Discussion

Even though the relationship between income and use of care has been moving toward equalization, blacks, persons receiving Medicaid, and, to a lesser extent, low-income persons still have distinctive patterns of obtaining care. The reasons for the higher reporting of places as a regular source for these groups is not totally clear. One explanation could be that there are fewer physicians in black areas. Another factor, relevant both to people with Medicaid and blacks, since many blacks have Medicaid, is that physicians are reluctant to accept Medicaid clients due to the restricted fee schedules. A different explanation that some sources have discussed is that certain types of places provide more open access to health care, including care in the evening and weekends which may be the only time that certain groups have available to visit physicians. This is particularly true of neighborhood health centers and emergency rooms. In addition, in the case of neighborhood health centers, there may be greater cultural congruence between the client and the delivery mode.[5-7]

Recent studies have concentrated attention on looking at socioeconomic differentials in use of care, but we also need to examine differences in the way care is received, the quality of the care, and whether people are receiving care in the manner they prefer. Different patterns, if they are the desires of certain groups, are both acceptable and desirable. Different patterns, if they are due to a lack of choice, and thus imply different medical care systems for different classes, would not be an acceptable goal for our medical care system. Further research into this question is indicated.

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Lung Cancer Mortality and Urban Air Pollution

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Over the past three decades cigarette smoke has been established as the major environmental inhalant causing lung cancer.[1] A small proportion of lung cancers are clearly attributable to occupational agents, including asbestos,[2] radiation,[3] and chemicals.[4] In addition, efforts have been made to link air pollution and lung cancer as cause and effect but the data are not very convincing.[5-7]

Since lung cancer has a long induction-latent period, its incidence should be examined in relation to environmental exposures years to decades before diagnosis. Available data in Philadelphia were examined with this in mind by studying the geographic variation of lung cancer mortality rates within the city in relation to the earliest obtainable information on the geographic variation in air pollution.

Philadelphia is divided geographically into ten health districts. Death certificate data were obtained on computer tape for the years 1968 through 1972 from the Philadelphia Department of Public Health by age, race, sex, and census tract of residence as well as underlying cause of death. Population data were obtained from the 1970 U.S. Census tapes by age, race, sex, and census tract of residence. Census tract data were combined into data for health districts.

Mortality rates were calculated as the average annual number of deaths per 100,000 and limited to age 30 and over since almost all lung cancer deaths occurred in this age group. The major proportion of cases (2,656) occurred in white males so rates were confined to this population segment. In two of the health districts, numbers 5 and 6, the numbers of white males aged 30 and over were small so the data were combined for the two districts. Rates were age-adjusted to the age distribution of all Philadelphia white males in 5-year age groups.

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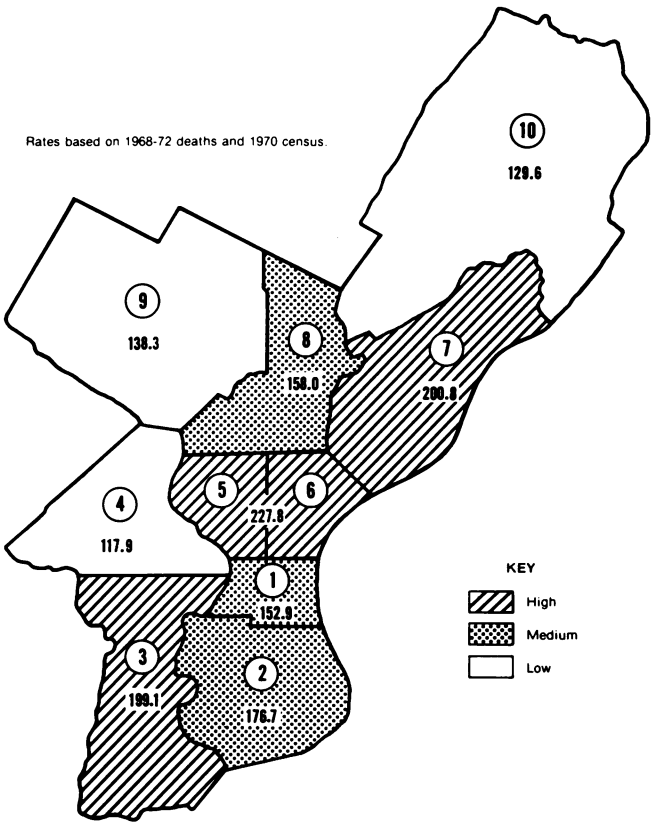

FIGURE 1—Annual Age-Adjusted Mortality Rates for Lung Cancer (no./100,000) for White Males Aged 30 and Over by Health District in Philadelphia.

Figure 1 is a map of Philadelphia showing the ten health districts with numbers 5 and 6 combined into a single area. The lung cancer mortality rate is shown for each of the nine areas. The variation was statistically highly significant (chi square = 118.70, d.f. = 8, $P < 0.0005$). The three areas with the highest rates are distinguished from the three with intermediate rates and the three with the lowest rates. Those areas with the high and intermediate rates form a broad band from the southern section into the near north-eastern section along the eastern border of the city.

These portions of Philadelphia have the following characteristics. Health District 1 is the central business area with heavy automotive traffic and high building density retarding dispersion. Health Districts 5, 6, and the lower portion of 8 are low income residential and commercial regions. Health District 7 in the near northeast section is heavily industrialized with a power generating station, municipal incinerator, chemical plants, sewage treatment plant, metal smelters, and rendering plants. Health District 2 and much of District 3 in the southern tip of the city form the second major industrial section of Philadelphia with emission sources including oil refineries, gasoline bulk loading terminals, U.S. Naval Shipyard, airport, salvage operations, municipal incinerator, two sewage treatment plants, and heavy automotive traffic. Health Districts 4, 9, and 10, with the lowest lung cancer mortality rates, are largely residential areas in the west, northwest, and far northeast sections of the city.

The only air pollution data available prior to the mortality data in enough detail to draw concentration isopleths were those for settled dust (ASTM Standard Method D1739) and sulfation rate (ASTM Standard Method D2010). Settled dust is given in terms of equivalent tons of particulate matter deposited per square mile per month and sulfate in terms of equivalent milligrams of SO_3 reacting per 100 square centimeters per day.

In 1960 there were eight sampling stations for particulate and nine for sulfate in Philadelphia. By 1969 there were 44 for each pollutant. Concentration isopleths were drawn on a map of the city based on data available in 1960 with continuity established by reference to concentration patterns and trends observed from the more numerous samplers employed since 1969. Figures 2 and 3 show concentration isopleths for these pollutants in 1960. It is obvious that the most heavily polluted areas in that year were associated with the highest lung cancer mortality rates a decade later.

No information on smoking habits could be found for white males by geographic section so no correction of the rates could be made for this important etiologic factor.

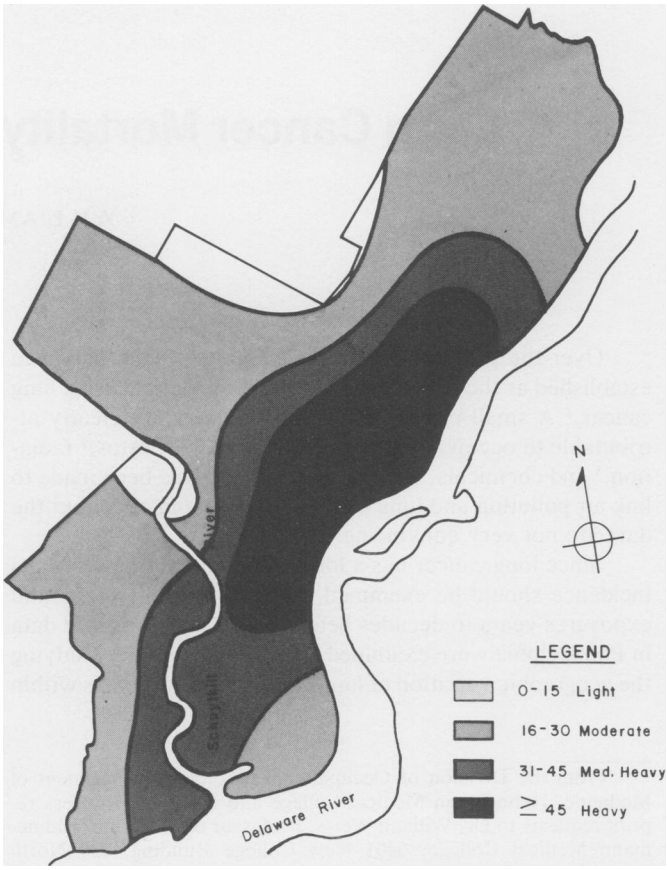

FIGURE 2—Concentration Isopleths for Total Settled Dust in Tons per Square Mile per Month for Philadelphia in 1960.

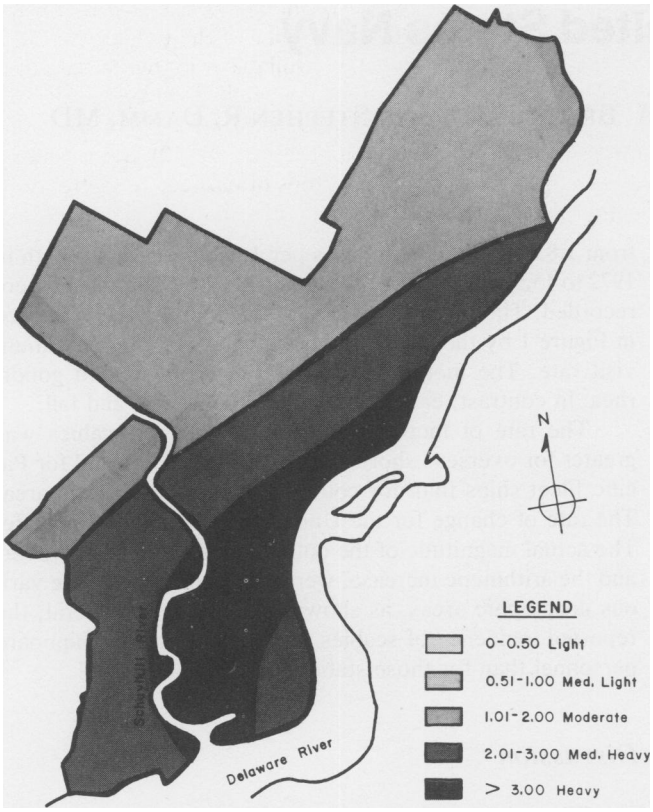

FIGURE 3—Concentration Isopleths for Sulfation Rate in Milligrams of SO_3 per 100 Square Centimeters per Day for Philadelphia in 1960.

The lung cancer mortality rates for white females aged 30 and over, age-adjusted to the white male distribution, ranged from 24.0 to 38.7 and there was a fair correlation between the male and female rates by health district (Spearman rank correlation coefficient = 0.70, t = 2.59, d.f. = 7, $P < 0.05$). The highest male:female ratios appeared in Health Districts 2, 3, and 7, the most heavily industrialized sections of the city. This suggests an occupational factor.

While the data shown in Figures 1–3 are consistent with the hypothesis that air pollution is a factor in the causation of lung cancer, interpretation of a cause-and-effect relationship is unjustified because other important factors have not been taken into account. Smoking and occupational exposure are the most significant of these. The long induction-latent period of lung cancer injects considerable uncertainty since, although the material presented provides for an 8- to 12-year lag between air pollution data and mortality data, population mobility in recent decades may be a serious confounding factor.

Another difficulty in evaluating the air pollution—lung cancer relationship is uncertainty as to which pollutants are carcinogenic and their relationship to those for which measurements are available. There also is a problem in estimating cumulative exposure. Until these difficulties are resolved, the role of air pollution in the causation of lung cancer and the role of other factors such as population density and socioeconomic status will remain unclear.

Long-term prospective investigations are most desirable but careful detailed case-control studies would be more productive than current searches for gross statistical correlations.

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ACKNOWLEDGMENTS

The author is grateful to Esther Wollin, DSc, Director of the Division of Health Program Analysis, Community Health Services of the Philadelphia Department of Public Health, for mortality data; the Delaware Valley Regional Planning Commission for 1970 census data; and Robert T. Ostrowski, Director of Staff Services, Air Management Services of the Philadelphia Department of Public Health for Figures 2 and 3. This research supported in part by the Rohm and Haas Company.