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Air Pollution and Human Health

The quantitative effect, with an estimate of the dollar benefit of pollution abatement, is considered.

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Air pollution is a problem of growing importance; public interest seems to have risen faster than the level of pollution in recent years. Presidential messages and news stories have reflected the opinion of scientists and civic leaders that pollution must be abated. This concern has manifested itself in tightened local ordinances (and, more importantly, in increased enforcement of existing ordinances), in federal legislation, and in extensive research to find ways of controlling the emission of pollutants from automobiles and smokestacks. Pollutants are natural constituents of the air. Even without man and his technology, plants, animals, and natural activity would cause some pollution. For example, animals vent carbon dioxide, volcanic action produces sulfur oxides, and wind movement insures that there will be suspended particulates; there is no possibility of removing all pollution from the air. Instead, the problem is one of balancing the need of polluters to vent residuals against the damage suffered by society as a result of the increased pollution (*1*). To find an optimum level, we must know the marginal costs and marginal benefits associated with abatement. This article is focused on measuring one aspect of the benefit of pollution abatement.

Polluted air affects the health of human beings and of all animals and plants (*2*). It soils and deteriorates property, impairs various production processes (for example, the widespread use of "clean rooms" is an attempt to reduce contamination from the air), raises the rate of automobile and airline accidents (*3*), and generally makes living things less comfortable and less happy. Some of these effects are quite definite and measurable, but most are ill-defined and difficult to measure, even conceptually. Thus, scientists still disagree on the quantitative effect of pollution on animals, plants, and materials. Some estimates of the cost of the soiling and deterioration of property have been made, but the estimates are only a step beyond guesses (*4*). We conjecture that the major benefit of pollution abatement will be found in a general increase in human happiness or improvement in the "quality of life," rather than in one of the specific, more easily measurable categories. Nonetheless, the "hard" costs are real and at least theoretically measurable.

In this article we report an investigation of the effect of air pollution on human health; we characterize the problem of isolating health effects; we derive quantitative estimates of the effect of air pollution on various diseases and point out reasons for viewing some earlier estimates with caution; we discuss the economic costs of ill health; and we estimate the costs of effects attributed to air pollution.

The Effect of Air Pollution on Human Health

In no area of the world is the mean annual level of air pollution high enough to cause continuous acute health problems. Emitted pollutants are diluted in the atmosphere and swept away by winds, except during an inversion; then, for a period that varies from a few hours to a week or more, pollutants are trapped and the dilution process is impeded. When an inversion persists for a week or more, pollution increases substantially, and there is an accompanying increase in the death rate.

Much time has been spent in investigating short-term episodes of air pollution (*5*). We are more concerned with the long-term effects of growing up in, and living in, a polluted atmosphere. Few scientists would be surprised to find that air pollution is associated with respiratory diseases of many sorts, including lung cancer and emphysema. A number of studies have established a qualitative link between air pollution and ill health.

A qualitative link, however, is of little use. To estimate the benefit of pollution abatement, we must know how the incidence of a disease varies with the level of pollution. The number of studies that allow one to infer a quantitative association is much smaller.

Quantifying the relation. Our objective is to determine the amount of morbidity and mortality for specific diseases that can be ascribed to air pollution. The state of one's health depends on factors (both present and past) such as inherited characteristics (that cause a predisposition to certain diseases), personal habits such as smoking and exercise, general physical condition, diet (including the amount of pollutants ingested with food), living conditions, urban and occupational air

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pollution, and water pollution (*6, 7*). Health is a complex matter, and it is exceedingly difficult to sort out the contributions of the various factors. In trying to determine the contribution of any single factor one must be careful neither to include spurious effects nor to conclude on the basis of a single insignificant correlation that there is no association. Laboratory experimentation is of little help in the sorting process (*8*).

The model implicit in the studies we have examined is a simple linear equation wherein the mortality or morbidity rate is a linear function of the measured level of pollution and, possibly, of an additional socioeconomic variable. In only a few cases do the investigators go beyond calculating a simple or partial correlation.

A number of criticisms can be leveled at ths simple model. No account is taken of possibly important factors such as occupational exposure to air pollution and personal habits. These and other factors influencing health must be uncorrelated with the level of pollution, if the estimated effect of pollution is to be an unbiased estimate. In addition, the linear form of the function is not very plausible, except insofar as one considers it a linear approximation over a small range.

Both because of the rather crude nature of the studies and because of the statistical estimation, there is a range of uncertainty concerning the quantitative effect of pollution on human health. This range is reflected in the estimate of the benefit of pollution abatement, discussed below.

Epidemiological studies. Epidemiological data are the kind of health data best adapted to the estimation of air pollution effects. These data are in the form of mortality (or morbidity) rates for a particular group, generally defined geographically (*9*). For example, an analyst may try to account for variations in the mortality rate among the various census tracts in a city. While these vital statistics are tabulated by the government and so are easily available, there are problems with the accuracy of the classification of the cause of death (since few diagnoses are verified by autopsy and not all physicians take equal care in finding the cause of death). Other problems stem from unmeasured variables such as smoking habits, occupations, occupational exposure to air pollution, and genetic health factors. Whenever a variable is unmeasured, the analyst is implicitly assuming either that it is constant across groups or else that it varies randomly with respect to the level of air pollution. Since there are many unmeasured variables, one should not be surprised to discover that some studies fail to find a significant relationship or that others find a spurious one. For the same reason, one should not expect the quantitative effect to be identical across various groups, even when the relationship in each group is statistically significant.

Sample surveys are a means of gathering a more complete set of data. For example, a retrospective analysis might begin with a sample of people who died from a particular disease. Through questionnaires and interviews, the smoking habits and residence patterns of the deceased can be established. The analysis would then consist of an attempt to find the factors implicated in the death of these individuals. Two types of problems arising from such a study are the proper measurement of variables such as exposure to air pollution (there are many pollutants and many patterns of lifetime exposure) and the possible contributions of variables which still are unobserved, such as occupational hazards, socioeconomic characteristics, and personal habits.

Whatever the source of data, the investigators must rest their cases by concluding that the associations which they find are so strong that it is extremely unlikely that omitted variables could have given rise to the observed correlations; they cannot account for all possible variables.

Episodic relationships. Another method of investigating the effects of pollution involves an attempt to relate daily or weekly mortality (or morbidity) rates to indices of air pollution during the interval in question (*10*). The conclusions of these studies are of limited interest, for two reasons. First, someone who is killed by an increase in air pollution is likely to be gravely ill. Air pollution is a rather subtle irritant, and it is unlikely that a healthy 25-year-old will succumb to a rise in pollution levels. Our interest should be focused on the initial cause of illness rather than on the factor that is the immediate determinant of death. Thus, morbidity data are more useful than mortality data. Second (and more important for the morbidity studies), there are many factors that affect the daily morbidity rate or daily rate of employee absences. Absence rates tend to be high on Mondays and Fridays for reasons that have nothing to do with air pollution or illness. One would expect little change in these absence rates if air pollution were reduced. Other factors, such as absence around holidays, give rise to spurious variation; this can be handled by ignoring the periods in question or by gathering enough data so that this spurious variation is averaged away. Some of these factors (such as high absence rates on Fridays and seasonal absence rates) may be correlated with variations in air pollution and no amount of data or of averaging will separate the effects. We have chosen to disregard the results of these episodic studies, with a few exceptions, cited below.

It is difficult to isolate the pollutants that have the most important effects on health on the basis of the studies we survey here. Measurement techniques have been crude, and there has been a tendency to base concentration figures on a single measurement for a large area. A more important problem is the fact that in most of these studies only a single pollutant was reported. Discovering which pollutants are most harmful is an important area, where further exploration is necessary. We have tried, nevertheless, to differentiate among pollutants in the survey that follows (*11*). The problem is complicated, since pollution has increased over time, and since lifetime exposure might bear little relation to currently measured levels. These problems are discussed elsewhere (*12*).

A Review of the Literature

We will proceed with a detailed review of studies made in an attempt to find an association between mortality or morbidity and air pollution indices.

Air pollution and bronchitis. Studies link morbidity and mortality from bronchitis to air pollution in England (*13*), the United States (*14, 15*), Japan (*16*), and other countries (*17*). Mortality rates by country boroughs in England and Wales have been correlated with pollution (as measured by the sulfation rate, total concentration of solids in the air, a deposit index, and the density of suspended particulates) and with socioeconomic variables (such as population density and social class). The smoking habits of the individuals studied have also been investigated. The

conclusion of these studies is that air pollution accounts for a doubling of the bronchitis mortality rate for urban, as compared to rural, areas.

We took data reported by Stocks (*18, 19*) and by Ashley (*20*) and performed a multiple regression analysis, as shown in Table 1. We fit the following equation to the data

$$MR_i = a_0 + a_1P_i + a_2S_i + e_i \quad (1)$$

where MR_i is the mortality rate for a particular disease in country borough i, P_i is a measure of air pollution in that borough, S_i is a measure of socioeconomic status in borough i, and e_i is an error term with a mean of zero. (We also fit other functional forms, as discussed below.) Under general assumptions, the estimated coefficients (a_0, a_1, a_2) will be best linear, unbiased estimates (*21*). Only if we want to perform significance tests must we make an assumption about the distribution of the error term (for example, the assumption that it is distributed normally).

The first regression in Table 1 relates the bronchitis mortality rate for men to a deposit index (see Table 1, footnote †), and the population density in each of 53 country boroughs. Thirty-nine percent of the variation in the mortality rate (across boroughs) is "explained" by the regression. It is estimated that a unit increase in the deposit index (1 gram per 100 square meters per month) leads to an increase of 0.18 percent in the bronchitis mortality rate (with population per acre held constant). An increase of 0.1 person per acre in the population density is estimated to lead to an increase of 0.02 percent in the mortality rate (with air pollution held constant). As indicated in Table 1 by the t statistics (the values in parentheses below the estimated coefficients), the air pollution variable is extremely important, whereas the socioeconomic variable contributes nothing to the explanatory power of the regression.

The first ten regressions in Table 1 are an attempt to explain the bronchitis death rate. Four different data sets are used, along with three measures of pollution and two socioeconomic variables. The coefficient of determination, R^2 (the proportion of the variation in the mortality rate explained by the regression), ranges from .3 to .8. Air pollution is a significant explanatory variable in all cases. In only three cases is the socioeconomic variable significant.

The implication of the first regression is that a 10 percent decrease in the deposit rate (38 g 100 m^{-2} month $^{-1}$) would lead to a 7 percent decrease in

Table 1. Multiple regressions based on data from England. Numbers in parentheses are the t statistic.

Category	R^2*	Index† Air pollution	Index† Socioeconomic
Bronchitis mortality rate			
1. Males, 53 county boroughs‡ (deposit index, persons/acre)	.386	.182 (4.80)	.016 (.22)
2. Females	.332	.182 (4.55)	−.031 (−.42)
3. Males, 28 county boroughs§ (smoke, persons/acre)	.433	1.891 (3.79)	.180 (1.86)
4. Females	.412	1.756 (3.23)	.252 (2.40)
5. Males, 26 areas ‖ (smoke, persons/acre)	.766	.310 (3.77)	.062 (.53)
6. Females	.559	.303 (2.85)	−.038 (−.25)
7. Males, 26 areas (smoke, social class)	.783	.301 (5.86)	.176 (1.44)
8. Females	.601	.213 (3.31)	.248 (1.59)
9. Both sexes, 53 urban areas ¶ (smoke, persons/acre)	.377	.199 (4.07)	.159 (3.02)
10. Both sexes, 53 urban areas (SO_2, persons/acre)	.300	.161 (3.05)	.151 (2.64)
Lung cancer mortality rate			
11. 53 County boroughs (deposit index, persons/acre)	.445	.041 (2.09)	.154 (4.23)
12. 28 County boroughs (smoke, persons/acre)	.576	.864 (4.08)	.161 (3.89)
13. Male, 26 areas (smoke, persons/acre)	.781	.137 (2.86)	.115 (1.70)
14. Male, 26 areas (smoke, social class)	.805	.161 (5.62)	.172 (2.47)
15. 53 Urban areas (smoke, persons/acre)	.344	−.086 (−2.42)	.184 (4.83)
16. 53 Urban areas (SO_2, persons/acre)	.378	−.105 −3.00	.197 (5.23)
Other cancers			
17. Stomach, male, 53 county boroughs (deposit index, persons/acre)	.167	.070 (3.08)	.005 (.12)
18. Stomach, female	.175	.070 (3.08)	−.023 (−.56)
19. Stomach, male, 28 county boroughs (smoke, persons/acre)	.257	.714 (2.57)	.065 (1.21)
20. Stomach, female	.454	.883 (4.13)	.066 (1.60)
21. Intestinal, 53 county boroughs (deposit index, persons/acre)	.041	.018 (1.45)	−.012 (−.52)
22. Intestinal, 28 county boroughs (smoke, persons/acre)	.129	.174 (1.26)	.036 (1.35)
23. Other cancer, male, 26 areas (smoke, persons/acre)	.454	.019 (.59)	.073 (1.60)
24. Other cancer, female, 26 areas (smoke, persons/acre)	.044	.039 (.93)	−.062 (−1.03)
25. Other cancer, male, 26 areas (smoke, social class)	.396	.060 (2.75)	.017 (.33)
26. Other cancer, female, 26 areas (smoke, social class)	.002	.005 (.17)	−.013 (−.19)
Pneumonia mortality rate			
27. Male, 26 areas (smoke, persons/acre)	.477	.118 (1.34)	.121 (.97)
28. Female	.253	.068 (.58)	.137 (.83)
29. Male, 26 areas (smoke, social class)	.475	.158 (2.82)	.126 (.93)
30. Female	.242	.124 (1.65)	.106 (.58)

* The coefficient of determination: a value of .386 indicates a multiple correlation coefficient of .62, and indicates that 39 percent of the variation in the death rate is "explained" by the regression. † The t statistic: for a one-tailed t-test with 23 degrees of freedom, a value of 1.71 indicates significance at the .05 level; for 25 or 50 degrees of freedom, the critical values are 1.71 and 1.68. ‡ Data for 53 county boroughs in England and Wales as reported by Stocks (*18*). Air pollution is measured by a deposit index (in grams per 100 square meters per month) whose observed range is 96 to 731, with a mean of 375. The socioeconomic index is expressed in numbers of persons per acre (multiplied by 10); the range is 69 to 364, and the mean is 163. Death rates are measured as index numbers, with the mean for all boroughs in England and Wales equal to 100. Ranges within this sample are as follows: bronchitis (males), 73 to 259; bronchitis (females), 72 to 268; lung cancer, 70 to 159; stomach cancer (males), 67 to 168; stomach cancer (females), 84 to 161; intestinal cancer, 87 to 123. § Data for 28 county boroughs in England and Wales as reported by Stocks (*18*). Air pollution is measured by a smoke index (suspended matter, in milligrams per 100 cubic meters); the range is 6 to 49. Again, the socioeconomic index is expressed in numbers of persons per acre (× 10); the range is 83 to 342. ‖ Data for 26 areas in northern England and Wales as reported in Stocks (*19*). Air pollution is measured by a smoke index, as for category 3; the range is 15 to 562 mg/1000 m^3 and the mean is 260. One socioeconomic variable is the number of persons per acre (× 10); the range is 1 to 342 and the mean is 102. The other socioeconomic variable is social class; the range is 61 to 295. Death rates are measured as for category 1; within this sample, the range for lung cancer is 23 to 165; for other cancer, 6 to 122 (males) and 88 to 154 (females); for bronchitis, 18 to 259 (males) and 12 to 240 (females); for pneumonia, 61 to 227 (males) and 40 to 245 (females). ¶ Data for 53 areas as reported by Ashley (*20*). Air pollution is measured (i) by a smoke index (as for category 3), with a range of 23 to 261 $\mu g/m^3$ and a mean of 124, or (ii) by an SO_2 index (apparently in the same units), with a range of 33 to 277 and a mean of 124. Death rates are measured as for category 1; within this sample, the range for lung cancer is 70 to 146, and for bronchitis, 64 to 186.

the bronchitis death rate. Another way of illustrating the effect of air pollution on health is to note that, if all the boroughs were to improve the quality of their air to that enjoyed by the borough having the best air of all those in the sample (a standard deposit rate for all boroughs of 96 g 100 m^{-2} month $^{-1}$), the average mortality rate (for this sample) would fall from 129 to 77. Thus, cleaning the air to the level of cleanliness enjoyed by the area with the best air would mean a 40 percent drop in the bronchitis death rate among males. In the fifth regression the pollution index is a smoke index (Table 1, footnote §), and a different set of areas is considered. This is a more successful regression in terms of the percentage of variation explained. As before, the air pollution coefficient is extremely significant, and the implication is that cleaning the air to the level of cleanliness currently enjoyed by the area with the best air (15 mg/100 m^3) would lower the average bronchitis mortality rate from 106 to 30, a drop of 70 percent. Results of the other regression analyses based on bronchitis mortality data have similar implications. Note that the effect is almost the same for males and females. This indicates reliability and suggests that the effect is independent of occupational exposure.

Winkelstein *et al.* (*14*) collected data on 21 areas in and around Buffalo, New York. A cross tabulation of census tracts by income level and pollution level shows that the mortality rate for asthma, bronchitis, and emphysema (in white males 50 to 69 years old) increases by more than 100 percent as pollution rises from level 1 to level 4 (see *22*).

These studies indicate a strong relationship between bronchitis mortality and a number of indices of air pollution. We conclude that bronchitis mortality could be reduced by from 25 to 50 percent depending on the particular location and deposit index, by reducing pollution to the lowest level currently prevailing in these regions. For example, if the air in all of Buffalo were made as clean as the air in those parts of the area that have the best air, a reduction of approximately 50 percent in bronchitis mortality would probably result.

Air pollution and lung cancer. The rate of death from lung cancer has been correlated with several indices of pollution and socioeconomic variables in studies that provided controls for smoking habits and other factors. For English nonsmokers, Stocks and Campbell (*23*, *24*) found a tenfold difference between the death rates for rural and urban areas. Daly (*25*), in comparing death rates in urban and rural areas of England and Wales, found the urban rate twice as high. Evidence for other parts of Europe also shows an association between lung cancer and air pollution (*26*).

Regressions 11 through 16 (Table 1) show our reworking of the data for lung cancer mortality for England and Wales (there is no control for smoking). Regressions 11 through 14 imply that, if the quality of air of all boroughs were improved to that of the borough with the best air, the rate of death from lung cancer would fall by between 11 and 44 percent. Regressions 15 and 16 show a relationship between air pollution and lung cancer which is either insignificant or inverse. The only contrary results come from Ashley's data. In the absence of more complete evidence, we must remain curious about these results. Use of such small samples and inadequate controls is certain to lead to some contrary results, but they are disconcerting when they appear.

In a study of 187,783 white American males (50 to 69 years old), Hammond and Horn (*27*) reported that the age-standardized rate of death due to lung cancer was 34 (per 100,000) in rural areas as compared to 56 in cities of population over 50,000. When standardized with respect both to smoking habits and to age, the rate was 39 in rural areas and 52 in cities of over 50,000.

Haenszel *et al.* (*28*) analyzed 2191 lung cancer deaths among white American males, that had occurred in 46 states, and data for a control group consisting of males who died from other causes. They found the crude rate of death from lung cancer to have been 1.56 times as high in the urban areas of their study as in the rural areas in 1958 and 1.82 times as high in the period 1948–49 (in subjects 35 years and older, with adjustments made for age). When adjustments are made for both age and smoking history, the ratio is 1.43. Also the ratio increased with duration of residence in the urban or rural area, from 1.08 for residence of less than 1 year to 2.00 for lifetime residence. Haenszel and Taeuber (*29*) report similar results for white American females. In a number of additional studies the association between air pollution and lung cancer is examined (*30*).

Buell and Dunn (*31*) review the evidence on lung cancer and air pollution; a summary of their findings is given in Table 2. For smokers, death rates (adjusted for age and smoking) ranged from 25 to 123 percent higher in urban areas than in rural areas. For nonsmokers, all differences exceeded 120 percent. "The etiological roles for lung cancer of urban living and cigarette smoking seem each to be complete," they say, "in that the urban factor is evident when viewing nonsmokers exclusively, and the smoking factor is evident when viewing rural dwellers exclusively." They argue that differences in the quality of diagnosis could not account for the observed differences for urban and rural areas.

Nonrespiratory-tract cancers and air pollution. Our reworking of data from England on rates of death from nonrespiratory-tract cancer is presented in Table 1 (regressions 17 through 26). In the regressions, stomach cancer is significantly related to a deposit index and a smoke index. The effects are nearly identical for males and females. Intestinal cancer appears to be only marginally related to indices of either deposit or smoke. For 26 areas in northern England and Wales, there appears to be little relationship between nonrespiratory-tract cancers and a smoke index. The single exception in the four regressions occurs for males when the socioeconomic variable is social class; here the smoke index explains a significant amount of the variation in the cancer mortality rate. (Apparently population density and smoke index are so highly related in these 26 areas that neither has significant power to explain such variation.)

Winkelstein and Kantor (*32*) investigated rates of mortality from stomach cancer in Buffalo, New York, and the immediate environs. Their measure of pollution is an index of suspended particulates averaged over a 2-year period. They found the rate of mortality due to stomach cancer to be more than twice as great in areas of high pollution as in areas of low pollution (*33*).

Hagstrom *et al.* (*34*) tabulated rates of death from cancer among middle class residents of Nashville, Tennessee, between 1949 and 1960, using four measures of air pollution. They found the cancer mortality rate to be 25 percent higher in polluted areas than in

areas of relatively clean air (*35*). They also found significant mortality-rate increases associated with individual categories of cancer, such as stomach cancer, cancer of the esophagus, and cancer of the bladder. The individual mortality rates are more closely related to air pollution after the data are broken down by sex and race.

Levin *et al.* (*36*) report, for all types of cancer, these relationships: The age-adjusted cancer-incidence rates for urban males was 24 percent higher than that for rural males in New York State (exclusive of New York City) (1949–51), 36 percent higher in Connecticut (1947–51), and 40 percent higher in Iowa (1950); the incidence rate for urban females was 14 percent higher than that for rural females in New York State, 28 percent higher in Connecticut, and 34 percent higher in Iowa. For both males and females, the incidence rate for each of 16 categories of cancer was higher in urban than in rural areas.

Cardiovascular disease and air pollution. Enterline *et al.* (*37*) found that mortality from heart disease is higher in central-city counties than in suburban counties, and, in turn, higher in suburban counties than in nonmetropolitan counties. Zeidberg *et al.* (*38*) found that both morbidity and mortality rates for heart disease are associated with air pollution levels in Nashville. The morbidity rate was about twice as high in areas of polluted air as in areas of clean air. The mortality rate was less closely associated; it was 10 to 20 percent higher in areas of polluted air than in areas of clean air (*39*).

Friedman (*40*) correlated the rate of mortality from coronary heart disease in white males aged 45 to 64 with the proportion of this group living in urban areas. The simple correlation for 33 states is .79. When cigarette consumption is held constant, the partial correlation is .67.

On the basis of these studies we conclude that a substantial abatement of air pollution would lead to 10 to 15 percent reduction in the mortality and morbidity rates for heart disease. We caution the reader that the evidence relating cardiovascular disease to air pollution is less comprehensive than that linking bronchitis and lung cancer to air pollution.

Total respiratory disease (41). Daly (*25*) found significant correlations between air pollution and death rates for all respiratory diseases (and for non-respiratory diseases as well) in England. Douglas and Waller (*42*) found significant relationships between air pollution and respiratory disease in 3866 British school children. Fairbairn and Reid (*43*) found significant correlations between air pollution and morbidity rates (for bronchitis, pneumonia, pulmonary tuberculosis, and lung cancer) in England. Regressions 27 through 30 in Table 1 show pneumonia mortality to be related only marginally to a smoke index.

Table 2. A summary of lung cancer mortality studies. Number of deaths from lung cancer per 100,000 population [from Buell and Dunn (*31*)].

Standardized for age and smoking			Nonsmokers			Study
Urban	Rural	Urban/ rural	Urban	Rural	Urban/ rural	
101	80	1.26	36	11	3.27	Buell, Dunn, and Breslow* (*67*)
52	39	1.33	15	0	∞	Hammond and Horn (*68*)†
189	85	2.23	50	22	2.27	Stocks (*69*)‡
			38	10	3.80	Dean (*70*)§
149	69	2.15	23	29	.79	Golledge and Wicken (*71*) ||
100	50	2.00	16	5	3.20	Haenszel *et al.* (*72*) ¶

* California men; death rates by counties. † American men. ‡ England and Wales. § Northern Ireland. || England; no adjustment for smoking. ¶ American men.

Zeidberg *et al.* (*44*) questioned 9313 Nashville residents about recent illnesses. Among males aged 55 and older from white middle-class families, the numbers of illnesses per respondent during the past year were 1.92, 1.15, and 1.26 for areas of high, moderate, and low pollution, respectively. There are a number of other comparisons, based on other measures of air pollution and on data for females and nonwhites (some of these are given in 45). However, we should add a word of caution: although the sample size in this study was large and controls for many socioeconomic variables were included, many important factors were ignored—for example, smoking habits and length of residence. Nonetheless, the finding is extremely strong and seems unlikely to be an artifact of unmeasured variables.

Hammond (*46*) studied over 50,000 men to find the relationships between emphysema, age, occupational exposure to pollution, urban exposure, and smoking. His results indicated that the effect of air pollution is significant and that heavy smokers have a much higher morbidity rate in cities than in rural areas; the effect becomes more marked as age increases.

Ishikawa *et al.* (*47*) estimated the incidence of emphysema in Winnipeg (Canada) and St. Louis. They examined the lungs of 300 corpses in each city (the samples were comparable). Findings for each age group (over 25 years old) indicated that the incidence and severity of emphysema is higher in St. Louis, the city with the more polluted air. (In the 45-year-old group 5 percent of those in Winnipeg and 46 percent of those in St. Louis showed evidence of emphysema.)

A number of studies have been made in England on homogeneous occupational groups, such as postmen. The results are relatively pure in that all members of the sample have comparable incomes, working conditions, and social status. Holland and Reid (*48*) found that the rates of occurrence of severe respiratory symptoms were 25 to 50 percent higher for London postmen than for small-town postmen (sample size, 770). Reid (*49*) found that, in the postmen of his study, absences due to bronchitis rose from an index number of 100 for the area of least air pollution, to 120 for an area of moderate pollution, to 250 and 283 for the areas of highest pollution. Corresponding figures for absences due to other respiratory illness were 100, 100, 150, and 151, respectively, and for absences due to infectious and parasitic diseases, 100, 115, 130, and 140. Cornwall and Raffle (*50*) made a similar study of bus drivers in London. They found that 20 to 35 percent of absences due to sickness of any kind could be ascribed to air pollution (they used a fog index as a measure of pollution). Fairbairn and Reid (*43*) tabulated absences due to sickness for postmen, for males working indoors, and for females working indoors. They found that the age-standardized morbidity rate for bronchitis and pneumonia in the postmen of their study rose from 40 man-years, per 1000 man-years, for the area of lowest air pollution (of the four areas studied) to 122 for the area of highest air pollution. Corresponding figures for morbidity from colds were 75 and 171

man-years, and for morbidity from influenza, 131 and 184 man-years. For males working indoors, the low and high morbidity rates were as follows: bronchitis and pneumonia, 32 and 39; colds, 53 and 64; influenza, 88 and 102.

Dohan (*51*) studied absences (of more than 7 days) of female employees in eight Radio Corporation of America plants. He found a correlation of .96 between atmospheric concentrations of SO_3 and absences due to respiratory disease in the five cities for which complete data were available. During Asian flu epidemics there was a 200 percent increase in illness in cities with polluted air and only a 20 percent increase in those with relatively unpolluted air.

Infant mortality and total mortality rates. Sprague and Hagstrom (*52*) compared air-pollution data for Nashville with fetal and infant mortality rates for Nashville as given in census tracts (for 1955 through 1960). Controls for socioeconomic factors were not included. For infant death rates (ages 28 days to 11 months), the highest correlation was with atmospheric concentrations of SO_3 (in milligrams per 100 square centimeters per day) and was .70. For the neonatal death rates (ages 1 day to 27 days), the highest correlation was with dustfall and was .49. For infants dying during their first day whose death certificate includes mention of immaturity, the highest correlation was with dustfall and was .45. The correlation of the fetal death rate with dustfall was .58.

In a study just being completed (*53*), we have collected data for 114 Standard Metropolitan Statistical Areas in the United States and have attempted to relate total death rates and infant mortality rates to air pollution and other factors. Socioeconomic data, death rates, and air-pollution data were taken from U.S. government publications (*54*). Regression 1 (Table 3) shows how the total death rate in 1960 varies with air pollution levels and with socioeconomic factors. As the (biweekly) minimum level of suspended particulates increases, the death rate rises significantly. Moreover, the death rate increases with (i) the density of population of the area, (ii) the proportion of nonwhites, (iii) the proportion of people over age 65, and (iv) the proportion of poor families. Eighty percent of the variation in the death rate across these 114 statistical areas is explained by the regression.

Regression 3 shows how the 1960 infant death rate (age, less than 1 year) varies. A smaller proportion (55 percent) of the variation in the death rate is explained by the regression, although the minimum air-pollution level, the percentage of nonwhites, and the proportion of poor families continue to be significant explanatory variables. Regression 5 is an attempt to explain variation in the neonatal death rate. The results are quite similar to those of regression 3. The fetal death rate is examined in regression 7. Here the minimum air-pollution level, population density, the percentage of nonwhites, and the percentage of poor families are all significant explanatory variables.

Regressions 2, 4, 6, and 8 are an attempt to relate these death rates to the atmospheric concentrations of sulfates for the 114 statistical areas of the study. Regression 2 shows that the total death rate is significantly related to the minimum level of sulfate pollution, to population density, and to the percentage of people over age 65; 81 percent of the variation is explained. Regressions 4, 6, and 8 show that the minimum atmospheric concentration of sulfates is a significant explanatory variable in three categories of infant death rates.

One might put these results in perspective by noting estimates on how small decreases in the air-pollution level affect the various death rates. A 10 percent decrease in the minimum concentration of measured particulates would decrease the total death rate by 0.5 percent, the infant death rate by 0.7 percent, the neonatal death rate by 0.6 percent, and the fetal death rate by 0.9 percent. Note that a 10 percent decrease in the percentage of poor families would decrease the total death rate by 0.2 percent and the fetal death rate by 2 percent. A 10 percent decrease in the minimum concentration of sulfates would decrease the total death rate by 0.4 percent, the infant mortality rate by 0.3 percent, the neonatal death rate by 0.4 percent, and the fetal death rate by 0.5 percent.

Each of the relations in Tables 1 and 3 was estimated in alternative ways, including transformation into logarithms, a general quadratic, and a "piecewise" linear form as documented elsewhere (*12*). The implications about the roles of air pollution and of the socioeconomic variables were unchanged by use of the different functional forms. Another result to be stressed is that, in Table 1, comparable

Table 3. Regressions relating infant and total mortality rates for 114 Standard Metropolitan Statistical Areas in the United States to air pollution and other factors. Values in parentheses are the *t* statistic.* For means and standard deviations (S.D.) of the variables, see †.

Category	R^2‡	Air pollution (minimum) concentrations)	Socioeconomic			
			P/m²§	Nonwhite (%)	Over 65 (%)	Poor (%)
		Total death rate				
1. Particulates	.804	0.102	0.001	0.032	0.682	0.013
		(2.83)	(2.58)	(3.41)	(18.37)	(0.93)
2. Sulfates	.813	0.085	0.001	0.033	0.652	0.006
		(3.73)	(1.86)	(3.56)	(17.60)	(0.49)
		Death rate for infants of less than 1 year				
3. Particulates	.545	0.393		0.190		0.150
		(3.07)		(6.63)		(3.28)
4. Sulfates	.522	0.150		0.200		0.123
		(1.91)		(6.83)		(2.70)
		Death rate for infants less than 28 days old				
5. Particulates	.260	0.273		0.089		0.063
		(2.48)		(3.61)		(1.60)
6. Sulfates	.263	0.170		0.097		0.047
		(2.57)		(3.96)		(1.23)
		Fetal death rate				
7. Particulates	.434	0.274	0.004	0.171		0.106
		(2.02)	(2.01)	(5.70)		(2.11)
8. Sulfates	.434	0.171	0.004	0.181		0.085
		(1.95)	(1.82)	(5.87)		(1.71)

* The *t* statistic: for a one-tailed *t*-test, a value of 1.65 indicates significance at the .05 level. † Total death rate per 10,000: mean, 91.5; S.D., 15.2. Infant death rate (age, < 1 year) per 10,000 live births: mean, 255.1; S.D., 36.1. Infant death rate (age, < 28 days) per 10,000 live births: mean, 188.0; S.D., 24.4. Fetal death rate per 10,000 live births: mean, 153.9; S.D., 34.4. Suspended particulates ($\mu g/m^3$), minimum reading for a biweekly period: mean, 45.2; S.D., 18.7. Total sulfates ($\mu g/m^3$) (× 10), minimum reading for a biweekly period: mean, 46.9; S.D., 30.6. Persons per square mile: mean, 763.4; S.D., 1387.9. Percentage of nonwhites in population (× 10): mean, 125.2; S.D., 102.8. Percentage of population over 65 (× 10): mean, 84.2; S.D., 21.2. Percentage of families with incomes under $3000 (× 10): mean, 181.6; S.D., 65.7. ‡ The coefficient of determination: a value of .804 indicates a multiple correlation coefficient of .90, and indicates that 80 percent of the variation in the death rate is "explained" by the regression. § Persons per square mile.

regressions for males and females show almost precisely the same effects for air pollution. This suggests that occupational exposure does not affect these results; the result lends credence to the estimates. A result that we document elsewhere (*53*) is that it is the minimum level of air pollution that is important, not the occasional peaks. People dealing with this problem should worry about abating air pollution at all times, instead of confining their concern to increased pollution during inversions.

Some Caveats

In preceding sections we have described a number of studies which quantify the relationship between air pollution and both morbidity and mortality. Is the evidence conclusive? Is it possible for a reasonable man still to object that there is no evidence of a substantial quantitative association? We believe that there is conclusive evidence of such association (*55*).

In the studies discussed, a number of countries are considered, and differences in morbidity and mortality rates among different geographical areas, among people within an occupational group, and among children are examined. Various methods are used, ranging from individual medical examinations and interviews to questionnaires and tabulations of existing data. While individual studies may be attacked on the grounds that none manages to provide controls for all causes of ill health, the number of studies and the variety of approaches are persuasive. It is difficult to imagine how factors such as general habits, inherited characteristics, and lifetime exercise patterns could be taken into account.

To discredit the results, a critic would have to argue that the relationships found by the investigators are spurious because the level of air pollution is correlated with a third factor, which is the "real" cause of ill health. For example, many studies do not take into account smoking habits, occupational exposure, and the general pace of life. Perhaps city dwellers smoke more, get less exercise, tend to be more overweight, and generally live a more strained, tense life than rural dwellers. If so, morbidity and mortality rates would be higher for city dwellers, yet air pollution would be irrelevant. This explanation cannot account for the relationships found.

Apparently there is little systematic relationship between relevant "third" factors and the level of air pollution. An English study (*19*) in which smoking habits are examined reveals little evidence of differences by residence. There is evidence in the United States that smoking is more prevalent among lower socioeconomic groups (*56*) but income or other socioeconomic variables would account for this effect and still leave the pollution coefficient unbiased. More importantly, the correlations between air pollution and mortality are better when one is comparing areas within a city (where more factors are held constant) than when one is comparing rural and urban areas (*57*). It is especially hard to believe that the apparent relation between air pollution and ill health is spurious when significant effects are found in studies comparing individuals within strictly defined occupational groups, such as postmen or bus drivers (where incomes and working conditions are comparable and unmeasured habits are likely to be similar).

When there are uncontrolled factors, some studies may show inconclusive or even negative results; only by collecting samples large enough to "average away" spurious effects can dependable results be guaranteed. In the main, each of the studies cited above was based on a substantial sample. It is the body of studies as a whole that we find persuasive.

An examination of contrary results. Uncontrolled factors, together with small samples, are certain to lead to some results contrary to the weight of evidence and to our expectations. For example, in some studies (*58*) no attempt is made to control even for income or social status. From the evidence of studies which did provide such controls, we know that failure to control for income leads to biased results, and so we place little credence in either the positive or negative findings of studies lacking these controls.

Sampling error can be extremely important. For example, Zeidberg *et al.* (*59*) find mixed results in cross-tabulating respiratory disease mortality with level of air pollution and with income class. In general the relationships are in the expected direction, but they are often insignificant. Insignificant results might occur often, if the samples are small, even if air pollution is extremely significant, since sampling errors dominate the explanatory variables.

Another study in which sampling error is important is reported by Ferris and Whittenberger (*7*). They compared individuals in Berlin, New Hampshire, with residents of Chilliwack (British Columbia), Canada, and—not surprisingly in view of the small samples—failed to find significant differences in the occurrence of respiratory disease. Prindle *et al.* (*60*) compared two Pennsylvania towns in the same fashion. These two studies are admirable in that individuals were subjected to careful medical examinations. However, only a few hundred individuals were studied, and this means that sampling errors tend to obscure the effects of air pollution. Moreover, there were no controls for other factors, such as smoking. Also, one must be careful to control for a host of other variables if the sample is small. For example, the ethnic origins of the population and their general habits and occupations are known to affect mortality rate. It is exceedingly difficult to control for these factors; use of carefully constructed large samples seems the best answer. Finally, air pollution is measured currently, and it is generally assumed that relative levels have been constant over time and that people have lived at their present addresses for a long period. It is hardly surprising that statistical significance is not always obtained when such assumptions are necessary.

Since investigators are more reluctant to publish negative results than positive ones, and since it is more difficult to get negative results published, it is probable that we are unaware of other studies that fail to find a strong association between air pollution and ill health. We are somewhat reluctant to come to strong conclusions without knowledge of such negative results. However, there seems to be no reasonable alternative to evaluating the evidence at hand and allowing for uncertainty. Thus, we conclude that an objective observer would have to agree that there is an important association between air pollution and various morbidity and mortality rates.

The Economic Costs of Disease

Having found a quantitative association between air pollution and both morbidity and mortality, the next question is that of translating the increased sickness and death into dollar units. The relevant question is, How much

is society willing to spend to improve health (to lower the incidence of disease)? In other words, how much is it worth to society to relieve painful symptoms, increase the level of comfort of sufferers, prevent disability, and prolong life? It has become common practice to estimate what society is willing to pay by totaling the amount that is spent on medical care and the value of earnings "forgone" as a result of the disability or death (*61*). This cost seems a vast underestimate for the United States in the late 1960's. Society seems willing to spend substantial sums to prolong life or relieve pain. For example, someone with kidney failure can be kept alive by renal dialysis at a cost of $15,000 to $25,000 per year; this sum is substantially in excess of forgone earnings, but today many kidney patients receive this treatment. Another example is leukemia in children; enormous sums are spent to prolong life for a few months, with no economic benefit to society. If ways could be found to keep patients with chronic bronchitis alive and active longer, it seems likely that people would be willing to spend sums substantially greater than the foregone earnings of those helped. So far as preventing disease is concerned, society is willing to spend considerable sums for public health programs such as chest x-rays, inoculation, fluoridation, pure water, and garbage disposal and for private health care programs such as annual physical checkups.

While we believe that the value of earnings forgone as a result of morbidity and mortality provides a gross underestimate of the amount society is willing to pay to lessen pain and premature death caused by disease, we have no other way of deriving numerical estimates of the dollar value of air-pollution abatement. Thus, we proceed with a conventional benefit calculation, using these forgone earnings despite our reservations.

Direct and indirect costs. Our figures for the cost of disease are based on *Estimating the Cost of Illness,* by Dorothy P. Rice (*61*). Unfortunately, Rice calculated disease costs in quite aggregate terms, and so the category "diseases of the respiratory system" must be broken down. It seem reasonable to assume that both direct and indirect costs would be proportional to the period of hospitalization (total patient-days in hospitals) by disease category (*62*).

Rice defines a category of direct disease costs as including expenditures for hospital and nursing home care and for services of physicians, dentists, and members of other health professions. "Other direct costs" (which would add about 50 percent to those just enumerated) consist of a variety of personal and nonpersonal expenditures (such as drugs, eyeglasses, and appliances), school health services, industrial in-plant health services, medical activities in federal units other than hospitals, medical research, construction of medical facilities, government public health activities, administrative expenditures of voluntary health agencies, and the net cost of insurance. Since Rice does not allocate "other direct costs" among diseases, we omit it from our cost estimates. However, we conjecture that respiratory diseases represent a substantial portion of this category. Thus, our direct cost estimate is likely to be a substantial underestimate of "true" direct costs (probably more than 50 percent too low).

Estimating indirect cost is an attempt to measure the losses to the nation's economy caused by illness, disability, and premature death. We would argue that such a calculation gives a lower bound for the amount people would be willing to pay to lower the morbidity and mortality rates. These costs are calculated in terms of the earnings forgone by those who are sick, disabled, or prematurely dead (*63*).

The Health Cost of Air Pollution

The studies cited earlier in this article show a close association between air pollution and ill health. The evidence is extremely good for some diseases (such as bronchitis and lung cancer) and only suggestive for others (such as cardiovascular disease and nonrespiratory-tract cancers). Not all factors have been taken into account, but we argue that an unbiased observer would have to concede the association. More effort can and should be spent on refining the estimates. However, the point of this exercise is to estimate the health cost of air pollution. We believe that the evidence is sufficiently complete to allow us to infer, roughly, the quantitative associations. We do so with caution, and proceed to translate the effects into dollars. We have attempted to choose our point estimates from the conservative end of the range.

We interpret the studies cited as indicating that mortality from bronchitis would be reduced by about 50 percent if air pollution were lowered to levels currently prevailing in urban areas with relatively clean air. We therefore make the assumption that there would be a 25 to 50 percent reduction in morbidity and mortality due to bronchitis if air pollution in the major urban areas were abated by about 50 percent. Since the cost of bronchitis (in terms of forgone income and current medical expenditures) is $930 million per year, we conclude that from $250 million to $500 million per year would be saved by a 50 percent abatement of air pollution in the major urban areas.

Approximately 25 percent of mortality from lung cancer can be saved by a 50 percent reduction in air pollution, according to the studies cited above. This amounts to an annual cost of about $33 million.

The studies document a strong relationship between all respiratory disease and air pollution. It seems likely that 25 percent of all morbidity and mortality due to respiratory disease could be saved by a 50 percent abatement in air pollution levels. Since the annual cost of respiratory disease is $4887 million, the amount saved by a 50 percent reduction in air pollution in major urban areas would be $1222 million.

There is evidence that over 20 percent of cardiovascular morbidity and about 20 percent of cardiovascular mortality could be saved if air pollution were reduced by 50 percent. We have chosen to put this saving at only 10 percent—that is, $468 million per year.

Finally, there is a good deal of evidence connecting all mortality from cancer with air pollution. It is difficult to arrive at a single figure, but we have estimated that 15 percent of the cost of cancer would be saved by a 50 percent reduction in air pollution—a total of $390 million per year.

Not all of these cost estimates are equally certain. The connection between bronchitis or lung cancer and air pollution is much better documented than the connection between all cancers or all cardiovascular disease and air pollution. The reader may aggregate the costs as he chooses. We estimate the total annual cost that would be saved by a 50 percent reduction in air-pollution levels in major urban areas, in terms of decreased morbidity and mortality, to be $2080 million. A more relevant indication of the cost would be the estimate that 4.5 percent

of all economic costs associated with morbidity and mortality would be saved by a 50 percent reduction in air pollution in major urban areas (*64*). This percentage estimate is a robust figure; it is not sensitive to the exact figures chosen for calculating the economic cost of ill health.

A final point is that these dollar figures are surely underestimates of the relevant costs. The relevant measure is what people would be willing to pay to reduce morbidity and mortality (for example, to reduce lung cancer by 25 percent). It seems evident that the value used for forgone earnings is a gross underestimate of the actual amount. An additional argument is that many health effects have not been considered in arriving at these costs. For example, relatively low levels of carbon monoxide can affect the central nervous system sufficiently to reduce work efficiency and increase the accident rate (*65*). Psychological and esthetic effects are likely to be important, and additional costs associated with the effect of air pollution on vegetation, cleanliness, and the deterioration of materials have not been included in these estimates (*66*).

References and Notes

1. For a general discussion of inherent problems in handling residuals, see R. U. Ayres and A. V. Kneese, *Amer. Econ. Rev.* **59**, 282 (1969).
2. For summaries of studies relating air pollution to health, see J. R. Goldsmith, in *Air Pollution*, vol. 1: *Air Pollution and Its Effects*, A. Stern, Ed. (Academic Press, New York, 1968), p. 547; E. C. Hammond, paper presented at the 60th annual meeting of the Air Pollution Control Association, 1967; H. Heimann, *Arch. Environ. Health* **14**, 488 (1967). For more general reviews of the literature, see A. G. Cooper, "Sulfur Oxides and other Sulfur Compounds," *U.S. Public Health Serv. Publ. No. 1093* (1965); ———, "Carbon Monoxide," *U.S. Public Health Serv. Publ. No. 1503* (1966); "The Oxides of Nitrogen in Air Pollution," *Calif. Dep. Public Health Publ.* (1966); "Air Quality Criteria for Sulfur Oxides," *U.S. Public Health Serv. Publ. No. 1619* (1967); *Effects of Chronic Exposure to Low Levels of Carbon Monoxide on Human Health, Behavior, and Performance* (National Academy of Sciences and National Academy of Engineering, Washington, D.C., 1969).
3. *Public Health (Johannesburg)* **63**, 30 (1963); D. M. Johnson, *Good Housekeeping* **1961**, 49 (June 1961).
4. See A. V. Kneese, in *Social Sciences and the Environment; Conference on the Present and Potential Contribution of the Social Sciences to Research and Policy Formulation in the Quality of the Physical Environment*, M. E. Garnsey and J. R. Hibbs, Eds. (Univ. of Colorado Press, Boulder, 1967), p. 165; R. G. Ridker, *Economic Costs of Air Pollution* (Praeger, New York, 1967); "Air Quality Criteria for Sulfur Oxides, *U.S. Public Health Serv. Publ. No. 1619* (1967), pp. 54–57.
5. L. Greenburg, M. B. Jacobs, B. M. Drolette, F. Field, M. M. Braverman, *Public Health Rep.* **77**, 7 (1962); M. McCarroll and W. Bradley, *Amer. J. Public Health Nat. Health* **56**, 1933 (1966); J. Firket, *Trans. Faraday Soc.* **32**, 1192 (1936); H. H. Schrenk, H. Heimann, G. D. Clayton, W. M. Gafafer, H. Wexler, "Air Pollution in Donora, Pennsylvania," *Public Health Bull. No. 306* (1949).
6. See J. R. Goldsmith, *Med. Thoracalis* **22**, 1 (1965).
7. B. G. Ferris, Jr., and J. L. Whittenberger, *N. Engl. J. Med.* **275**, 1413 (1966).
8. For a summary of laboratory experiments see "Air Quality Criteria for Sulfur Oxides," *U.S. Public Health Serv. Publ. No. 1619* (1967), pp. 79–93.
9. Chronic effects, where the incidence of the disease is small, can be studied only for large samples (millions of man-years of exposure); see J. R. Goldsmith, *Arch. Environ. Health* **18**, 516 (1969); J. Rumford, *Amer. J. Public Health* **51**, 165 (1961). Morbidity data would be more useful than mortality data, since death may result from a cause having no direct relationship to the original pollution-induced disease.
10. For example, M. McCarroll and W. Bradley [*Amer. J. Public Health Nat. Health* **56**, 1933 (1966)] correlate the daily mortality rate in New York City with daily pollution indices. See also J. R. McCarroll, E. J. Cassell, W. A. B. Ingram, D. Wolter, "Distribution of families in the Cornell air pollution study" and "Health profiles vs. environmental pollutants," papers presented at the 92nd annual meeting of the American Public Health Association, New York, 1964; ———, *Arch. Environ. Health* **10**, 357 (1965); W. Ingram, J. R. McCarroll, E. J. Cassell, D. Wolter, *ibid.*, p. 364; E. J. Cassell, J. R. McCarroll, W. Ingram, D. Wolter, *ibid.*, p. 367. Other workers have attempted to explain daily variations in hospital admissions [see L. Greenburg, F. Field, J. I. Reed, C. L. Erhardt, *J. Amer. Med. Ass.* **182**, 161 (1962); W. W. Holland, C. C. Spicer, J. M. G. Wilson, *Lancet* **1961-II**, 338 (1961); G. F. Abercrombie, *ibid.* **1953-I**, 234 (1953); A. E. Martin, *Mon. Bull. Min. Publ. Health Lab. Serv. Directed Med. Res. Counc.* **20**, 42 (1961); R. Lewis, M. M. Gilkeson, Jr., R. O. McCaldin, *Public Health Rep.* **77**, 947 (1962); T. D. Sterling, S. V. Pollack, D. A. Schumsky, I. Degroot, *Arch. Environ. Health* **13**, 158 (1966); T. D. Sterling, S. V. Pollack, J. Weinkam, *ibid.* **18**, 462 (1969)]; absence rates (see J. Ipsen, F. E. Ingenito, M. Deane, *ibid.*, p. 462); symptoms in school children [see B. Paccagnella, R. Pavanello, R. Pesarin, *ibid.*, p. 495; T. Toyama, *ibid.* **8**, 153 (1964)]; the incidence of asthma attacks [see L. D. Zeidberg, R. A. Prindle, E. Landau, *Amer. Rev. Resp. Dis.* **84**, 489 (1961); C. E. Schoettlin and E. Landau, *Public Health Rep.* **76**, 545 (1961); R. Lewis, *J. La. State Med. Soc.* **115**, 300 (1963)]; and other morbidity [see J. T. Boyd, *Brit. J. Prev. Soc. Med.* **14**, 123 (1960); R. G. Loudon and J. F. Kilpatrick, *Arch. Environ. Health* **18**, 641 (1969)].
11. The most complete investigation of various pollutants was that of the Nashville studies. See L. D. Zeidberg, R. A. Prindle, E. Landau, *Amer. Rev. Resp. Dis.* **84**, 489 (1961); L. D. Zeidberg and R. A. Prindle, *Amer. J. Public Health* **53**, 185 (1963); L. D. Zeidberg, R. A. Prindle, E. Landau, *ibid.* **54**, 85 (1964); L. D. Zeidberg, R. J. M. Horton, E. Landau, *Arch. Environ. Health* **15**, 214 (1967); ———, *ibid.*, p. 225; R. M. Hagstrom, H. A. Sprague, E. Landau, *ibid.*, p. 237; H. A. Sprague and R. Hagstrom, *ibid.* **18**, 503 (1969). It is conceptually possible to differentiate among pollutants, since, for example, the correlation between mean level of suspended particulates and mean level of sulfates for 114 U.S. Standard Metropolitan Statistical Areas is only .20.
12. L. B. Lave, "Air pollution damage" in *Research on Environmental Quality*, A. Kneese, Ed. (Johns Hopkins Press, Baltimore, in press).
13. D. J. B. Ashley, *Brit. J. Cancer* **21**, 243 (1967); C. Daly, *Brit. J. Prev. Soc. Med.* **13**, 14 (1959); J. Pemberton and C. Goldberg, *Brit. Med. J.* **2**, 567 (1954); P. Stocks, *ibid.* **1**, 74 (1959); R. E. Waller and P. J. Lawther, *ibid.* **2**, 1356 (1955); ———, *ibid.* **4**, 1473 (1957); P. J. Lawther, *Proc. Roy. Soc. Med.* **51**, 262 (1958); ———, *Nat. Acad. Sci. Nat. Res. Counc. Publ. No. 652* (1959), pp. 88–96; ———, *Instrum. Pract.* **11**, 611 (1957); J. Pemberton, *J. Hyg. Epidemiol. Microbiol. Immunol. (Prague)* **5**, 189 (1961); J. L. Burn and J. Pemberton, *Int. J. Air Water Pollut.* **7**, 5 (1963); E. Gorham, *Lancet* **1958-I**, 691 (1958); P. Stocks, *Brit. J. Cancer* **14**, 397 (1960). These studies are updated and summarized in S. F. Buck and D. A. Brown, *Tobacco Res. Counc. Res. Paper No. 7* (1964).
14. W. Winkelstein, Jr., S. Kantor, E. W. Davis, C. S. Maneri, W. E. Mosher, *Arch. Environ. Health* **14**, 162 (1967).
15. International Joint Commission U.S. and Canada, "Report on the pollution of the atmosphere in the Detroit River Area" (Washington and Ottawa, 1960).
16. T. Toyama, *Arch. Environ. Health* **8**, 153 (1964).
17. F. L. Petrilli, G. Agnese, S. Kanitz, *ibid.* **12**, 733 (1966); A. Bell, in *Air Pollution by Metallurgical Industries*, A. Bell and J. L. Sullivan, Eds. (Department of Public Health, Sydney, Australia, 1962), pp. 2:1–2:144.
18. P. Stocks, *Brit. Med. J.* **1**, 74 (1959).
19. ———, *Brit. J. Cancer* **14**, 397 (1960).
20. D. J. B. Ashley, *ibid.* **21**, 243 (1967).
21. That the least-squares method provides the best linear unbiased estimates is the conclusion of the Gauss-Markov theorem, for which $E(ee') = \sigma^2 I$ and $E(e) = 0$ are the basic assumptions. These assumptions are that the basic model must be linear and that the distribution of the errors must have an expected value of zero, have finite variance, have a constant distribution over the various observations, and be independent. In addition, no explanatory variables may be omitted which are correlated with included variables. It is also convenient to assume that the explanatory variables are measured without error, although the framework can easily be adusted to handle errors. In order to perform significance tests, one must make an assumption about the distribution of the error term. For all the relations we estimated, we plotted the residuals and discovered that all distributions were unimodel, symmetric, and basically consistent with the normal distribution. Thus, in the discussion that follows, we have assumed that the error term is distributed normally.
22. For example, for economic level 1 (defined below), the death rates (per 100,000) for pollution levels 2 to 4 (defined below) are 126, 271, and 392. For economic level 2, the death rates for air pollution levels 1 to 4 are 136, 154, 172, and 199. For economic level 4, the death rates for pollution levels 1 to 3 are 70, 80, and 177. The five economic levels, based on median family income in a census tract, are as follows: \$3005–\$5007; \$5175–\$6004; \$6013–\$6614; \$6618–\$7347; and \$7431–\$11,792. The four air pollution levels (in micrograms) of suspended particulates (per cubic meter per 24 hours) are as follows: less than 80, 80–100, 100–135, and more than 135.
23. P. Stocks and J. M. Campbell, *Brit. Med. J.* **2**, 923 (1955).
24. In most of the early studies, pollution measures were not available, and so urban mortality rates were contrasted with rural rates. In these studies a substantial "urban factor" was found, which, unfortunately, was a compound of air pollution and many other factors. In the later studies the portion ascribable to air pollution is separated out.
25. C. Daly, *Brit. J. Prev. Soc. Med.* **13**, 14 (1959).
26. Buck and Brown [*Tobacco Res. Counc. Res. Paper No. 7* (1964)], in examining data from England, control for population per acre, for social class, and for smoking habits. They find no relationship between smoking and lung cancer, and a relationship between SO_2 and lung cancer that is not consistent. Stocks uses three sets of data to isolate the effect of air pollution on lung cancer. Contrasting data for eight northern European cities, he finds a correlation between lung cancer and air pollution of .60, and correlations between lung cancer and smoking that range between .27 and .36. Contrasting data for 19 countries, he finds that an index of solid fuel consumption is a much stronger variable than cigarette consumption per capita. Finally, with data from northern England, he finds confirmation of an association between lung cancer and air pollution. See P. Stocks, *Brit. J. Prev. Soc. Med.* **21**, 181 (1966).
27. E. C. Hammond and D. Horn, *J. Amer. Med. Ass.* **166**, 1294 (1958).
28. W. Haenszel, D. B. Loveland, M. G. Sirken, *J. Nat. Cancer Inst.* **28**, 947 (1962).
29. Haenszel and Taeuber analyzed data for 683 white American females who died of lung cancer, and for a control group. They found the crude rate of death from lung cancer to be 1.32 times as high in urban areas as in rural areas for 1958–1959 and 1.29 times as high for 1948–1949 (in subjects 35 years and older, with adjustments made for

age). When adjustments were made for both age and smoking history, the ratio was 1.27. This ratio increased with the duration of residence in the urban or rural area, from 0.80 for residence of less than 1 year to 1.76 for lifetime residence. See W. Haenszel and K. E. Taeuber, *J. Nat. Cancer Inst.* **32**, 803 (1964).

30. L. D. Zeidberg, R. J. Horton, and E. Landau [*Arch. Environ. Health* **15**, 214 (1967)] are not able to isolate an air pollution effect on mortality from lung cancer from data for Nashville for the years 1949 through 1960; C. A. Mills [*Amer. J. Med. Sci.* **239**, 316 (1960)] investigated rates of death from lung cancer in Ohio. Stratifying according to the amount of driving done by the decreased, he found that the death rate varied with driving and urban exposure; L. Greenburg, F. Field, J. I. Reed, M. Glasser [*Arch. Environ. Health* **15**, 356 (1967)] investigated 1190 cancer deaths that occurred on Staten Island between 1959 and 1961 and found a relationship between lung cancer and air pollution; M. L. Levin, W. Haenszel, B. E. Carroll, P. R. Gerhardt, V. H. Handy, S. C. Ingraham II [*J. Nat. Cancer Inst.* **24**, 1243 (1960)] found significant differences between urban and rural mortality rates (for periods around 1950) in New York State, Connecticut, and Iowa. For males, the death rates were 41 percent higher in urban areas in New York, 57 percent higher in Connecticut, and 184 percent higher in Iowa. For females, the differences were 7 percent, 24 percent, and 47 percent, respectively; P. Buell, J. E. Dunn, L. Breslow [*Cancer* **20**, 2139 (1967)] utilized 69,868 questionnaires covering 336,571 man-years, in their study of lung cancer in California veterans. They found rates of death from lung cancer (adjusted for differences in age and smoking habits) to be 25 percent higher in the major metropolitan areas than in the less urbanized areas. Among nonsmokers, the rates of death from lung cancer were 2.8 to 4.4 times as high for major metropolitan areas as for more rural areas.
31. P. Buell and J. E. Dunn, Jr., *Arch. Environ. Health* **15**, 291 (1967).
32. W. Winkelstein, Jr., and S. Kantor, *ibid.* **18**, 544 (1969).
33. For economic level 2 (see *22*), the mortality rate per 100,000 for gastric cancer in white males 50 to 69 years old changed from 45 to 41, 48, and 84 as the pollution level (see *22*) rose. For economic level 4, the rates were 15, 38, and 63 for the first three pollution levels. For white women 50 to 69 years old, the death rates for economic level 2 were 8, 18, 25, and 40 per 100,000. For economic level 4, the death rates were 5 and 21 for the first two pollution levels.
34. R. M. Hagstrom, H. A. Sprague, E. Landau, *Arch. Environ. Health* **15**, 237 (1967).
35. The four measures of pollution are suspended particulates (soiling), dustfall, SO_3 and SO_2. For all cancer deaths, the number per 100,000 for middle class residents (defined to include about 75 percent of all residents) fell from 153 for high-pollution areas, to 130 for moderate-pollution areas, to 124 for low-pollution areas when a soiling index (concentration of haze and smoke per 1000 linear feet) was used to classify air pollution. When SO_3 (milligrams per 100 square centimeters per day) was used as a basis for classification, the corresponding death rates were 150, 129, and 145, respectively. With dustfall as a measure, the figures were 145, 130, and 131, and with 24-hour SO_2, in parts per million, they were 141, 129, and 138.
36. M. L. Levin, W. Haenszel, B. E. Carroll, P. R. Gerhardt, V. H. Handy, S. C. Ingraham II, *J. Nat. Cancer Inst.* **24**, 1243 (1960).
37. P. E. Enterline, A. E. Rikli, H. I. Sauer, M. Hyman, *Public Health Rep.* **75**, 759 (1960).
38. L. D. Zeidberg, R. J. M. Horton, E. Landau, *Arch. Environ. Health* **15**, 225 (1967).
39. When air-pollution level was measured on the basis of sulfation (SO_3, in milligrams per 100 square centimeters per day), the morbidity rates (for white, middle-class males aged 55 and older) were 64.0 man-years per 1000 man-years for high-pollution areas, 34.1 for moderate-pollution areas, and 36.8 for low-pollution areas. Measurement of air pollution on the basis of 24-hour concentrations of SO_2 gave morbidity rates of 47.2, 36.8, and 22.2, respectively. For these same white, middle-class males, in areas of high atmospheric concentrations of SO_3, the mortality rate was 425.6 per 100,000 population; in moderate-concentration areas, 327.41; and in low-concentration areas, 361.97. With SO_2 concentrations as a measure, the corresponding figures were 424.87, 319.19, and 364.93. When soiling (smoke or suspended particles) was used as the pollution index, the figures were 376.65, 339.13, and 399.88, respectively.
40. G. Friedman, *J. Chronic Dis.* **20**, 769 (1967).
41. The effect of air pollution on pneumonia, tuberculosis, and asthma has also been investigated. C. Daly (see *25*) reports simple correlations of .60 for pneumonia mortality and pollution from domestic fuel and .52 for pneumonia mortality and pollution from industrial fuel. For tuberculosis mortality the correlations are .59 and .22, respectively. The death rates for pneumonia rise from 30 to 52 per 100,000, and those for tuberculosis rise from 47 to 89, as one goes from rural settings to conurbations. Stocks (*19*) reports data on pneumonia mortality, by sex, for 26 areas of northern England and Wales. As shown by regressions 27 through 30 in Table 1, there appears to be a strong relationship between a smoke index and pneumonia mortality. The relationship is much stronger for men than for women. C. A. Mills [*Amer. J. Hyg.* **37**, 131 (1943)], in a classic study of wards in Pittsburgh and Cincinnati for 1929–30, reports substantial correlation between pneumonia death rates and local pollution indices. He found the correlation between dustfall and rates for pneumonia mortality in white males to be .47 for Pittsburgh and .79 for Cincinnati. The actual variation in these death rates is 41 to 165 per 100,000 population for Cincinnati and 0 to 7852 for Pittsburgh. Mills argues that omitted socio-economic variables could not account for these correlations, but he made no attempt to control for such variables in his studies. He also found that death rates fell significantly as the altitude of an individual's residence increased; there was a drop of approximately 10 percent in death rate for every 100 feet (30 meters) of elevation [see also C. A. Mills, *Amer. J. Med. Sci.* **224**, 403 (1952); E. Gorham, *Lancet* **1959-II**, 287 (1959)]. Zeidberg, Prindle, and Landau [*Amer. Rev. Resp. Dis.* **84**, 489 (1961)] studied 49 adult and 35 child asthma patients for a year. They found that the attack rate (attacks per person per day) for adults rose from .070 during days when atmospheric concentrations of sulfates were low to .216 when concentrations were high. In children, the effect of increased concentrations of sulfates was insignificant. Schoettlin and Landau [*Public Health Rep.* **76**, 545 (1961)] investigated 137 asthma patients in Los Angeles during the fall months. They found that 14 percent of the variance in daily attacks ($n = 3435$) could be explained by the maximum atmospheric concentrations of oxidants for that day. These two studies document a strong relationship between asthma and air pollution; Lewis, Gilkeson, and McCaldin [*Public Health Rep.* **77**, 947 (1962)] found no association between the daily frequency of visits to charity hospitals for treatment of asthma attacks and measures of air pollution.
42. J. W. B. Douglas and R. E. Waller, *Brit. J. Prev. Soc. Med.* **20**, 1 (1966).
43. A. S. Fairbairn and D. D. Reid, *ibid.* **12**, 94 (1958).
44. L. D. Zeidberg, R. A. Prindle, E. Landau, *Amer. J. Public Health* **54**, 85 (1964).
45. Morbidity rates associated with a soiling index were 140, 122, and 96, respectively, for high, moderate, and low pollution; corresponding rates associated with an SO_2 index were 177, 117, and 81. For white females, morbidity rates associated with an SO_3 index were 169, 134, and 160; with a soiling index, 158, 139, and 127; and with an SO_2 index, 172, 136, and 116. For nonwhite males, the morbidity rates associated with an SO_3 index were 86 for high concentrations and 84 for moderate and low concentrations; corresponding rates associated with a soiling index were 94 and 67, and with an SO_2 index, 84 and 88. For nonwhite females, morbidity rates of 136 and 140 were associated with high and with moderate and low SO_3 concentrations, respectively; corresponding rates associated with soiling were 140 and 129, and with SO_2 concentrations, 145 and 126. The effects for working women and for housewives, between the ages of 14 and 65, were similar.
46. E. C. Hammond, paper presented at the 60th annual meeting of the Air Pollution Control Association, 1967.
47. S. Ishikawa, D. H. Bowen, V. Fisher, J. P. Wyatt, *Arch. Environ. Health* **18**, 660 (1969).
48. W. W. Holland and D. D. Reid, *Lancet* **1965-I**, 445 (1965).
49. D. D. Reid, *ibid.* **1958-I**, 1289 (1958).
50. C. J. Cornwall and P. A. B. Raffle, *Brit. J. Ind. Med.* **18**, 24 (1961).
51. F. C. Dohan, *Arch. Environ. Health* **3**, 387 (1961); ——— and E. W. Taylor, *Amer. J. Med. Sci.* **240**, 337 (1960).
52. H. A. Sprague and R. Hagstom, *Arch. Environ. Health* **18**, 503 (1969).
53. L. B. Lave and E. P. Seskin, in preparation.
54. "County and City Data Book," *U.S. Dep. Commerce Publ.* (1962); "Analysis of Suspended Particulates, 1957–61," *U.S. Public Health Serv. Publ. No. 978* (1962); "Vital Statistics of the United States (1960)," *U.S. Dep. Health Educ. Welf. Publ.* (1963); "Vital Statistics of the United States (1961)," *U.S. Dep. Health Educ. Welf. Publ.* (1963).
55. For a discussion of the limitations of these studies, see B. G. Ferris, Jr., and J. L. Whittenberger (*7*) and J. R. Goldsmith, *Arch. Environ. Health* **18**, 516 (1969).
56. See "Smoking and Health Report of the Advisory Committee to the Surgeon General of the Public Health Service," *U.S. Public Health Serv. Publ. No. 1103* (1964), p. 362.
57. This might be explained by noting that farmers tend to be exposed to a high level of pollution in the course of their work (from fertilizers, insecticides, and the exhaust fumes from farm equipment), which causes more deaths from respiratory disease than would be expected from the low level of general air pollution in rural areas.
58. See, for example, T. Toyama (*16*) and F. L. Petrilli, G. Agnese, S. Kanitz, *Arch. Environ. Health* **12**, 733 (1966).
59. L. D. Zeidberg, R. J. M. Horton, E. Landau, *Arch. Environ. Health* **15**, 214 (1967).
60. R. A. Prindle G. W. Wright, R. O. McCaldin, S. C. Marcus, T. C. Lloyd, W. E. Bye, *Amer. J. Public Health* **53**, 200 (1963).
61. D. P. Rice, "Estimating the Cost of Illness," *Public Health Serv. Publ. No. 947-6* (1966).
62. The category "diseases of the respiratory system" encompasses numbers 470 through 527 of the 1962 International Classification of Diseases, Adapted (ICDA). A report of the Commission on Professional and Hospital Activities, entitled *Length of Stay in Short-Term General Hospitals (1963–1964)* (McGraw-Hill, New York, 1966), gives details on the average lengths of stay and number of patients in 319 U.S. general hospitals for 1963 and 1964 by specific ICDA classifications. From these figures we were able to compute the ratio of total hospitalization by specific disease to total hospitalization for all respiratory diseases. Of the 2,410,900 inpatient days for all respiratory diseases, 232,222 were for acute bronchitis and 177,232 were for "bronchitis, chronic and unspecified." Thus, approximately 17 percent of all inpatient days for respiratory diseases were for some form of bronchitis. On the basis of current hospitalization rates, we find the direct cost of diseases of the respiratory system to be $1581 million annually. An estimated 17 percent of this amount is due to bronchitis; thus, the direct cost of bronchitis is about $268.8 million annually.
63. To calculate the indirect cost of bronchitis, we must do more than take 17 percent of the total indirect cost ($3,305,700) of all diseases of the respiratory system. Almost 50 percent of respiratory disease patients are hospitalized for "hypertrophy of tonsils and adenoids" (ICDA 510). Hospitalization is categorized by age of patient in the Commission on Professional and Hospital Activities report, and we note that 80 percent of these "tonsil and adenoid" patients were under 20 years of age. Thus, it seems clear that the "forgone earnings" of these patients is negligible, and so no indirect costs should be allocated to this group. We therefore excluded the hospitalization of "tonsil and adenoid" patients before computing the percentage of hospitalization due to bronchitis. Thus, we estimated that 20 percent of the indirect cost of respiratory disease can be ascribed to bronchitis.
64. There is one bit of evidence that 25 to 50 percent of total morbidity (and therefore mortality) can be associated with air pollution; see L. D. Zeidberg, R. A. Prindle, E.

Landau, *Amer. J. Public Health* **54**, 85 (1964). If one accepted this evidence as conclusive, it would follow that the annual cost of air pollution, because of health effects, would run between $14 billion and $29 billion.
65. See J. H. Schulte, *Arch. Environ. Health* **7**, 524 (1963); A. G. Cooper, "Carbon Monoxide," *U.S. Public Health Serv. Publ. No. 1503* (1966); *Effects of Chronic Exposure to Low Levels of Carbon Monoxide on Human Health, Behavior, and Performance* (National Academy of Sciences and National Academy of Engineering, Washington, D.C., 1969).
66. Another way to estimate the cost of air pollution is to examine the effect of air pollution on property values. See R. J. Anderson, Jr., and T. D. Crocker, "Air Pollution and residential property values," paper presented at a meeting of the Econometric Society, New York, December 1969; H. O. Nourse, *Land Econ.* **43**, 181 (1967); R. G. Ridker, *Economic Costs of Air Pollution* (Praeger, New York, 1967); R. G. Ridker and J. A. Henning, *Rev. Econ. Statist.* **49**, 246 (1967); R. N. S. Harris, G. S. Tolley, C. Harrell, *ibid.* **50**, 241 (1968).
67. P. Buell, J. E. Dunn, Jr., L. Breslow, *Cancer* **20**, 2139 (1967).
68. E. C. Hammond and D. Horn, *J. Amer. Med. Ass.* **166**, 1294 (1958).
69. P. Stocks, "British Empire Cancer Campaign," supplement to "Cancer in North Wales and Liverpool Region," part 2 (Summerfield and Day, London, 1957).
70. G. Dean, *Brit. Med. J.* **1**, 1506 (1966).
71. A. H. Golledge and A. J. Wicken, *Med. Officer* **112**, 273 (1964).
72. W. Haenszel, D. B. Loveland, M. G. Sirken, *J. Nat. Cancer Inst.* **28**, 947 (1962).
73. The research discussed in this article was supported by a grant from Resources for the Future, Inc. We thank Morton Corn, Allen Kneese, and John Goldsmith for helpful comments. Any opinions and remaining errors are ours.

A Theory of the Origin of the State

Traditional theories of state origins are considered and rejected in favor of a new ecological hypothesis.

Robert L. Carneiro

For the first 2 million years of his existence, man lived in bands or villages which, as far as we can tell, were completely autonomous. Not until perhaps 5000 B.C. did villages begin to aggregate into larger political units. But, once this process of aggregation began, it continued at a progressively faster pace and led, around 4000 B.C., to the formation of the first state in history. (When I speak of a state I mean an autonomous political unit, encompassing many communities within its territory and having a centralized government with the power to collect taxes, draft men for work or war, and decree and enforce laws.)

Although it was by all odds the most far-reaching political development in human history, the origin of the state is still very imperfectly understood. Indeed, not one of the current theories of the rise of the state is entirely satisfactory. At one point or another, all of them fail. There is one theory, though, which I believe does provide a convincing explanation of how states began. It is a theory which I proposed once before (*1*), and which I present here more fully. Before doing so, however, it seems desirable to discuss, if only briefly, a few of the traditional theories.

Explicit theories of the origin of the state are relatively modern. Classical writers like Aristotle, unfamiliar with other forms of political organization, tended to think of the state as "natural," and therefore as not requiring an explanation. However, the age of exploration, by making Europeans aware that many peoples throughout the world lived, not in states, but in independent villages or tribes, made the state seem less natural, and thus more in need of explanation.

Of the many modern theories of state origins that have been proposed, we can consider only a few. Those with a racial basis, for example, are now so thoroughly discredited that they need not be dealt with here. We can also reject the belief that the state is an expression of the "genius" of a people (*2*), or that it arose through a "historical accident." Such notions make the state appear to be something metaphysical or adventitious, and thus place it beyond scientific understanding. In my opinion, the origin of the state was neither mysterious nor fortuitous. It was not the product of "genius" or the result of chance, but the outcome of a regular and determinate cultural process. Moreover, it was not a unique event but a recurring phenomenon: states arose independently in different places and at different times. Where the appropriate conditions existed, the state emerged.

Voluntaristic Theories

Serious theories of state origins are of two general types: *voluntaristic* and *coercive*. Voluntaristic theories hold that, at some point in their history, certain peoples spontaneously, rationally, and voluntarily gave up their individual sovereignties and united with other communities to form a larger political unit deserving to be called a state. Of such theories the best known is the old Social Contract theory, which was associated especially with the name of Rousseau. We now know that no such compact was ever subscribed to by human groups, and the Social Contract theory is today nothing more than a historical curiosity.

The most widely accepted of modern voluntaristic theories is the one I call the "automatic" theory. According to this theory, the invention of agriculture automatically brought into being a surplus of food, enabling some individuals to divorce themselves from food production and to become potters, weavers, smiths, masons, and so on, thus creating an extensive division of labor. Out of this occupational specialization there developed a political integration which united a number of previously independent communities into a state. This argument was set forth most frequently by the late British archeologist V. Gordon Childe (*3*).

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