Inferring causation from correlations between statistics drawn from an unrepresentative sample of those states with adequate data is the kind of desperate maneuver to which we are driven by the problems of underfunded, policy-relevant evaluation in prevention and health education with youth. Sometimes we have the advantage of more complete data than Drs. Robertson and Zador could find, or comparable data on several points in time that would allow at least the correlation of time-lagged trends between program participation and outcomes such as fatal injuries. When left with no choice but to make do with the kind of cross-sectional statistics available to Robertson and Zador, we must acknowledge the numerous threats to validity and generalizability of conclusions drawn for policy. Most particularly we must caution the consumers of such reports to beware of the possible "fallacy of the ecological correlation,"[4] which might lead policy makers to infer that a positive correlation between the percentages of populations participating in our programs and the percentages of the same populations having positive outcomes means the same people who got the programs also had positive outcomes. In the data analyzed by Robertson and Zador, for example, there is no evidence that the teenagers who had the fatal crashes were the same teenagers who had the driver training.

It would be prudent, as Robertson and Zador suggest, for society to insist on more scientific testing of programs before their widespread application, but it would be wasteful policy and presumptuous advice to dismantle programs already widely established on the basis of such limited analysis as that of Robertson and Zador. Some of the more obvious alternative explanations for the correlations found need to be examined, such as alcohol use by teenagers and alcohol involvement in teenage crashes by state, socioeconomic factors that might simultaneously account for availability of automobiles to teenage drivers and availability of driver education programs in the schools.

As with other educational and public health methods, driver education is a potentially useful tool if applied at the proper time with the right population. The timing and targeting of preventive methods will be critical not only in maximizing their benefits but also in allocating resources for prevention. The policy question is not simply *whether* society should support driver education, but rather it is a complex question of who should receive driver education, through what channels, by what methods. Such questions in this, as in other areas of prevention, will require controlled evaluative studies on a scale comparable to the Food and Drug Administration's testing of new drugs. The difference is that preventive measures can be allowed and even encouraged before they have been fully evaluated. Obviously, however, they should not be designed or timed to place people at greater risk.

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Improving Life Expectancy: An Uphill Road Ahead

An article in this issue of the Journal[1] draws attention to one of the predicaments that underlies the slow health progress of recent years despite ever increasing numbers of medical personnel, better equipped hospitals, and larger expenditures on research. In the 35 years since 1940, national per capita expenditures for health care (in 1967 dollars) have gone from under $100 to over $300. Meanwhile life expectancy went up less than 15 per cent.

The American health system is not without its defects. But we might well be dissatisfied even if it were perfect and every dollar of its hundred billion dollar budget were spent to maximum effect. For there appears to be an intrinsic ceiling against which present and prospective improvements are pressing.

Cancer deaths will serve as an example. We define cancer deaths as those due to malignant and benign neoplasms—causes B-18 and B-19 in the International List. These now number 350,000 per year and are one-sixth of all deaths. If a general cure for cancer were discovered and applied this month, there would be about 30,000 fewer deaths next month, and nearly 350,000 fewer next year. Mortality would seem to be permanently lowered by one-sixth and life expectation increased by one-sixth.

Unfortunately this last statement would be true only if the population were homogeneous, in the sense that everyone stood the same chance of dying of cancer and other causes. Only in such a population would the reduction of the deaths and the death rate by one-sixth extend the ex-

pectation of life by one-sixth. Only then could each of us expect to live 12 more years as a result of the discovery of a cure for cancer.

In fact, the population is not homogeneous. Some of us stand a much greater chance of dying of cancer and other diseases than do others, and this wholly vitiates the simple proportionate calculation. If the people who are rescued from cancer are at high risk from heart disease and other causes, then that greatly diminishes the number of years added to human life by the discovery of a cancer cure.

We lack data on most of the risk factors in cancer or other diseases. Fortunately, however, one of the risks is well documented: plentiful data are available to show, for example, that a 20-year-old man has just one chance in 10,000 of dying of cancer within the year, while a man of age 70 has one chance in 100. The man of 20 has an expected 50 more years to live; the man of 70 has only 10. Most of the cases of cured cancer would necessarily be at the ages at which cancer predominantly occurs—among older people. If most of the cures are on people with only 10 years to go, and if cancer deaths are one-sixth of all deaths, then the average extension of life will be about one-sixth of 10, or less than two years. Granted that this is a rough way to do the calculation, but it illustrates the point: universal elimination of cancer would increase life expectancy by only about two years—not the 12 years that would apply if the population were homogeneous.

During the first month after a cure for cancer was deployed throughout the nation, there would be a decrease of 30,000 in the number of deaths, but, because of the shape that the top survivorship curve of the figure attained, as the people who did not die moved along in age they would succumb to the other causes of death, and especially the cardiovascular renal group. After the abrupt fall, the number of deaths would begin to rise again within two or three years, not quite replacing the former 30,000 per month, but about six-sevenths of that figure.

For all causes of death together, the results can be expressed in terms of a constant H that tells what per cent increase in life expectancy would result from a drop of 1 per cent in all age-specific rates. The constant H tends to fall with time as the curve of survivorship rises. For Swedish women, H is currently down to 0.12, i.e., a drop of all age-specific death rates by 1 per cent increases the expectation of life by only 0.12 per cent. When everyone dies between ages 75 and 85, the constant H will be close to zero; further percentage improvement in mortality applied to the age-specific rates will make no appreciable difference to the expectation of life. Since no one is really interested in death rates—what we all worry about is life expectancy—this result is important and discouraging.

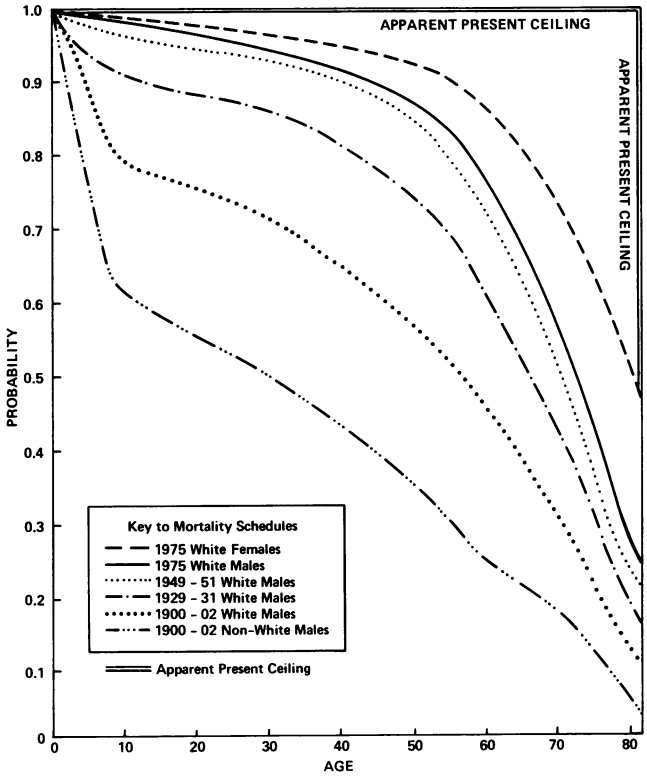
These and other effects are demonstrated in the article by Tsai, Lee, and Hardy.[1] They show that a 30 per cent reduction in deaths due to malignant neoplasms would extend life by only 0.71 years. Their results are in accord with the conclusions of Donald Shepard and Richard Zeckhauser,[2] as well as myself, working at the Harvard School of Public Health.

But even the gain so calculated (two years if cancer is eliminated) is almost certainly an overestimate of the benefit. For within any given age group, the people subject to any one ailment tend to have higher than average risks from other ailments.

Physicians become aware of the clustering of causes of death when they fill out death certificates. The determination of the cause of a particular death depends on judgment—often on simple guessing—because of the strong tendency to multiple causes. Any positive correlation among risks at given ages means that the true effect of eliminating a particular cause of death, or of eliminating a fraction of the deaths due to a particular cause, is less than shown by the age calculations of Tsai and colleagues.[1]

There are exceptions, of course. Precautions at ski resorts save the lives of people who are in better health than the average for their age, and so have more than a proportional effect on expectation of life. Since accidental fatalities in general often occur at young ages and to healthy people, their reduction has more effect on length of life than an equal absolute reduction in heart disease deaths. But for most of the causes of death, the effect of therapy that reduces age-specific rates is much less than a proportional difference in the mean age at death.

This was less true in the past. Since 1900, the expectation of life at birth has risen 25 years, due mostly to the virtual elimination of acute infectious diseases. Such diseases tended to strike young people, and when they were avoided the person whose life was saved had a long time to live before the next serious ailment caught up with him. We cannot expect the large payoff in life expectancy from ad-



Probability of Living to a Given Age, Based on U.S. Deaths for Four Periods

vances against cancer that followed the conquest of diphtheria and tuberculosis.

The accompanying chart shows, for selected U.S. population segments, the probability of surviving to various ages at various times in the twentieth century. At present rates, over 88 per cent of the white female children born live to age 60. This survivorship can be compared to those for other groups and earlier dates in the chart. The greatest contrast is with the non-white males in 1900–1902, at whose rates only 24 per cent lived to age 60. (Averages are from the Death Registration states up to 1928, for the United States after that. They represent the mortality to which people of the several ages were subject in the specified years.)

We can easily imagine a time when close to 100 per cent of the entire population—black and white, male and female—live to age 60, and indeed to age 70 or 80. To believe in such an extrapolation does not call for excessive optimism or imagination, since the chart shows white females of 1975 as having already traversed most of the distance to this goal, represented by the horizontal line dropping to zero after age 80. Then the question will be posed even more clearly than now—what can be done to increase the expectation of life at the oldest ages? The improvement at age 70 has been, at best, about four years since the beginning of this century; for white males, about two years. There is now little variation among racial groups. The expectation of life for black and white males alike averages a little over 10 years at age 70. Hence we cannot hope for much gain in years of life after 70 by raising the economically disadvantaged.

It is almost as though the most favored sector of the population—white females who are reasonably well-off economically—with a life expectancy now close to 80 years, shows us the way the rest of the population is going. Even if all social, racial, and sex groups could be pulled up to the same level as the most favored, the overall expectation of life would not be more than 80 years. Parenthetically, one should caution that whether rates for males will follow those for females is uncertain, given the increasing differential; males now lag eight years behind females in life expectancy at birth.

Is an expectation of 80 years the intrinsic ceiling against which medicine is pressing? Is future progress to consist solely in extending present achievements to less favored groups? Important as it is to do this, we cannot easily reconcile ourselves to a condition where progress on the frontier suddenly comes to an end.

What will be needed, then, to break through the barrier that now seems to be set at about 80 years if "mere" eradication of cancer does not even come close to doing it? The coincidence in age of the presently remaining causes of death suggests that, beyond attacking the individual causes, future research should focus on what underlies them all—the deterioration and senescence of the cells of the human body.

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