vascular lesions affecting the central nervous system, and diseases of the heart. Accidents, homicides, suicides, and deaths in early infancy remained normal (Greenburg et al. 1967). A linear relationship between SO_2 concentration and excess mortality in Oslo and London has been demonstrated (Sweden 1972, p. 33). A two-year study in Buffalo, New York, divided the city into four areas on the basis of average measured pollution (suspended particulate levels) and then correlated the death rate from all causes with pollution and with economic level as shown in Figure 2-42.

A painstaking multiple regression analysis of the impact of air pollution and socioeconomic factors on the death rates in 114 U.S. metropolitan areas indicates a strong correlation between minimum measured air pollution and mortality (Lave and Seskin 1970). The conclusion of that study is that a 10 percent decrease in the minimum concentration of measured particles would decrease the total death rate by 0.5 percent; a 10 percent decrease in the concentration of sulfates would decrease the death rate by 0.4 percent; and an increase in density of 1 person per acre, holding all pollution and socioeconomic factors constant, would increase the death rate by 0.2 percent.

A proper dynamic representation of the local pollution aspect of crowding would relate population density to average exposure to various sorts of local pollutants and then relate pollution exposure to changes in life expectancy. For a particular geographic area such a model would require a great deal of information about human settlement patterns, industrial activities, and weather, as well as epidemiological data. On a broad global scale it is probably sufficient to recognize only the difference between rural and urban populations. If only 10 percent of the population lives in urban areas, 10 percent of the population is exposed to local urban pollution. The amount of exposure received by the urban population depends on the general type and level of industrial activity and on the measures taken to reduce pollution generation from that activity. The causal diagram in Figure 2-43 represents two assumptions: that all city dwellers are equally exposed to pollutants generated in cities, and that virtually all industrial air pollution is generated in the vicinity of cities.

Economic Level	Particulate Level 1 (Low)	2	3	4 (High)	Total
1 (low)	—	36	41	52	43
2	24	27	30	36	29
3	—	24	26	33	25
4	20	22	27	—	22
5 (high)	17	21	20	—	19
Total	20	24	31	40	26

Figure 2-42 Correlation between particulate levels and death rate

Note: Average annual death rates per 1,000 population from all causes according to economic and particulate levels: white males, 50–69 years of age, Buffalo and environs, 1959–1961.

Source: Winkelstein 1967.

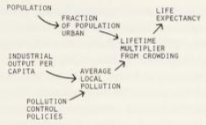

Figure 2-43 Influence of crowding on life expectancy through local pollution

4. Social stress. Since the triumph of medicine over infectious diseases, many researchers in epidemiology have turned to the study of the chronic, degenerative diseases that are now the leading causes of death in the industrialized countries of the world. From this study a new theory of disease is emerging, one that suggests two equally important factors in human health: the presence of harmful foreign substances or virulent microorganisms, and the ability of the body to resist the harmful incursions of these substances or microorganisms. For some diseases, but not all, the resistance or intrinsic health of the human body may be the determining factor in the manifestation of the disease.

> . . . the microbial diseases most common in our communities today arise from the activities of microorganisms that are ubiquitous in the environment, persist in the body without any obvious harm under ordinary circumstances, and exert pathological effects only when the infected person is under conditions of physiological stress. In such a type of microbial disease, the event of infection is of less importance than the hidden manifestations of the smouldering infectious process and than the physiological disturbances that convert latent infection into overt symptoms and pathology. [Dubos 1965, pp. 164–165]

The "physiological stress" leading to disease has been interpreted by several authors to include social or psychological stress that in turn seem to be associated with industrialization, urbanization, and crowding (for a review, see Dodge and Martin 1970, chap. 2). Numerous indications of a relationship between crowding and physiological stress have been cited. They range from observations of animal behavior under crowding (Calhoun 1962, Welch 1964) to measurements of blood pressure as a function of age in different rural and urban societies (summarized in Cassel 1971). A careful statistical analysis of crowding in the city of Chicago indicates a clear correlation between social pathologies, including higher mortality, and crowding (measured by persons per room and rooms per housing unit) even when other socioeconomic variables are controlled (Galle, Gove, and McPherson 1972). In the United States the age-adjusted death rate for arteriosclerotic heart disease is 301.3 per hundred thousand in highly urbanized California and only 155.5 in largely rural New Mexico. The age-adjusted death rate from coronary heart disease in New York City is consistently higher than the age-adjusted death rate from *all* causes in North Dakota (Dodge and Martin 1970, p. 8). Certainly these differentials may be partially attributed to local pollution, diet, and exercise rather than social stress. It is more