell ca.
20	squamous cell carcinoma
21	squam. cell ca., adenoca., alveolar cell ca.
22	squam. cell ca., adenoca., undiff., unspec. ca.
23	squam. cell ca., adenoca., undiff. large cell ca.
24	squam. cell ca., adenoca., undiff. small cell ca.
25	squam. cell ca., adenoca., alveolar cell ca., mesothelioma
26	squam. cell ca., sarcoma
27	adenoca., sarcoma
28	small cell undiff. ca., sarcoma
29	adenoca. carcinoid type
30	adenocarcinoma
31	adenocanthoma (adenoca. with squamous metaplasia)
32	undiff. large cell ca. with squamous metaplasia
33	alveolar cell (bronchiolar) carcinoma
34	bronchial carcinoid
35	bronchial adenoma
36	adenoca., clear cell type
37	bronchial adenoma, adenocarcinoma
38	bronchial adenoma, alveolar cell ca., undiff. small cell ca.
39	bronchial adenoma, squamous cell ca.
40	undiff. large cell ca.
41	undiff. large cell ca. with squamous metaplasia
42	undiff. large cell ca., clear cell ca.
43	alveolar cell ca. with squamous metaplasia
45	undiff. clear cell ca.
46	mesothelioma, alveolar cell ca.
47	mesoth., adenoca., and alveolar cell ca.
48	mesoth., adenoca., and undiff. large cell ca.
50	undiff. small cell ca.
51	undiff. small cell ca. with squamous metaplasia
52	undiff. small cell ca., undiff. unspec. ca.

APPENDIX B2

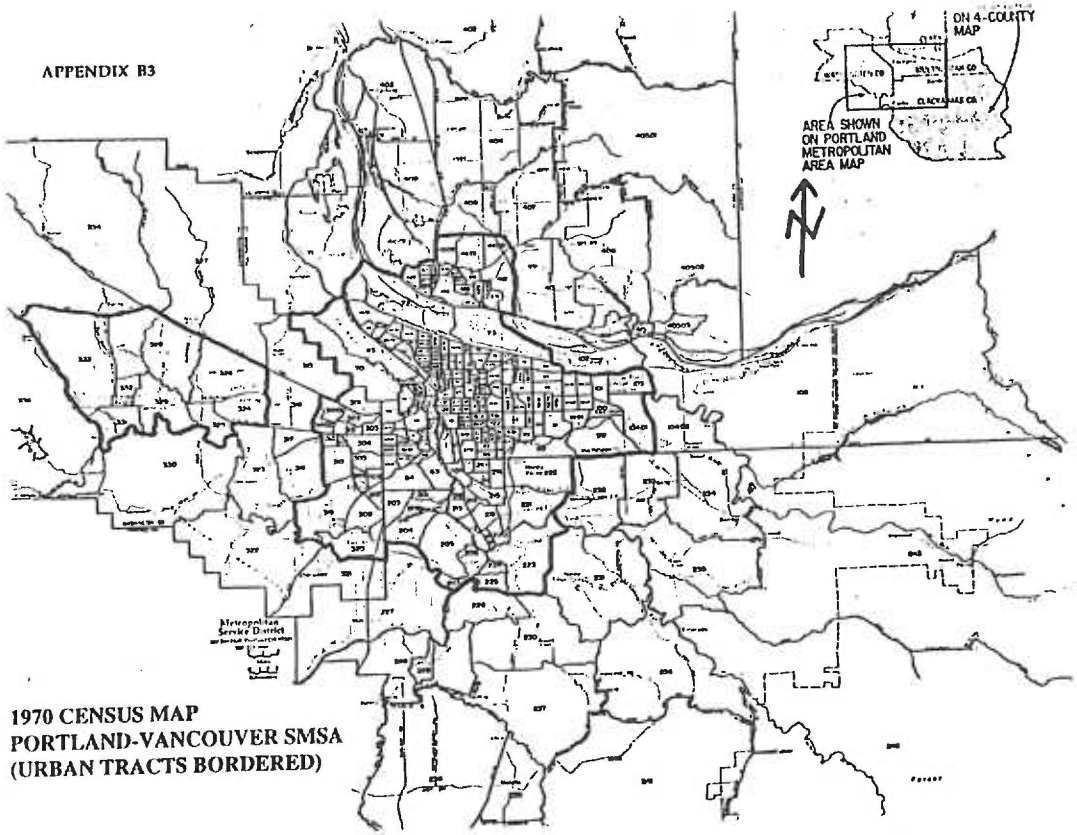
Histologic Groups Developed for Data Management

1. Total Lung Cancer
2. Squamous Cell
3. Adenocarcinomas
4. Clear Cell
5. Alveolar Cell
6. Undifferentiated Large Cell
7. Undifferentiated Small Cell
8. Undifferentiated Cancer
9. Sarcomas
10. Mesothelioma
11. Unknown
12. Combination-(Adenocarcinomas + Clear Cell + Alveolar Call)
13. Squamous alone[1]
14. Adenocarcinoma alone
15. Clear cell alone
16. Alveolar cell alone
17. Undifferentiated Large Cell alone
18. Undifferentiated Small Cell alone
19. Undifferentiated Cancer alone
20. Mesothelioma alone
21. Sarcomas alone
22. Other

---

[1]. "Alone" signifies that this was the only cell type observed.

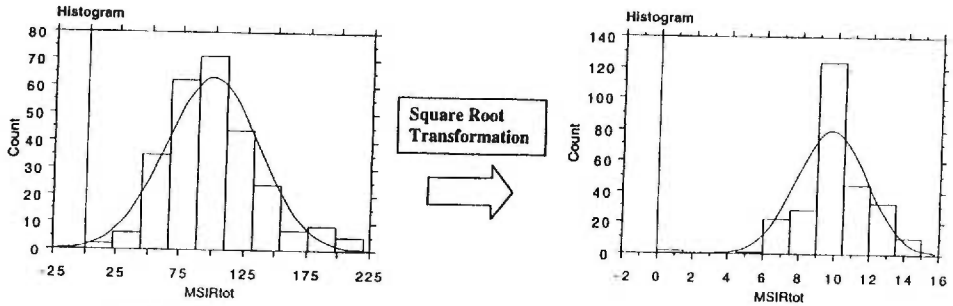
APPENDIX B3



1970 CENSUS MAP
PORTLAND-VANCOUVER SMSA
(URBAN TRACTS BORDERED)

APPENDIX B4

Figures showing the pre and post transformation distributions of the male standardized incidence ratio (SIRs) for total lung cancer (as represented by the variable MSIRtot) in Portland, Oregon-Vancouver Washington SMSA, 1963-1977.



Descriptive Statistics

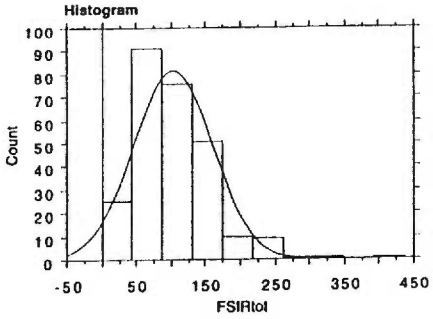
	MSIRtot
Mean	99.785
Std. Dev.	36.787
Std. Error	2.260
Count	265
Skewness	.607
Kurtosis	.777
Median	95.000

Descriptive Statistics

	MSIRtot
Mean	9.826
Std. Dev.	1.977
Std. Error	.121
Count	265
Skewness	-.668
Kurtosis	3.794
Median	10.000

APPENDIX B5

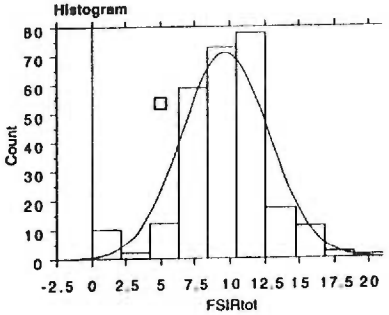
Figures showing the pre and post transformation distributions of the female standardized incidence ratio (SIRs) for total lung cancer (as represented by the variable FSIRtot) in Portland, Oregon-Vancouver Washington SMSA, 1963-1977.



Descriptive Statistics

	FSIRtot
Mean	103.143
Std. Dev.	56.452
Std. Error	3.468
Count	265
Skewness	1.289
Kurtosis	4.631
Median	96.000

Square Root Transformation

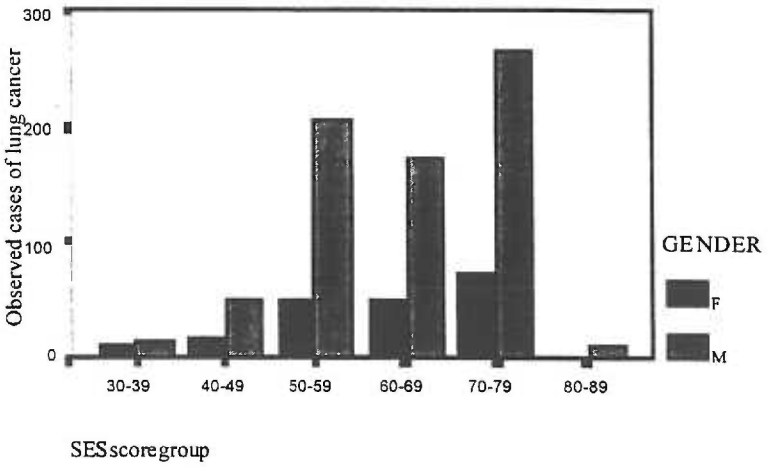


Descriptive Statistics

	FSIRtot
Mean	9.683
Std. Dev.	3.112
Std. Error	.191
Count	265
Skewness	-.632
Kurtosis	2.432
Median	10.000

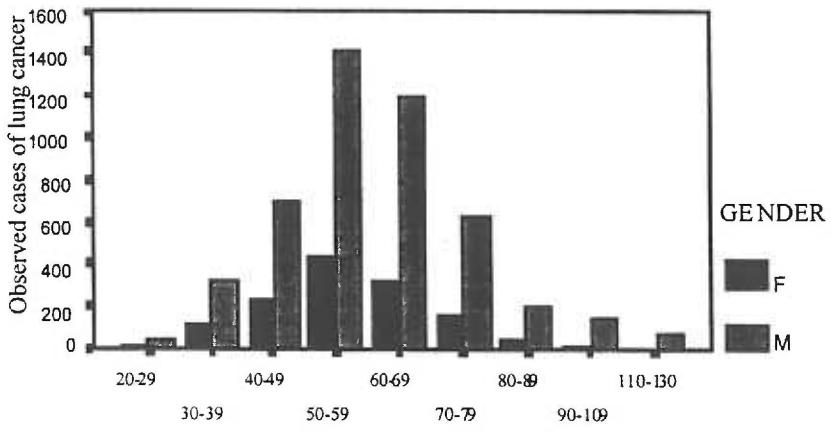
APPENDIX C1a

The distribution of lung cancer cases by sex in the rural census tracts in the Portland, Oregon-Vancouver, Washington SMSA, 1963-1977.



APPENDIX C1b

The distribution of lung cancer cases by sex in the urban census tracts in the Portland, Oregon-Vancouver, Washington SMSA, 1963-1977.



APPENDIX C2

Table showing the β-values (±Standard error) for the remaining histogroups for simple regression models describing census tract correlations between lung cancer cell type incidence and SES Score in males and females in the Portland, Oregon-Vancouver, Washington SMSA, 1963-1977.

Cancer Incidence§ (Y)	Environmental Variable SES Score					
	Male			Female		
	B	R^2	p◊	B	R^2	p◊
Total lung cancer	0.043±0.008*	0.103	0.00	0.022±0.013	0.011	0.09
Squamous cancer	0.066±0.011*	0.115	0.00	0.024±0.025	0.003	0.34
Adenocarcinoma	0.017±0.016	0.004	0.29	-0.022±0.024	0.003	0.36
Clear cell	0.093±0.041*	0.109	0.03	0.003±0.038	0.000	0.93
Alveolar cancer	0.014±0.029	0.001	0.62	-0.076±0.033*	0.019	0.02
Undifferentiated large	0.028±0.022	0.006	0.21	-0.028±0.032	0.003	0.38
Undifferentiated small	0.046±0.017*	0.026	0.01	0.035±0.030	0.005	0.24
Undifferentiated cancer	0.042±0.019*	0.019	0.03	0.045±0.030	0.008	0.14
Sarcomas	-0.029±0.041	0.002	0.49	-0.026±0.044	0.001	0.57
Mesotheliomas	0.021±0.038	0.001	0.58	0.079±0.041	0.014	0.05
Unknown	0.048±0.021*	0.019	0.02	-0.028±0.033	0.003	0.40
Combination	0.011±0.014	0.002	0.43	-0.035±0.023	0.009	0.13
Squamous Subtype	0.066±0.013*	0.091	0.00	0.007±0.026	0.000	0.80
Adenocarcinoma Subtype	0.019±0.019	0.004	0.30	-0.019±0.026	0.002	0.46
Clear cell Subtype	-0.024±0.035	0.002	0.49	0.003±0.038	0.000	0.92
Alveolar Subtype	-0.180±0.032	0.001	0.58	-0.066±0.035*	0.014	0.05
Undifferentiated large Subtype	0.033±0.026	0.002	0.21	-0.030±0.033	0.003	0.37
Undifferentiated cancer Subtype	0.042±0.018*	0.020	0.02	0.055±0.034	0.010	0.11
Undifferentiated small Subtype	0.035±0.023	0.009	0.13	0.042±0.031*	0.007	0.02
Sarcoma Subtype	-0.012±0.011	0.001	0.70	—	—	—
Mesothelioma Subtype	0.015±0.040	0.001	0.70	-0.065±0.041	0.009	0.12
Other	0.048±0.046	0.004	0.29	—	—	—

* $p<0.05$ (statistically significant)
◊ p value for the entire simple regression model

APPENDIX C3

Table showing the β-values (±Standard error) for the remaining histogroups for multiple regression models describing census tract correlations between lung cancer cell type incidence and SES Score in males in the Portland, Oregon-Vancouver, Washington SMSA, 1963-1977.

Cancer Incidence§ (Y)	Environmental Variables				
	SES Score	Urbanicity	Ses Score*Urbanicity	R^2	p◊
Total lung cancer	-0.010±0.026	-3.237±1.766	0.062±0.027*	0.137	0.00
Squamous	-0.013±0.038	-5.112±2.582*	0.090±0.040*	0.139	0.00
Adenocarcinoma	-0.076±0.053	-6.081±3.591	0.105±0.055	0.022	0.12
Clear cell	-0.000±0.141	7.991±9.553	0.092±0.147	0.026	0.07
Alveolar	-0.154±0.098	-11.015±6.656	0.190±0.103	0.018	0.20
Undifferentiated large	-0.101±0.076	-9.184±5.158	0.014±0.080	0.018	0.19
Undifferentiated small	-0.033±0.059	-5.092±3.979	0.090±0.061	0.038	0.02
Undifferentiated cancer	0.008±0.063	-3.232±4.306	0.057±0.066	0.023	0.11
Sarcomas	-0.004±0.139	1.147±9.463	-0.032±0.146	0.003	0.85
Mesotheliomas	-0.114±0.128	-8.530±8.690	0.156±0.134	0.010	0.47
Unknown	-0.002±0.071	-3.595±4.805	0.088±0.074	0.045	0.01*
Combination	-0.087±0.048	-6.413±3.249	0.110±0.050	0.025	0.08
Squamous Subtype	-0.023±0.042	-5.188±2.882	0.105±0.044	0.140	0.00*
Adenocarcinoma Subtype	-0.044±0.065	-3.786±4.393	0.074±0.068	0.140	0.29
Clear cell Subtype	-0.000±0.120	2.476±8.134	-0.021±0.125	0.004	0.77
Alveolar Subtype	-0.069±0.108	-2.672±7.363	0.064±0.114	0.007	0.61
Undifferentiated large Subtype	-0.032±0.090	-4.288±6.112	0.074±0.094	0.009	0.51
Undifferentiated cancer Subtype	0.083±0.078	4.628±5.281	-0.043±0.081	0.026	0.08
Undifferentiated small Subtype	-0.026±0.062	-3.886±4.190	0.076±0.065	0.031	0.04
Sarcoma Subtype	-0.281±0.105	-20.895±7.156	0.821±0.110	0.045	0.01
Mesothelioma Subtype	-0.122±0.135	-8.943±9.171	0.156±0.141	0.007	0.62
Other	-0.135±0.155	-12.814±10.536	0.201±0.163	0.010	0.45

* $p<0.05$ (statistically significant)
◊ p value for the entire multiple regression model

APPENDIX C4

Table showing the β-values (±Standard error) for the remaining histogroups for multiple regression models describing census tract correlations between lung cancer cell type incidence and SES Score in females in the Portland, Oregon-Vancouver, Washington SMSA, 1963-1977.

Cancer Incidence§ (Y)	Environmental Variables				
	SES Score	Urbanicity	Ses Score*Urbanicity	R^2	p◊
Total lung cancer	-0.014±0.044	-2.573±2.971	0.038±0.046	0.014	0.31
Squamous	0.026±0.086	0.792±5.853	0.002±0.090	0.007	0.62
Adenocarcinoma	0.007±0.081	1.242±5.529	-0.038±0.085	0.009	0.48
Clear cell	-0.00±0.129	0.254±8.793	0.007±0.136	0.001	0.97
Alveolar cancer	-0.135±0.113	-4.590±7.694	0.061±0.119	0.021	0.13
Undifferentiated large	0.005±0.107	0.797±7.285	-0.048±0.112	0.015	0.27
Undifferentiated small	-0.062±0.101	-5.747±6.846	0.114±0.106	0.016	0.24
Undifferentiated cancer	-0.069±0.102	-5.890±6.911	0.140±0.107	0.038	0.02
Sarcomas	-0.072±0.151	-3.120±10.245	0.053±0.158	0.002	0.93
Mesotheliomas	0.021±0.140	-3.773±9.477	0.066±0.146	0.015	0.26
Unknown	-0.082±0.113	-4.044±7.678	0.057±0.118	0.004	0.80
Combination	-0.018±0.079	0.502±5.355	-0.024±0.083	0.013	0.32
Squamous Subtype	-0.012±0.088	-0.890±5.981	0.024±0.092	0.002	0.92
Adenocarcinoma Subtype	0.048±0.089	3.713±6.072	-0.082±0.094	0.013	0.33
Clear cell Subtype	-0.00±0.129	0.254±8.793	0.007±0.136	0.001	0.97
Alveolar Subtype	-0.114±0.118	-2.244±8.022	0.058±0.124	0.019	0.17
Undifferentiated large Subtype	-0.059±0.113	-3.340±7.700	0.023±0.119	0.011	0.42
Undifferentiated small Subtype	-0.066±0.104	-6.796±7.049	0.124±0.109	0.015	0.26
Undifferentiated cancer Subtype	-0.017±0.144	-2.913±7.772	0.094±0.120	0.031	0.04
Sarcoma Subtype	—	—	—	—	—
Mesothelioma Subtype	0.021±0.140	-2.147±9.498	0.055±0.147	0.012	0.35
Other	—	—	—	—	—

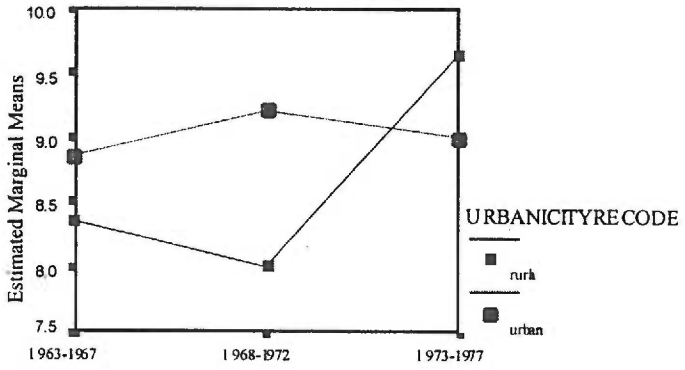
* $p<0.05$ (statistically significant)

◊ p value for the entire multiple regression model

APPENDIX C5

Estimated Marginal Means of Squamous cell cancer incidence (SIR)

For Males

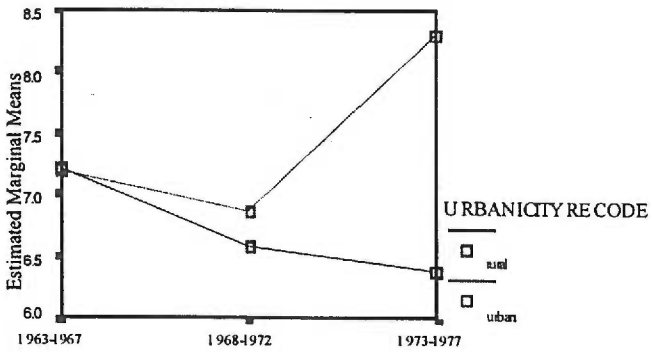


Time F= 0.303, p= 0.739
Time*Urbanicity F= 1.232, p= 0.293
SES was controlled for in this model

APPENDIX C6

Estimated Marginal Means of Undifferentiated small cell lung cancer incidence (SIR)

For Males

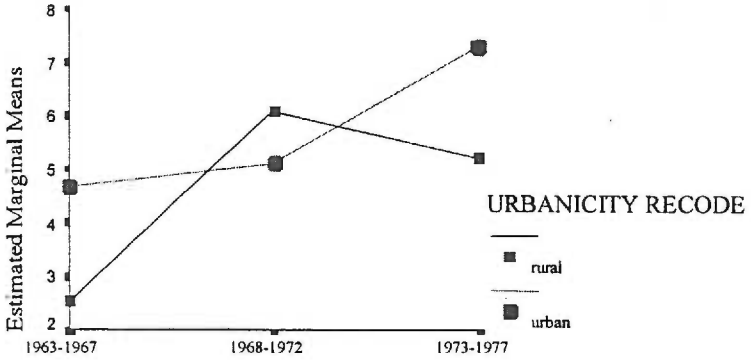


Time F= 0.812, p= 0.445
Time*Urbanicity F= 2.128, p= 0.120
SES Score was controlled for in this model

APPENDIX C7

Estimated Marginal Means of Squamous cell lung cancer incidence(SIR)

For Females

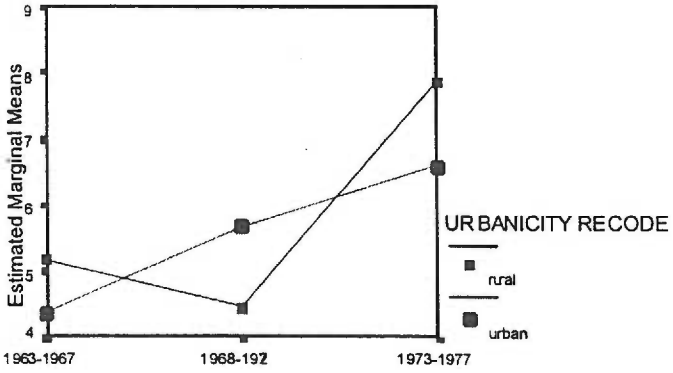


Time F= 0.537, p=0.585
Time*Urbanicity F=1.161, p=0.314
SES Score was controlled for in this model

APPENDIX C8

Estimated MarginalMeans of Adenocarcinoma lung cancer incidence (SIR)

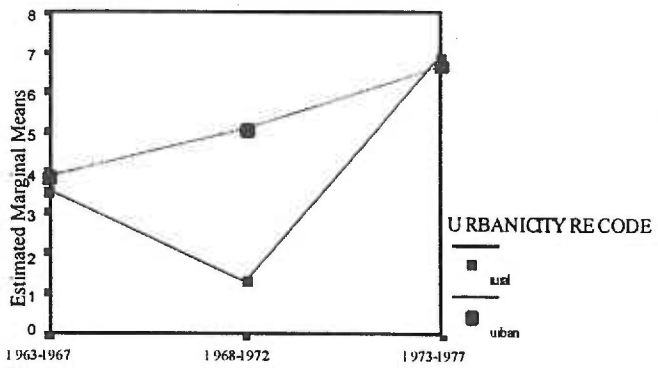
For Females



Time F= 0.537, p= 0.585
Time*Urbanicity F= 1.161, p= 0.314
SES Score was controlled for in this model

APPENDIX C9

Estimated Marginal Means of Undifferentiated small cell lung cancer incidence (SIR) For Females



Time F= 0.339, p= 0.712
Time*Urbanicity F= 0.905, p= 0.405
SES Score was controlled for in this model