Although some of these facts are known for some pollutants, all the information required for a dynamic analysis is not yet available for any single pollutant, much less for the entire mix of pollutants actually encountered by the globe's population. We shall give a few specific examples here merely to establish the fact that global pollutants may come to have a significant influence on human life expectancy, not to give an exhaustive summary of the field of environmental health.

The heavy metal lead is a fairly well understood and carefully measured persistent pollutant with proved deleterious effects on human health. As a local pollutant its concentration in the air averages 1.0 micrograms per cubic meter in U.S. cities; as a more widely distributed pollutant its air concentration averages 0.5 micrograms per cubic meter in U.S. rural areas (Chisolm 1971). Peak readings in New York City have reached 34 micrograms per cubic meter (Bazell 1971). As an aerosol, lead is globally distributed by air currents, and its rate of deposition in the Greenland ice sheet appears to be rising rapidly (Weiss, Koide, and Goldberg 1971; see also discussion in Dickson and Patterson et al. 1972). The intake of lead in food and water in the United States averages 300 micrograms per person per day (Schroeder 1970). Approximately 5 percent of the lead ingested and 40 percent of that inhaled are retained in the body (Patterson 1965).

Symptoms of lead toxicity begin to appear in adults with blood lead concentrations of 60–80 micrograms per 100 milliliters (ml). For comparison with that figure, blood samples from residents of the rural United States averaged 16 micrograms per 100 ml and urban citizens averaged 21 micrograms per 100 ml (Chisolm 1971). One-fourth of 80,000 preschool children tested in New York City had blood lead concentrations of 40 micrograms per 100 ml or more. Newborn babies in New York City average the same blood lead concentrations as their mothers—20–30 micrograms per 100 ml (Bazell 1971). The delay between high blood lead levels and observable symptoms may be only a few weeks when the levels are high (leading to acute plumbism) but as long as ten years when levels are maintained at the lower limits of 60–80 micrograms per 100 ml (leading to chronic nephritis) (Chisolm 1971). Little statistical information is available about the effects of lead pollution on aggregate mortality, and none about the interactions of lead with other pollutants in the body.

As an example of a very different sort of pollutant, one of the most recently discovered and least understood of the global pollutants is polychlorobiphenyl (PCB). The abbreviation PCB actually stands for a whole family of closely related chlorinated hydrocarbons that have been used for a variety of industrial purposes since the 1920s. Polychlorobiphenyl was first identified as a common environmental contaminant in 1966, when it was found in bird feathers, fish, and human fat tissues (for a review, see Jensen 1972). The present PCB concentration in U.S. water samples ranges from 0.1 to 4.0 parts per billion, and in the average U.S. diet it is about 0.5 parts per million (Maugh 1972). The typical concentration in adipose tissue from American subjects is 1 part per million (Maugh 1972); the blood of Swedish subjects carried 10 parts per billion (Jensen 1972). Japanese patients exhibiting acute PCB intoxication from an industrial accident had concentrations of about 29 parts per million. Although human deaths from acute PCB poisoning have occurred (Jensen 1972) and PCB has caused great disruption in wild and domestic animal populations (Maugh 1972), PCB is not believed to be very toxic in low concentrations. The long-term influence of low-level exposure on total mortality is unknown. PCB is chemically similar to DDT, and the retention of PCB in fat tissue seems to be enhanced by DDT (Södergren and Ulfstrand 1972). Other possible synergistic effects have not yet been explored.

Numerous other global persistent pollutants are known to have a negative influence on human health; obvious examples are cadmium, mercury, long-lived pesticides, and a number of radioactive isotopes. There are undoubtedly others that, like PCB, will only be discovered and fully characterized after years of regular use and accumulation in the environment. Given that even those pollutants that are recognized are not understood well enough to be represented in a complete dynamic model, it is clearly impossible to represent a lifetime multiplier from all pollutants on a global level with accuracy. On the other hand, some qualitative knowledge does exist, and that knowledge should be included in a global model, both as a summary of what is now known and as a placeholder for a more precise statement that may be added later as more information becomes available.

If an increase in the average 1970 global pollution level by a factor of 100 would have no effect on life expectancy, the lifetime multiplier from pollution would always be 1.0 and could be represented by the straight line A in Figure 2-53. Even the partial information currently available about global pollutants suggests that increases in ambient pollution levels by factors far less than 100 may have a significant negative effect on human life expectancy. Thus we have enough information about the effects of pollution on human health to rule out curve A and to eliminate all curves such as B that have a rising slope, which would imply that pollution increases life expectancy. The correct relationship must have a negative slope and must be included somewhere within the family of curves labeled C in Figure 2-53.

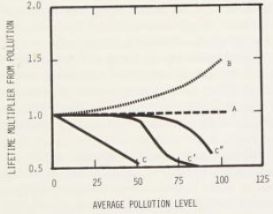

Figure 2-53 Possible effects of pollution on life expectancy