function of industrial output per capita IOPC. The equations for the relationship follow, and the table illustrating the range of CMI is shown in Figure 2-52.

```
CMI.K=TABHL(CMIT,IOPC.K,0,1600,200)                    27, A
CMIT=.5/.05/-.1/-.08/-.02/.05/.1/.15/.2                27.1, T
    CMI    - CROWDING MULTIPLIER FROM INDUSTRIALIZATION
               (DIMENSIONLESS)
    TABHL  - A FUNCTION WITH VALUES SPECIFIED BY A TABLE
    CMIT   - CMI TABLE
    IOPC   - INDUSTRIAL OUTPUT PER CAPITA (DOLLARS/
               PERSON-YEAR)

LMC.K=1-(CMI.K*FPU.K)                                  28, A
    LMC    - LIFETIME MULTIPLIER FROM CROWDING
               (DIMENSIONLESS)
    CMI    - CROWDING MULTIPLIER FROM INDUSTRIALIZATION
               (DIMENSIONLESS)
    FPU    - FRACTION OF POPULATION URBAN
               (DIMENSIONLESS)
```

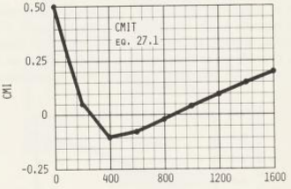

Figure 2-52 Crowding multiplier from industrialization table

The preceding equations incorporate the assumption that at very low industrial output per capita IOPC crowding affects life expectancy entirely through the spread of infectious disease. We estimate that the maximum reduction of life expectancy for urban populations under this condition is 50 percent, as illustrated by the line IOPC = 0 in Figure 2-51. Thus at IOPC = 0, the crowding multiplier from industrialization CMI equals 0.50 (see Figure 2-52). This estimate is based on the data from the seventeenth- and eighteenth-century European cities cited earlier. As IOPC rises, the threat of infectious disease decreases, and at an intermediate IOPC (225–900 dollars per person-year) we assumed that higher urbanization somewhat increases life expectancy by making health care programs easily accessible to the population (line IOPC = 400 in Figure 2-51). Above IOPC = 900, crowding again begins to have a deleterious effect because of local pollution and social stress-related

diseases. Crowding at high IOPC never becomes as detrimental to health as crowding at low IOPC. The maximum reduction in life expectancy from crowding in economically developed cities was assumed to be 20 percent (CMI = 0.20 at IOPC = 1600).*

As represented in World3, crowding implies no absolute upper limit to population density, as did crowding in World2. The main dynamic effect of crowding occurs only under the condition of a very high population combined with very low industrialization, one that is rarely generated in world model runs. It may be that an absolute psychosocial limit to crowding and urbanization could be encountered after another doubling or two of the earth's population. Since present systems offer ample evidence of local populations living and growing in extremely high-density environments, however, we did not postulate an absolute crowding limit, at least within the few doublings that the population is allowed by other limits in World3. If other modelers feel that such a crowding limit is justified, it can easily be added to the model equations.

Lifetime Multiplier from Pollution LMP The lifetime multiplier from pollution LMP expresses only the effect of globally distributed, persistent pollutants on human health. The effect of short-lived local pollution is included in the lifetime multiplier from crowding LMC discussed earlier. Interference of pollution with other forms of life, and thus with the human food supply, is represented in World3 by the land yield multiplier from air pollution and the land fertility degradation rate, both described in the agriculture sector (Chapter 4).

A complete dynamic analysis of the relationship between any single pollutant and the health of the human population should ideally be based upon the following information:

1. The present ambient concentration and concentration trends over time of the pollutant in the primary substances that enter the human body—air, water, and food.
2. The rate of absorption of the pollutant into human body tissues as a function of its concentration in air, water, and food.
3. The rate of excretion or metabolic transformation to a harmless form, as a function of the concentration of the pollutant in various tissues.
4. The relation between the concentration of the pollutant in various tissues and the appearance of pathologic symptoms, both short term and long term.
5. The quantitative relationship and the average delay time between pollution-induced pathology and mortality.
6. The extent to which the effects of the pollutant are enhanced or mitigated by the presence of other pollutants.

*In future models the bimodal curve shown in Figure 2-52 could be resolved into the two influences it actually represents: a decreasing crowding multiplier from effective health services per capita and an increasing crowding multiplier from industrial output per capita.