Health and the Urban Environment

V. Air Pollution and Illness in a Normal Urban Population

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THE DIFFICULTY in demonstrating a meaningful relationship between usual urban levels of air pollution and illness in "normal" populations has been a frustrating experience for investigators in this area.

Much of this difficulty results from the fact that the host response customarily studied—some symptom of disease noted by a subject—may also be the result of many other factors. That portion of symptoms in a population which is related to air pollution must somehow be separated from the background count of the same symptoms resulting from infection, allergy, and numerous other causes. Our problem is to determine if there is a regular relationship, either immediate or delayed, between the appearance of an illness symptom in a population and some measure of air pollution.

Materials and Methods

The population studied was a reasonable cross section of New York City residents described in detail elsewhere.[1] All 1,822 persons lived within a half-square-mile area of reasonably homogeneous air pollution. A daily record of the appearance of each of 21 symptoms of disease (as well as much other health information) was obtained by weekly interview. Each participant was expected to remain in the study for a full year and the average length of time each person participated in fact was 48 weeks. This analysis is based on the daily illness experience of the 1,090 adults over the age of 15. These represent 35,400 person-weeks or 247,800 person-days of observation on this population.

A monitoring station was established in the center of our study area to supply appropriate meteorologic and air pollution data.[1] To determine whether the levels of air pollution as measured at our station might be related to variations in health in our population, we carried out the following analysis. We selected two symptoms—cough and eye irritation—which might be expected to have some relation to air pollution. Persons with chronic coughs were excluded from this analysis as were all children under age 15. The presence of either symptom for each of the 1,054 days of the study was determined by a positive answer to the questions "Did you have a cough?" and "Did you have itching, burning or tearing of your eyes?" The population was analyzed in three groups: heavy smokers (those smoking 20 or more cigarettes daily), moderate and nonsmokers (using 0-19 cigarettes daily), and total adults (age 15 and over).

As indicators of the levels of air pollution to which the population was exposed, we used sulfur dioxide as measured by the Davis conductivity instrument expressed in parts per million and particulate density as measured by the A.I.S.I. sampler and expressed in COH units. For the purpose of this analysis 24-hour averages were used.

Correlation coefficients between each symptom

Cross-Correlations Between Two Symptoms and Two Pollutants in One-Month Periods With Proportion of Cross-Correlations Showing an Increase or Decrease From Lag of Zero to One Day (Adults, 15 Years and Over)

	Symptoms			
	Cough		Eye Irritation	
	Increase	Decrease	Increase	Decrease
Pollutants				
SO_2	23/29	6/29	11/29	18/29
Particulate density	20/29	8/29 *	14/29	15/29

$$\text{Each Ratio} = \frac{\text{No. showing increase or decrease}}{\text{Total No. of cross-correlations}}$$

* One correlation between cough and particulate density showed no difference between value for lag 0 and lag 1.

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DISCUSSION

DR. BOREN: There is a study by Dr. Reid in which intra-alevolar installation of various mucins is followed by the development of dark-containing pigment within alveolar macrophages. My question to Drs. Thurlbeck and Myrvik, since the question of pigment has repeatedly been discussed for the last several years, is whether or not this might not represent failure of removal of surfactant or else retrograde flow of mucin to the alveolar level.

DR. THURLBECK: I'm not precisely sure what this question was. You wish to know in what way is the pigment that Tom is describing related to surfactant?

DR. BOREN: You don't know what this pigment is related to. Is there a possible origin of this pigment from phagocytosis of mucin or such substances at the alveolar level in the disease state?

DR. THURLBECK: Well, to be honest, nobody can tell you the answer insofar as the pigment is not known, but if one is allowed to make an inspired or uninspired guess, the answer is no.

DR. ANDERSON: Dr. Myrvik, have you an inspiration?

DR. MYRVIK: I know of no information that would suggest that it might originate from surfactant. There are various types of pigments that seem to accumulate in alveolar macrophages, but we have no information how long they might persist.

DR. KILBURN: I'd like to ask Dr. Petty if cough did not decrease, did sputum production decrease in the people who stopped smoking?

DR. PETTY: It is very difficult, to quantitate both cough and sputum production. It is our impression that sputum production parallels cough, but we really have no measured data on this. I really don't know.

DR. ANDERSON: In the pre-print, there is a statement that you have recently ascertained that chronic cough and expectoration during life correlate only roughly with the histological lesions, mucous gland hyperplasia found in the postmortem examination. I wonder if you might comment further.

DR. PETTY: I would be happy to. Chronic cough and expectoration, the cardinal symptoms of chronic airway obstruction, have been carefully recorded in our patients from the emphysema registry, many of whom eventually come to autopsy. Chronic cough and expectoration correlate much better with emphysema than with mucous gland hyperplasia.

CHAIRMAN: Dr. Thurlbeck, would you agree?

DR. THURLBECK: It depends on exactly whose numbers you want to believe. I know of two studies. One is Dr. Reid's original paper and one is my own. What you describe is a good measurement, almost a perfect one. In our hands, using essentially the same technique the figure, I believe, is something like 30% of the people with very large mucous glands, did not form spit, but again this is retrospective from clinical charts, and what is this worth really?

DR. PETTY: There is a real problem with the accuracy of histories from clinical charts which may explain some of the differences.

DR. BOUCOT: Dr. Petty, did you attempt to correlate the presence of pigment with occupation?

DR. PETTY: We did not, however, we did exclude patients that might have a great degree of anthracosis from the coal or hardrock mining industry in Colorado.

These patients were excluded from the pigment study. We found no other correlation with occupation.

DR. WINKELSTEIN: I wonder if Dr. Petty and some of the other pathologists might comment on the problem of sampling in autopsies. One of the aspects which comes up repeatedly in epidemiological studies is the difficulty of getting a representative sample of deaths for autopsy. Could this be improved if instead of asking for complete autopsies, studies were designed which would go after particular information.

For example, it might be possible to get one slice of the lung of a random sample of deaths in a community. Families might be willing to give permission for partial autopsy where they wouldn't be willing to give permission for total autopsy.

Could this be perhaps an approach to improving our understanding of the epidemiology of some of these conditions?

DR. PETTY: Fortunately, we are able to obtain autopsies in our hospitals 92% of the time. Thus we can go to the autopsy room and get a lung from almost every case.

We obtained a single lung from each of these cases, losing very few. Regarding localized autopsies, these may be an advantage to some places.