SPECIAL ARTICLES

PROGRESS AGAINST CANCER?

JOHN C. BAILAR III AND ELAINE M. SMITH

Abstract We assessed the overall progress against cancer during the years 1950 to 1982. In the United States, these years were associated with increases in the number of deaths from cancer, in the crude cancer-related mortality rate, in the age-adjusted mortality rate, and in both the crude and the age-adjusted incidence rates, whereas reported survival rates (crude and relative) for cancer patients also increased.

In our view, the best single measure of progress against cancer is change in the age-adjusted mortality rate associated with all cancers combined in the total population. According to this measure, we are losing the war against cancer, notwithstanding progress against several uncommon forms of the disease, improvements in palliation, and extension of the productive years of life. A shift in research emphasis, from research on treatment to research on prevention, seems necessary if substantial progress against cancer is to be forthcoming. (N Engl J Med 1986; 314:1226-32.)

THE primary purpose of this article is to assess the overall progress against cancer during the years 1950 to 1982, the most recent year for which reliable data are available. During this time there was very rapid and extensive growth of private and governmental support of research on cancer, and toward the end of the period there was also substantial emphasis on the effective delivery of research results to physicians, patients, and the public. It is time for an open debate to take stock of past achievements and to consider what levels of funds should be invested in what kinds of future efforts. We offer some observations and interpretations relevant to such a debate.

In 1962, cancer was the recorded cause of death for 278,562 Americans. In 1982, just 20 years later, 433,795 persons died of cancer — a 56 percent increase (Table 1). But the population was growing, and the proportions of persons in older age categories were changing. Crude mortality rates, which adjust for population size, increased by 25 percent (from 151.0 to 188.8 per 100,000) in this 20-year period, and age-adjusted mortality rates, which adjust for changes in age distributions as well as population size, increased by only 8.7 percent (from 170.2 to 185.0 per 100,000).

Mortality data do not tell the whole story. We might ask, not how many Americans die of cancer, but how many contract the disease. From 1973 to 1981 the crude incidence rate for all neoplasms combined rose by 13.0 percent, and the age-adjusted incidence rate by 8.5 percent (Table 1).

Or, we might focus on neither incidence nor mortality, but on the long-term survival of patients who have had a diagnosis of cancer. Unadjusted five-year survival rates for patients with all forms of cancer combined increased by 4.2 percent from 1973 to 1978 (from 38.5 to 40.1 percent), while rates adjusted for "expected" mortality from all other causes of death rose by 5.1 percent (from 46.8 to 49.2 percent).

From the Harvard School of Public Health, Boston, and the University of Iowa Medical Center, Iowa City. Address reprint requests to Dr. Bailar at the Department of Biostatistics, Harvard School of Public Health, Boston, MA 02115.

Supported in part by grants from the Alfred P. Sloan Foundation, the Mobil Corporation, and the University of Iowa Research Foundation.

Which of these conflicting pictures of change, if any, captures the "truth" about recent advances in the control of cancer? More specifically, what yardstick should we use to measure the overall success of the long and intense effort to control and eventually eliminate these diseases? Interest in this matter is sharpened by the recent announcement that the goal of the National Cancer Institute is a 50 percent reduction in cancer-related mortality (on an age-adjusted basis) by the year 2000.[3] To answer these questions, we first discuss the kinds of data that are available and the methods used to reduce them to simple index figures.[4,5] We then give our views on which measures are most appropriate and what they indicate.

MORTALITY DATA

In the United States, nearly all national cancer-related mortality data are derived from death certificates submitted through local and state channels to the National Center for Health Statistics. The Death Registration Area has included the entire United States since 1933. Major changes since then include five revisions of the standard system for coding causes of death, as well as continual improvement in medical procedures for antemortem diagnosis.[6-8] However, these changes have had less effect on the certification of deaths from cancer than on certification of deaths from other major causes.

Mortality records can be used in many ways, each of which is best suited for specific purposes. Sometimes there is a need for information about changes in mortality that are independent of demographic changes such as shifts in the age distribution of the population or shifts in place of residence (for geographically related cancers). "Adjusted" rates may be used to remove the effects of the variable or variables adjusted, so that other effects can be more easily detected and measured.[4,5]

One common method of adjustment for age is the "direct" method, which is a simple weighted average of observed age-specific rates, with weights determined by some fixed "standard" population, such as the U.S. population of 1980. For this paper all adjustments were made by the direct method with reference

Table 1. Cancer in the United States: Selected Measures of Recent Changes.*

Measure	Year		Total % Change	Average % Change/Yr
	1962	*1982*		
Mortality				
No. of deaths	278,562	433,795	55.7	+7.8
Crude rate†	151.0	188.8	25.1	+1.3
Age-adjusted rate†	170.2	185.1	8.7	+0.4
	1973	*1981*		
Incidence				
Crude rate†	365.2	412.7	13.0	+1.6
Age-adjusted rate†	368.2	399.4	8.5	+1.1
	1973	*1978*		
Five-year survival (%)‡				
Absolute survival rate	38.5	40.1	4.2	+0.8
Relative survival rate§	46.8	49.2	5.1	+1.0

*Sources: McKay et al.,[1] the National Center for Health Statistics,[2] and unpublished data from the SEER Program, National Cancer Institute.

†Rates are per 100,000 population. Age adjustments are to the 1980 U.S. population. Incidence data for 1981 include two areas not in the 1973 data; the base population reflects this change.

‡White population only.

§Relative to survival of the U.S. white population with the same age distribution.

to the U.S. population of 1980. There has been recent discussion about whether, in view of diagnostic errors at older ages, age-adjusted mortality rates should include the entire age span.[9,10] Ours do, because cancer is a common cause of death and because (contrary to the situation with some other causes of death) the available data do not suggest that net errors are so high as to make the figures unreliable for overall evaluation. Furthermore, changes in mortality rates for persons in specific age categories may be useful for understanding causes of cancer, but cannot measure progress against cancer in all age groups.

We believe that to study overall trends in cancer-related mortality (how they have changed in recent years and how they could change by the year 2000), the best single measure of mortality is the age-adjusted death rate associated with all cancers combined, supplemented by age-adjusted rates and sometimes age-specific rates, for specific sex and broad racial categories. These measures remove the effect of changing population size and changing distribution according to age, sex, and race.

Figure 1 shows age-adjusted mortality rates for all forms of cancer from 1950 to 1982 in the entire population and according to sex and race, with age adjusted to the 1980 population. Cancer-related mortality, measured in this way, rose steadily among white males; fell slightly, plateaued, and recently began to rise again among white females; rose rapidly and steadily among nonwhite males; and declined slightly and recently plateaued among nonwhite females. In all race and sex groups combined, there was a moderate increase in age-adjusted mortality. (The small discontinuity in 1957 is a result of a change in methods of classifying a cause of death on death certificates; more recent changes have had only a minor influence on cancer-related mortality rates.[6-8])

Site-Specific Mortality Data

Although mortality from all forms of cancer combined provides the most important information, study of specific sites (Fig. 2) can both illuminate the overall changes and show why the site-specific analyses alone may be misleading. To preserve comparability across sites, Figure 2 shows rates of each cancer (including the sex-specific cancers) relative to the total population. Rates of breast cancer among women only and rates of prostatic cancer among men only are approximately twice the rates given in this graph.

There has been no apparent change in mortality from breast cancer among white or nonwhite women since 1950. Rates among nonwhites (not shown) vary about their mean more than the rates among whites, but this appears to be due to the effect of smaller numbers of deaths and, hence, larger random variability.

The sharp and continuing rise in deaths from lung cancer (Fig. 2), nearly all from cigarette smoking, is now widely recognized as a medical, social, and political scandal. The increase was evident before 1950 among white and nonwhite men, and it has been evident among white and nonwhite women since the late

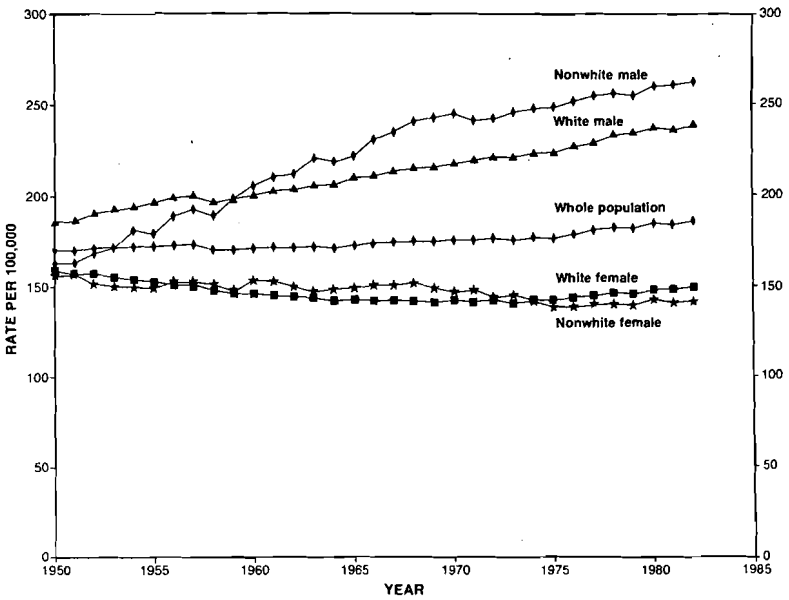

Figure 1. Mortality from All Malignant Neoplasms, 1950 through 1982, in the U.S. White Population and According to Race (White or Nonwhite) and Sex. Age was adjusted to the U.S. population of 1980.

1960s. These changes in death rates from lung cancer have substantially affected mortality rates from all cancers combined (Fig. 1). (Later on we will discuss the effect of excluding lung and other cancers from the trends shown in Figure 2.) Data on nationwide mortality trends with smokers and nonsmokers separated are not available.

Mortality from cancer of the prostate (Fig. 2) has not changed appreciably in the entire male population, despite continual increases among nonwhite men since 1950.

Mortality from stomach cancer (Fig. 2) has steadily declined in all four race and sex groups. This decline reflects changes in incidence rather than better methods of treatment, earlier diagnosis, or changes in definition.[6-8] Mortality from cervical cancer (not shown) has also declined dramatically as a result of widespread screening programs, improved standards of living, and a high rate of hysterectomy.[11]

Mortality from colorectal cancer (Fig. 2) has been declining slowly and steadily for reasons not fully understood but probably including better diagnostic procedures and improvements in treatment.

These data, taken alone, provide no evidence that some 35 years of intense and growing efforts to improve the treatment of cancer have had much overall effect on the most fundamental measure of clinical outcome — death. Indeed, with respect to cancer as a whole we have slowly lost ground, as shown by the rise in age-adjusted mortality rates in the entire population (Fig. 1). This is not to say that without these efforts at treatment the trends would have been the same, but overall, the effort to control cancer has failed — so far — to attain its objectives.

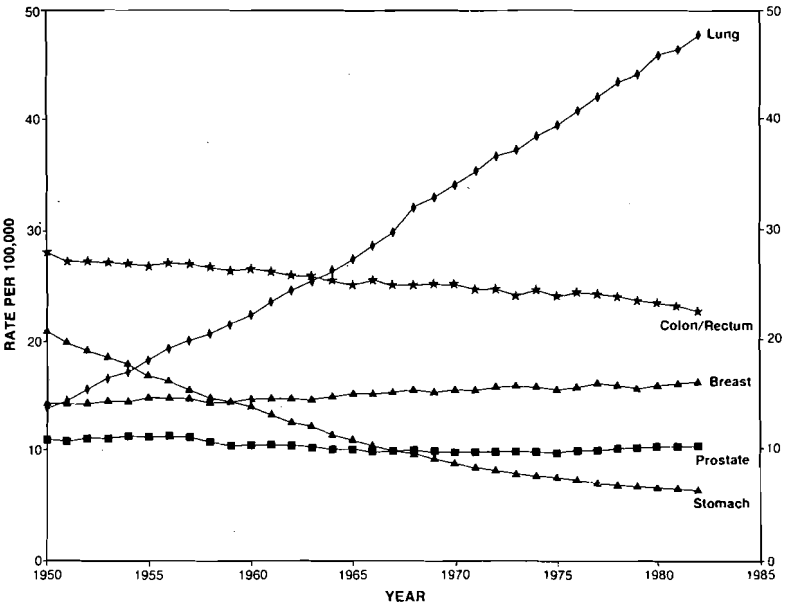

Figure 2. Mortality from Cancer of Selected Sites, 1950 through 1982, in the Total U.S. Population.
Age was adjusted to the U.S. population of 1980.

This generally dismal picture obscures some striking successes, however. For example, age-adjusted mortality from all cancers combined has dropped notably in patients under the age of 30, though such deaths account for only about 1 to 2 percent of total mortality from cancer.[12,13] In older persons, mortality from small-cell lung cancer and from non-seminoma testicular cancer has also decreased (data not shown).

Incidence Data

The possible measures for the incidence of cancer are similar to those for mortality from the disease — counts, crude rates, and several kinds of adjusted rates, each of which may be limited to particular demographic segments or particular forms of cancer.[4,5] The incidence statistic that we chose for a measure of overall progress against cancer is the direct age-adjusted rate for all cancers combined (U.S. 1980 standard), but supplemented by rates for certain narrower segments that illuminate specific problems.

Table 1 shows cancer incidence data from the SEER (Surveillance, Epidemiology, and End Results) Program, which was developed under the auspices of the National Cancer Institute. One can compare these statistics with the mortality data in Table 1, but keeping in mind that most superficial skin cancers are excluded, that the SEER data are for a nonrandom sample of about 10 percent of the U.S. population from 10 diverse geographic areas (4 states, 5 metropolitan areas, and Puerto Rico), that the series begins only in 1973 for 8 of 10 areas (the others were added in 1974 and 1975), and that the incidence data are subject to substantial shifts in diagnosis and reporting during that time.[14] Data on cancer incidence are limited to the white population because the number of nonwhites in the SEER population was too small to provide reliable estimates of risks and because the distribution of nonwhites across specific racial categories was substantially different from that in the United States as a whole.

Cancer incidence rates are shown in Figure 3. Overall trends are upward among both white males and white females, suggesting a failure to prevent or control new or current causes of cancer.

Site-Specific Incidence Data

The reported incidence rates for breast cancer show a distinct one-year peak in 1974 and a slower rise in more recent years (Fig. 4). The reported incidence of cancer of the prostate (Fig. 4) has increased slightly among white men and more sharply among nonwhite men (data not shown). Incidence rates for lung cancer have been ris-

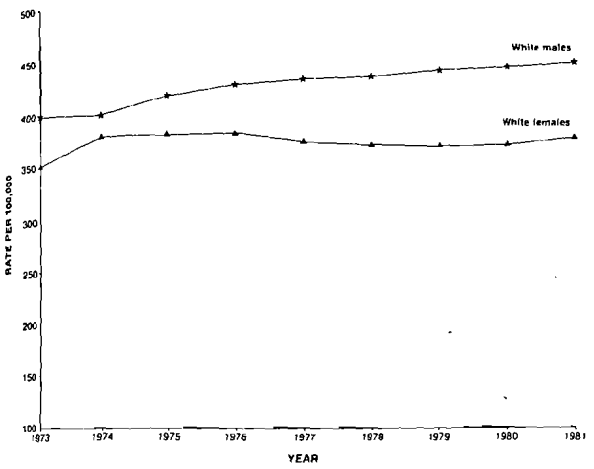

Figure 3. Incidence of All Cancers, 1973 through 1981, According to Sex, in the White Population of the SEER Registry Area.
Age was adjusted to the U.S. population of 1980.

ing rapidly in white men and white women, largely in response to changes in tobacco smoking in recent decades.

Again, we see no reason for optimism about overall progress during recent years. There is no reason to think that, on the whole, cancer is becoming any less common.

Survival Data

There are many divergent measures of case survival, just as there are for mortality and incidence. One can count a group of patients with cancer, then count the number who are alive at some specific time after diagnosis (e.g., 2, 5, or 10 years) and calculate the percentage surviving at that time. However, that mixes the lethal effects of cancer with deaths from unrelated causes. One might instead compute the percentage who are alive and appear to be free of cancer at five years, or exclude those who have died of causes other than cancer during the period, or try to calculate the lifetime probability that someone with cancer will eventually die of it. A common device is to avoid difficult judgments about the presence of recurrent cancer or the cause of death and, instead, adjust for "expected" survival estimated from rates in the general population with the same age and sex distribution. The ratio of observed survival (cancer patients) to expected survival (general population), called the relative survival rate, is a commonly reported measure of case survival.[15]

Any of these survival measures can be applied to cancer overall, to specific forms of cancer, or to specific demographic groups of patients. Again we have many measures, with none of them clearly best. The difficulty in interpreting survival rates after cancer is illustrated by recent congressional testimony stating that the United States is on the verge of attaining a five-year survival rate of 50 percent. News stories did not always make it clear that the computation of such a high rate required exclusion of the nonwhite population and the use of relative rather than absolute survival rates.[16]

Problems in Interpreting Recent Incidence and Survival Data

Changing standards of diagnosis and medical care of patients with cancer may affect incidence and survival rates substantially more than they affect mortality rates. At one time, a cancer was a cancer, and it could be assumed that a truly malignant neoplasm would eventually appear in hospital records (for treatment) or in death records (if treatment was unsuccessful or not attempted). The major exception, most forms of superficial skin cancer, could be excluded from the registry system by definition (the biologic behavior of superficial skin cancer is unlike that of other neoplasms because metastatic spread, the main reason for death from cancer, is uncommon). Other neoplasms lacking metastatic behavior used to be considered infrequent and were not regarded as a source of serious bias in the interpretation of trends. That assumption can no longer be made. The implications are substantial.

The 1974 peak in the incidence of breast cancer (Fig. 4) corresponded to the occurrence of public disclosures that the wives of the U.S. President and Vice President had breast cancer and a major public effort to promote screening for the disease by mammography. Although the 1974 peak was well beyond the limits of random variation, there has been no apparent corresponding change in mortality from breast cancer (Fig. 2) or in case survival rates (Table 2). We believe that the 1974 peak in incidence is spurious and reflects the inclusion of a proportion of benign and borderline lesions that in other years would not have been detected and reported. That such shifts in diagnostic criteria do occur, and specifically for breast cancer, is well documented.[7,17,18] After the 1974 peak the

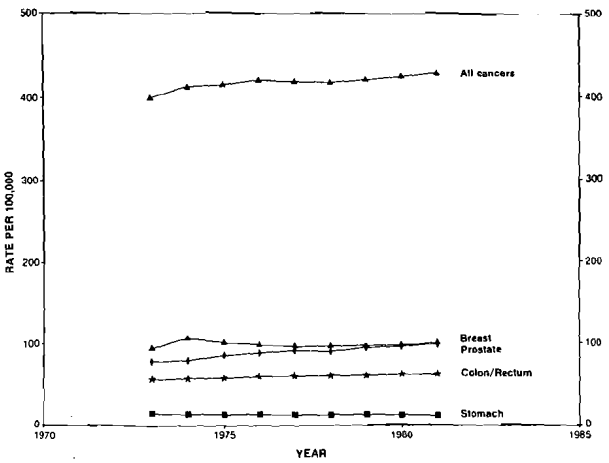

Figure 4. Incidence of All Cancers and Cancers of Selected Sites, 1973 through 1981, in the White Population of the SEER Registry Area.
Age was adjusted to the U.S. population of 1980.

rates plateaued at a lower level, then started to rise slowly but steadily among both white and nonwhite women. A recent resurgence of screening programs may account for some of this latest increase, but present data do not permit a definitive conclusion about whether it is artifactual or represents a true increase in incidence.

Cancer of the prostate is a common incidental finding when unselected tissue specimens of old men are examined, whether at autopsy (after death from another cause) or biopsy (at surgery for a benign condition). Reports of prevalence rates in the 25 percent range are not rare.[7] It is less widely recognized that such lesions, especially those found incidentally at prostate surgery, are commonly reported as cancer in the incidence statistics. There appear to be no data on what proportion of these prevalent prostatic "cancers" had shown evidence of malignant behavior. Incidence rates for this disease do not exceed 1.2 percent per year even in the oldest age groups, including some proportion of patients with incidental diagnoses; the incidence of clinically apparent prostatic cancer must be lower, and mortality rates are lower still. We must conclude that the prevalence rates are seriously inaccurate and that most of the tumors found, which do have the microscopical appearance of malignancy, do not have the behavior we associate with the word "cancer." Such an interpretation, combined with an increasing frequency of incidental tissue diagnosis, would be consistent with the rapid changes in survival after prostatic cancer shown in Table 2.

Lung cancer has increased rapidly in all major population segments. As a result, several kinds of screening programs have been developed and tested. Findings tend to be that in comparison to a randomized control group, the screened group has more cancers detected, the cancers are in earlier stages, more are considered suitable for curative treatment, and case survival rates are substantially higher. However, overall mortality is little affected.[19-21] This again seems to be a result of detecting and reporting lesions that have the microscopical appearance of cancer but not its biologic behavior. As a result of adding these benign conditions, the pool of real "cancers" is diluted, and we find high detection rates, early stage, resectability, and improved case survival, but with little or no change in outcome as measured by deaths.

Thus, the incidence and case survival data for three major forms of cancer may not mean what they at first suggest. Because of these uncertainties about the current meaning of "cancer" of the breast, prostate, and lung, neither incidence rates nor case survival rates for these diseases can be taken as reliable indicators of change in the overall progress against cancer. One must wonder whether similar problems affect the data on other forms of cancer. Mortality data do, in contrast, measure biologic behavior rather directly. That is mainly why we believe that mortality rates, age-adjusted to a current standard, are the best single measure of overall progress. Specifically, we disagree with the decision of the National Cancer Institute to emphasize survival (and the short-range goal of a five-year overall relative case survival rate of 50 percent), because it is subject to substantial bias from changing standards of diagnosis and reporting. A reported survival rate of 50 percent, if many of the patients do not have the biologic disease in question, would only mislead and confuse the public, the news media, governmental representatives, and health professionals who are not sophisticated in biostatistical and epidemiologic analysis.

Enstrom and Austin[22] have also discussed the problems of interpreting cancer survival rates. Although this matter needs further study, the uncertainties are great enough to make case survival an inappropriate measure of progress.

Colleagues have argued that the overall picture of cancer mortality is dominated by rising rates of death from lung cancer and that this disease should therefore be omitted from any summary measure of progress against cancer. Reasons for such an omission have not been clearly stated, although it conveniently reverses the overall rise in mortality from cancer. Lung cancer is in fact the best illustration of our primary conclusion that despite great effort over many years, research on cancer treatment has failed to deal effectively with

Table 2. Absolute and Relative Survival Rates.*

Five-Year Survival	Year of Diagnosis					
	1973	1974	1975	1976	1977	1978
	percent					
Absolute rate†						
All neoplasms	38.5	40.6	41.0	41.2	40.8	40.1
Colorectal cancer	36.2	37.8	38.2	39.6	39.4	38.7
Lung cancer	9.0	9.8	9.7	10.3	10.6	11.0
Breast cancer	64.3	65.2	67.0	66.0	66.0	65.0
Prostate cancer	41.2	43.9	45.9	47.7	47.8	47.4
Hodgkin's disease	57.7	63.3	66.6	71.9	69.5	67.3
Non-Hodgkin's disease	34.3	37.9	40.0	41.0	39.0	39.0
Relative rate†						
All neoplasms	46.8	49.3	50.0	50.3	50.0	49.1
Colorectal cancer	46.4	48.8	49.4	51.1	51.3	50.3
Lung cancer	11.0	11.9	11.8	12.5	13.0	13.4
Breast cancer	72.3	73.6	75.8	74.7	75.1	74.1
Prostate cancer	60.7	65.0	67.6	70.2	69.9	69.4
Hodgkin's disease	61.5	67.6	71.0	76.5	74.3	72.0
Non-Hodgkin's disease	40.9	45.1	47.3	48.6	46.8	47.1

*Source: Unpublished data from the SEER Program, National Cancer Institute.

†Rates are for white males and white females only, relative to survival of the U.S. white population with the same age distribution.

the cancer problem. We have nevertheless calculated age-adjusted mortality rates excluding lung cancer; with this exclusion the change in overall age-adjusted mortality from cancer since 1950 shifts from an 8 percent increase to a 13 percent decrease. If one also excludes cancer of the stomach and cervix, whose rates have also been changing for reasons largely unrelated to treatment (Fig. 5), age-adjusted mortality shifts from 130.1 in 1950 to 128.9 in 1980 — a change of less than 1 percent. It is difficult to claim success in the war against cancer on the basis of these figures.

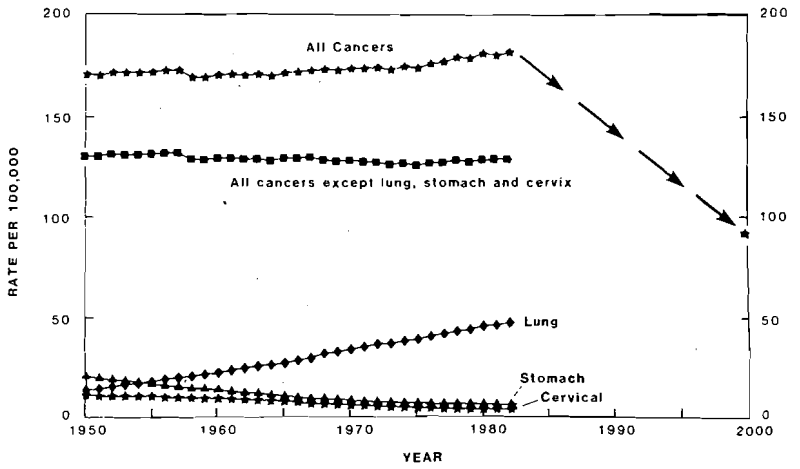

Figure 5. Mortality from Cancer of All Sites and Selected Sites, 1950 through 1982, in the U.S. Population.

Age was adjusted to the U.S. population of 1980. Extension to the year 2000 is shown to reflect the stated goal of the National Cancer Institute.

In Figure 5 the time scale is extended to the year 2000. We have marked on the figure the National Cancer Institute goal of a 50 percent reduction in mortality by that year. It is clear that the goal will not be attained unless the present upward trend is reversed very soon and there is a precipitous and unprecedented decline. We do not believe that hopes for such a change are realistic.

Conclusions

Some measures of efforts to control cancer appear to show substantial progress, some show substantial losses, and some show little change. By making deliberate choices among these measures, one can convey any impression from overwhelming success against cancer to disaster.

Our choice for the single best measure of progress against cancer is the mortality rate for all forms of cancer combined, age-adjusted to the U.S. 1980 standard. This measure removes the effects of changes in the size and age composition of the population, prevents the selective reporting of data to support particular views, minimizes the effects of changes in diagnostic criteria related to recent advances in screening and detection, and directly measures the outcome of greatest concern — death. The National Cancer Institute has also adopted this standard for its prospective goal of halving cancer mortality by the year 2000, but continues to use relative case survival rates to assess progress in years past.[3,16]

Age-adjusted mortality rates have shown a slow and steady increase over several decades, and there is no evidence of a recent downward trend. In this clinical sense we are losing the war against cancer. Substantial increases in our understanding of the nature and properties of cancer have not led to a corresponding reduction in incidence or mortality. On the basis of the age-adjusted trends that we have presented, it is unlikely that the National Cancer Institute will attain its stated goal of reducing age-adjusted mortality from cancer by 50 percent by the year 2000 — just 14 years from now.

These comments about lack of progress are in no way an argument against the earliest possible diagnosis and the best possible treatment of cancer. The problem is the lack of any substantial recent improvement in treating the most common forms.

Cairns[23] has also discussed the results of the effort to develop cures for cancer. His approach is largely clinical and biologic; ours is largely epidemiologic and statistical, yet we come to similar conclusions about the poor rate of success to date and the need to reconsider present directions in both research and applications. His paper should be read in conjunction with ours for a more comprehensive view of the matter.

The main conclusion we draw is that some 35 years of intense effort focused largely on improving treatment must be judged a qualified failure. Results have not been what they were intended and expected to be. We think that there could be much current value in a comprehensive, consolidated, objective review of the technical reasons for this failure. What forces led to overlapping waves of interest and program emphasis, such as chemotherapy screening, virology, immunology, and perhaps now molecular biology, that have appeared to hold more promise than they have fulfilled? Why were hopes so high, what went wrong, and can future efforts be built on more realistic expectations? Why is cancer the only major cause of death for which age-adjusted mortality rates are still increasing?[24]

A full analysis of current program plans and directions would require substantial expertise, time, and support. On the basis of past medical experience with infectious and other nonmalignant diseases, however, we suspect that the most promising areas are in cancer prevention rather than treatment. Although no one can be certain about the benefits of preventive efforts, history suggests that savings in both lives and dollars could be great. For example, opinions that attempts to prevent smoking have been discouraging are wrong. In scarcely 20 years of half-hearted effort, this country

has reversed historic trends in smoking and altered its casual tolerance of smokers. Societal antismoking norms have changed, and those who use tobacco are now on the defensive. Research opportunities in other areas of cancer prevention may well merit sharp increases in support, even if this requires that current treatment-related research must be substantially curtailed. Certainly, the background of past disappointments must be dealt with in an objective, straightforward, and comprehensive manner before we go much further in pursuit of the cure that always seems just out of reach.[24]

We are indebted to Jesse Berlin and Julia Bailey for help in processing the data presented here, to Dr. Michael Shimkin for encouragement, and to many colleagues for helpful comments on earlier drafts.

References

1. McKay FW, Hanson MR, Miller RW. Cancer mortality in the United States: 1950-1977. Bethesda, Md.: National Cancer Institute, 1982. (National Cancer Institute monograph no. 59). (NIH publication no. 82-2435).
2. Vital statistics in the United States. Vol. 2. Mortality, Part B. Annual. Rockville, Md.: National Center for Health Statistics, 1950-82.
3. Division of Cancer Prevention and Control. Cancer control: objectives for the nation. 1985-2000. Monograph Series (in press).
4. Hill AB. Principles of medical statistics. 9th ed. New York: Oxford University Press, 1971.
5. Armitage P. Statistical methods in medical research. New York: John Wiley, 1971.
6. Faust NM, Dollman AB. Comparability of mortality statistics for the sixth and seventh revisions, United States, 1958. (Vital Statistics special report 51(4)). Rockville, Md.: National Center for Health Statistics, March 1965.
7. Klebba AJ, Dollman AB. Comparability of mortality statistics for the seventh and eighth revisions of the International Classification of Diseases, United States. (Vital and Health Statistics 2(66)). Rockville, Md.: National Center for Health Statistics, October 1975.
8. Klebba AJ, Scott JH. Estimates of selected comparability ratios based on dual coding of 1976 death certificates by the eighth and ninth revisions of the International Classification of Diseases. (Vital Statistics report 28(11), Supplement). Hyattsville, Md.: National Center for Health Statistics, February 1980.
9. Doll R, Peto R. The causes of cancer: quantitative estimates of avoidable risks of cancer in the U.S. today. New York: Oxford University Press, 1981.
10. Davis DL, Lilienfeld AD, Gittelsohn A, et al. Increasing cancer in elderly Americans: fact or artifact? Am J Public Health (in press).
11. Kelsey JL, Hildreth NG. Breast and gynecologic cancer epidemiology. Boca Raton, Fla.: CRC Press, 1983.
12. Miller RW, McKay FW. Decline in the US childhood cancer mortality: 1950 through 1980. JAMA 1984; 251:1567-70.
13. Hammond D, Chard RL Jr, D'Angio GJ, et al. Pediatric malignancies. In: Hoogstraten B, ed. Cancer research: impact of the cooperative groups. New York: Masson, 1980:1-23.
14. Horm JW, Asire AJ, Young JL Jr, et al., eds. SEER Program: cancer incidence and mortality in the United States, 1973-81. Bethesda, Md.: Department of Health and Human Services, 1984. (NIH publication no. 85-1837).
15. Ederer F, Axtell LM, Cutler SJ. The relative survival rate: a statistical methodology. Bethesda, Md.: National Cancer Institute, 1961. (National Cancer Institute monograph no. 6).
16. DeVita VT. Cancer patient survival statistics. (NCI update). Bethesda, Md.: National Cancer Institute, November 26, 1984.
17. Thomas LB, Ackerman LV, McDivitt RW, Hanson TAS, Hankey BF, Prorok PC. Report of NCI ad hoc pathology working group to review the gross and microscopic findings of breast cancer cases in the HIP study (Health Insurance Plan of Greater New York). JNCI 1977; 59:495-541.
18. Beahrs OH, Shapiro S, Smart C. Detailed report of the working group's review of BCDDP. Section III. Pathology review of minimal breast cancers detected in BCDDP. JNCI 1979; 62:673-84.
19. Berlin NI, Buncher CR, Fontana RS, et al. The National Cancer Institute cooperative early lung cancer detection program: results of the initial screen (prevalence): introduction. Am Rev Respir Dis 1984; 130:545-9.
20. Early lung cancer detection: summary and conclusions. Am Rev Respir Dis 1984; 130:565-70.
21. Bailar JC III. Screening for lung cancer — where are we now? Am Rev Respir Dis 1984; 130:541-2.
22. Enstrom JE, Austin DF. Interpreting cancer survival rates. Science 1977; 195:847-51.
23. Cairns J. The treatment of diseases and the war against cancer. Sci Am 1985; 253(5):51-9.
24. Bailar JC III. The case for cancer prevention. JNCI 1979; 62:727-31.

of accelerated coronary artery disease in the transplanted heart.

Stanford University
School of Medicine
Stanford, CA 94305

JOHN SPEER SCHROEDER, M.D.
SHARON A. HUNT, M.D.

REFERENCES

1. Bailey LL, Nehlsen-Cannarella SL, Doroshow RW, et al. Cardiac allotransplantation in newborns as therapy for hypoplastic left heart syndrome. N Engl J Med 1986; 315:949-51.
2. Oyer PE, Stinson EB, Jamieson SW, et al. Cyclosporin-A in cardiac allografting: a preliminary experience. Transplant Proc 1983; 15:1247-52.
3. The Canadian Multicentre Transplant Study Group. A randomized clinical trial of cyclosporine in cadaveric renal transplantation: analysis at three years. N Engl J Med 1986; 314:1219-25.
4. European Multicentre Trial Group. Cyclosporine in cadaveric renal transplantation: one-year follow-up of a multicentre trial. Lancet 1983; 2:986-9.
5. Austen WG, Cosimi AB. Editorial retrospective: heart transplantation after 16 years. N Engl J Med 1984; 311:1436-8.
6. Solis E, Kaye MP. The Registry of the International Society of Heart Transplantation: third official report — June 1986. J Heart Transplant 1986; 5:2-5.
7. Mason JW, Stinson EB, Hunt SA, Schroeder JS, Rider AK. Infections after cardiac transplantation: relation to rejection therapy. Ann Intern Med 1976; 85:69-72.
8. Starzl TE, Porter KA, Iwatsuki S, et al. Reversibility of lymphomas and lymphoproliferative lesions developing under cyclosporine steroid therapy. Lancet 1984; 1:583-7.
9. Pope SE, Stinson EB, Daughters GT III, Schroeder JS, Ingels NB Jr, Alderman EL. Exercise response of the denervated heart in long-term cardiac transplant recipients. Am J Cardiol 1980; 46:213-8.
10. Clark DA, Schroeder JS, Griepp RB, et al. Cardiac transplantation in man: review of first three years' experience. Am J Med 1973; 54:563-76.
11. Stinson EB, Griepp RB, Schroeder JS, Dong E, Shumway NE. Hemodynamic observations one and two years after cardiac transplantation in man. Circulation 1972; 62:55-60.
12. Savin WM, Haskell WL, Schroeder JS, Stinson EB. Cardiorespiratory responses of cardiac transplant patients to graded, symptom-limited exercise. Circulation 1980; 62:55-60.

CORRESPONDENCE

Letters to the Editor are considered for publication (subject to editing and abridgment), provided that they are submitted in duplicate, signed by all authors, typewritten in double spacing, and do not exceed 40 typewritten lines of manuscript text (excluding references). Submission of a letter constitutes permission for the Massachusetts Medical Society, its licensees, and its assignees to use it in the *Journal*'s various editions (print, data base, and optical disk) in anthologies, revisions, and any other form or medium. Letters should not duplicate similar material being submitted or published elsewhere, and they should not contain abbreviations. Financial associations or other possible conflicts of interest should always be disclosed.

Letters referring to a recent *Journal* article must be received within six weeks of the article's publication. We are unable to provide pre-publication proofs, and unpublished material will not be returned to authors unless a stamped, self-addressed envelope is enclosed.

PROGRESS AGAINST CANCER?

To the Editor: The report by Bailar and Smith (May 8 issue)* certainly provoked a great deal of discussion among American oncologists. I think it is clear from the data they provided that mortality from lung cancer in the United States continues to be a serious health problem. Lung cancer is increasing in incidence and appears to be reaching epidemic proportions. I disagree with the authors' conclusion that wholehearted efforts to prevent lung cancer will substantially change the mortality figures. Incidence rates for lung cancer have been rising in both white men and white women. To date, there is nothing to suggest that the halfhearted efforts of government and society to modify smoking behavior will change incidence figures in the future. Rather, I believe that in addition to fruitless attempts to educate society about the evils of smoking, the most expedient way of changing smoking habits is to tax cigarettes more heavily. The revenues generated could be used to fund primary prevention studies, and there would also be a monetary incentive to stop smoking.

*Bailar JC III, Smith EM. Progress against cancer? N Engl J Med 1986; 314:1226-32.

JACOB D. BITRAN, M.D.
Chicago, IL 60616 Michael Reese Hospital and Medical Center

To the Editor: The pessimistic review of cancer-mortality statistics from 1950 to 1982 presented by Bailar and Smith does not reflect the lag between improvements in breast cancer treatment since 1975 and their effect. Since median survival after breast cancer (Stages I and IV) is at least seven years, the authors' conclusion that improved treatment must be judged a "qualified failure" may be invalid. Although adjuvant chemotherapy was initiated in the mid-1970s, it was not widely employed until the 1980s, and it is still not being recommended even for high-risk postmenopausal patients, according to the September 1985 National Institutes of Health (NIH) Consensus Conference.

A survey of the practice patterns of 634 oncologists by the Chemotherapy Foundation[1] in the summer of 1985 indicates that most oncologists use only the mild convenience regimen consisting of cyclophosphamide, methotrexate, and fluorouracil (CMF) for premenopausal women and less than 35 percent employ the more aggressive (Cooper-type) regimen consisting of cyclophosphamide, methotrexate, fluorouracil, vincristine, and prednisone or regimens containing doxorubicin. For postmenopausal cancer, a study begun in 1980 by Bonadonna et al.[2] in high-risk patients with Stage I breast cancer has recently shown that an intensified CMF regimen is highly effective in prolonging long-term survival. Bailar and Smith's data cannot indicate the effect of the potential curability of chemotherapy in breast cancer, even though it is now firmly established by several 8- to 19-year-old studies indicating increased disease-free survival of 12 to 27 percent.[3-5] Early perioperative and aggressive curative programs are virtually ignored by the profession. The current conservative trend has even extended to chemoprevention, since the NIH Consensus Conference failed to encourage the use of tamoxifen in Stage I postmenopausal breast cancer. The effect of tamoxifen for Stage II breast cancer, although its use has been established since 1979, has yet to be felt on American mortality statistics. I estimate that 10,000 lives could be saved by the early aggressive use of polychemotherapy in breast cancer, as compared with the negligible number of lives, perhaps several thousand, now being saved. At the recent meeting of the American Society of Clinical Oncologists, DeVita estimated[6] that 4000 patients with lymphoma annually are not achieving 10-year disease-free survival as a result of inadequate dosages of otherwise good treatment regimens.

Before condemning current treatment as futile, one needs to examine the extent to which improved treatments are actually being employed.

The paper by Bailar and Smith may perhaps rouse us from our lethargy. But, unfortunately, it is reminiscent of Bailar's study[7] a decade ago emphasizing the negative risk–benefit ratio involved in mammography, which was based on the use of antiquated radiologic equipment. What good are improved methods not optimally applied?

EZRA M. GREENSPAN, M.D.
New York, NY 10029 Mount Sinai Medical Center

1. Greenspan E, Goldsmith M, Silverman J. Pattern of care study of breast cancer adjuvant chemotherapy. Proc Am Soc Clin Oncol 1986; 5:73. abstract.
2. Bonadonna G, Zambetti M, Valagussa P, et al. Adjuvant CMF in node negative breast cancer. Proc Am Soc Clin Oncol 1986; 5:74. abstract.

3. Cooper RG. Combination chemotherapy of breast cancer. Mt Sinai J Med (NY) 1985; 52:443-6.
4. Rivkin SE, Glucksberg H, Foulkes M, et al. Adjuvant chemotherapy for operable breast cancer with positive axillary nodes: a comparison of CMFVP versus L-PAM. World J Surg 1985; 9:719-22.
5. Nissen-Meyer R, Host H, Kjellgren K, Mansson B, Norin T. Short perioperative versus long-term adjuvant chemotherapy. In: Metzger U, Largiadèr F, Senn H-J, eds. Perioperative chemotherapy: rationale, risk and results. Berlin: Springer-Verlag, 1985:91-8.
6. DeVita V. Rosenthal Memorial Lecture. Presented at the American Society of Clinical Oncology Meeting, Los Angeles, May 1986.
7. Bailar JC III. Mammography: a contrary view. Ann Intern Med 1976; 84: 77-84.

To the Editor: The article "Progress against Cancer?" does not aim at stimulating discussion of the progress of research in the United States. Rather, it attempts to summarize some 35 years of the cancer-research experience (including the most recent 15 years, since the National Cancer Institute launched its current intensive effort) in an elementary manner. It refers to only a single measure of progress. On this basis the article concludes that "we are losing the war against cancer." This is an erroneous view.

The article contains glaring weaknesses. For example, the use of the age-adjusted mortality rate as the sole measure of progress addresses only one of many dimensions of the accomplishments of the national cancer program and deserves to be questioned. Even if one examines treatment results to the exclusion of all other progress in the cancer program, the use of mortality data that measure old events is not a good assessment of the state of the art today. The intensive effort, referred to by Bailar and Smith as "the war against cancer," began with special funding in 1972. The mortality data cited for 1982 measure events in diagnosis and treatment for many cancers that occurred between 1972 and 1975 and sometimes earlier. Reports on curative therapies for some cancers and the definitive adjuvant-treatment studies in breast cancer, for example, were not even published at that time.

The article fails even to mention the types and magnitude of prevention research now in progress. And although we agree that prevention research merits increasing support, so too do research on treatment and basic research on the mechanisms that determine normal cell division and differentiation and on the failure of these mechanisms that leads to neoplasia. Basic research, research on prevention, and research on treatment need not be considered competitors and should not compete, given adequate levels of financial support. We need to stimulate all these areas of scientific inquiry, and current programming reflects those directions.

The progress of a scientific program of the scope and dimensions of the national cancer program cannot be assessed in a Special Article by two authors with a unidimensional approach and a limited knowledge of the extent of the research effort supported by the National Cancer Institute. It is a complex program constantly open to scrutiny, criticism, advice, and public debate. It is, however, a program based in science, and it deserves a valid scientific assessment. This article does not provide such an assessment.

VINCENT T. DEVITA, JR., M.D.
Bethesda, MD 20892 — National Cancer Institute

DAVID KORN, M.D.
Palo Alto, CA 94305 — Stanford University School of Medicine

To the Editor: The suggestion that the age-adjusted mortality rate is the appropriate standard for evaluating success in cancer treatment may be as erroneous as the more positive data reported by the National Cancer Institute. The use of death-certificate diagnosis as the basis for statistical studies is pragmatic but not reliable or precise enough to justify the negative conclusions of the paper. In the 25 years referred to in this article there have been many changes in cancer diagnostic techniques and in the attitudes toward cancer of physicians and patients that could have altered the reported incidence of cancer or mortality data.

Physicians who critically analyze the results of cancer treatment do not rely on data based on death certificates. We have learned how to assess the statistics of treatment results, such as those regularly reported in the *Journal*. Specific mortality and detailed morbidity evaluations are both important considerations in cancer treatment.

I have to reject the conclusions of Bailar and Smith that emphasis in cancer research should be switched from treatment to prevention. Certainly, prevention should be an important priority in cancer research and educational efforts. The inaccuracies of the data-collection technique used in this article do not justify our abandoning treatment-based research efforts. Indeed, "progress against several uncommon forms of the disease, improvements in palliation, and extension of the productive years of life"* argue in favor of the expansion of these efforts.

GARY A. RATKIN, M.D.
St. Louis, MO 63108 — American Society of Clinical Oncology

*Bailar JC III, Smith EM. Progress against cancer? N Engl J Med 1986; 314:1226-32.

To the Editor: Bailar and Smith noted an 8 percent increase in the age-adjusted cancer mortality rates since 1950 and concluded that there has been no progress against cancer in patients over 30 years of age. However, their use of summary age-adjusted rates to examine cancer mortality trends has obscured important age-specific differences. When the 1980 age-specific rates are compared with the corresponding 1950 rates[1] (Table 1, columns 1 through 4), marked decreases in cancer mortality are apparent in all five-year intervals under age 50, although the age categories over 50 show substantial increases. Bailar and Smith claimed further support for their conclusion when, after excluding deaths from lung, stomach, and cervical cancer in order to remove the effects of cancers whose rates have changed for reasons unrelated to treatment, they found a trivial decrease of less than 1 percent. However, inspection of the deflated age-specific rates (Table 1, columns 5 through 7) reveals a material decline in cancer mortality in all age groups under 65. Rate increases persist in the age categories over 65. The comparison of the 1980 and 1950 age-specific rates reveals a classic crossover phenomenon, a severe type of interaction that precludes the use of age adjustment as a valid means of summarizing data.[2] Changes in the age-specific cancer mortality rates since 1950 suggest that progress may in fact have been achieved in the treatment of cancer patients under the age of 65.

Table 1. Age-Specific Cancer Mortality Rates for 1950 and 1980.*

AGE GROUP	ALL CANCER MORTALITY			ALL CANCER MORTALITY EXCEPT LUNG, STOMACH, AND CERVIX		
	1950 RATES	1980 RATES	PERCENT CHANGE	1950 RATES	1980 RATES	PERCENT CHANGE
30–34	25.1	17.3	−31.1	19.9	14.9	−25.1
35–39	45.6	33.5	−26.5	35.1	26.4	−24.8
40–44	80.9	66.7	−17.6	60.6	47.7	−21.3
45–49	136.5	128.3	−6.0	99.6	84.9	−14.8
50–54	216.3	228.9	+5.8	157.4	148.1	−5.9
55–59	328.7	358.1	+8.9	239.0	230.8	−3.4
60–64	467.4	525.8	+12.5	341.8	336.7	−1.5
65–69	597.6	722.7	+20.9	445.0	475.5	+6.9
70–74	828.3	940.9	+13.6	631.5	646.7	+2.4
75–79	1065.4	1142.6	+7.3	823.9	835.8	+1.4
80–84	1321.4	1378.7	+4.3	1047.9	1086.0	+3.6
>85	1451.0	1594.6	+9.9	1176.1	1326.3	+12.8

*Rates are per 100,000 person-years.

Before one accepts the concomitant interpretation that treatment efforts over the past 35 years have actually hastened rather than retarded cancer mortality in the older age groups, two alternative hypotheses deserve consideration. First, the apparent increase in cancer mortality in the older age groups, even after the deletion of deaths from lung, stomach, and cervical cancer, may be due to the inclusion of deaths from other cancers attributable to tobacco use. Epidemiologic studies have shown that cancers of the larynx, esophagus, oral cavity, bladder, kidney, and pancreas are also associated with cigarette smoking.[3] Second, the 1950 cancer mortality rates may have been underestimated in the older age strata. The accuracy of the primary cause of death as stated on the death certificate has certainly improved since 1950.[4,5] That the most dramatic improvement has occurred in the older age groups is suggested by reductions in the age-specific mortality rates for deaths due to senility and other ill-defined conditions,[1] which more than offset the increases in cancer mortality (data available on request). Explanations for improvements in the accuracy of assigning cause of death include the expansion of elderly people's access to health care through Medicare, as well as advances in physicians' diagnostic acumen. The alternative hypotheses we have proposed are not mutually exclusive, and we suspect that both factors have contributed to the peculiar age-dependent trends in cancer mortality since 1950.

DEBORAH J. DEL JUNCO
JOHN F. ANNEGERS
University of Texas
Houston, TX 77225 School of Public Health

1. Vital Statistics in the United States. Vol. 2. (Mortality, Parts A and B.) Rockville, Md.: National Center for Health Statistics, 1950 and 1980.
2. Fleiss JL. Statistical methods for rates and proportions. 2nd ed. New York: John Wiley, 1981:246-7.
3. Wynder EL, Hoffman D. Tobacco. In: Schottenfeld D, Fraumeni JF Jr, eds. Cancer epidemiology and prevention. Philadelphia: WB Saunders, 1982: 277-92.
4. Kircher T, Nelson J, Burdo H. The autopsy as a measure of accuracy of the death certificate. N Engl J Med 1985; 313:1263-9.
5. Bauer FW, Robbins SL. An autopsy study of cancer patients. I. Accuracy of the clinical diagnoses (1955 to 1965) Boston City Hospital. JAMA 1972; 221:1471-4.

To the Editor: Bailar and Smith have done a public service with their thoughtful analysis of cancer mortality in the United States.

Many scientific advances with practical applications have been achieved with the use of funds allocated to cancer research. Monoclonal antibodies serve well as a recent example. The total mortality from cancer, however, has not been reduced; it has climbed slowly but steadily. The reduction of cancer mortality among younger patients is an important advance, but it applies to about 10 percent of the total cancer burden of the population. The big killers — cancers of the lung, colorectum, breast, and prostate — are more resistant.

The Bailar–Smith analysis should not be considered a criticism of cancer research, although it questions its allocations. And its implications do militate against the promotion of cancer research through public relations based on wishful thinking or worse.

Bailar and Smith put their confidence in the ability of physicians and the general public to reach correct conclusions when they are confronted with facts that are uncomfortable. Congratulations.

MICHAEL B. SHIMKIN, M.D.
La Jolla, CA 92093 University of California, San Diego

To the Editor: The lack of progress against cancer, as described by Bailar and Smith, is not confined to the United States alone. In 1985 the World Health Organization (WHO) reported its study of cancer-mortality trends covering the period 1960 to 1980 in 28 countries, including the United States, representing 75 percent of the population of the developed world.[1,2] The conclusions were essentially the same and are summarized in Table 1.

The cancer that had the steepest rise in mortality and that was the dominant factor in the overall increase was lung cancer. It is primarily a self-induced, preventable cancer. Sadly, if there had been an active commitment to reduce tobacco consumption, this increase could have been avoided.

Table 1. Age-Adjusted Increase (or Decrease) in Cancer Mortality in 28 Developed Countries from 1960 to 1980.*

CANCER	MALES	FEMALES
	percent	
Lung	76	135
Stomach	(45)	(58)
Breast	—	22
Cervical	—	(30)
All cancers	19	(2)

*Parentheses indicate a decrease in mortality.

Stomach cancer is decreasing in nearly all countries, and although the specific reasons for this effect are unknown, the most plausible explanation appears to be changes in diet and food preparation and not improved therapy.

Cancer of the cervix uteri is virtually the only common tumor in which there has been a substantial decrease in mortality due primarily to action taken by the medical community. Reductions in mortality from cervical cancer were seen in countries that had clear screening policies and a well-organized cytologic screening system.

In the WHO analysis, the important role of preventive measures and the value of carefully designed screening programs were clear. The influence of therapeutic treatment on overall mortality for the common cancers was very limited, although there has been dramatic progress in therapy for some less common cancers.

Contrary to common belief, there are more cases of cancer in absolute numbers in the developing countries than in the industrialized countries. Although accurate time-trend information is not available from most developing countries, sufficient data exist to permit the conclusion that the relative increase in cancer has occurred at a faster rate than in developed countries.[3,4] The primary reasons for this are an increase in life expectancy and a dramatic increase in the consumption of tobacco. For example, in Shanghai County, an area near the city of Shanghai in China, cancer was the sixth leading cause of death in 1960. By 1980 cancer had become the leading cause of death.[5]

In accordance with these and other findings, WHO advises member states to plan national cancer-control programs with the primary focus on prevention — especially education and legislation concerning tobacco — early detection, and treatment of the disease and on the provision of pain relief.[6]

The need to establish new priorities and strategies in cancer comes not only from analyses such as the one outlined above, but also from cost-effectiveness investigations that clearly point to the value of preventive programs, especially regarding tobacco control.[7] Experience in the Scandinavian countries (tobacco smoking) and in India (tobacco chewing) has shown that life styles can be changed in a positive fashion.[8] To win the war against cancer we must also win the battle against tobacco.

JAN STJERNSWÄRD, M.D.
KENNETH E. STANLEY, PH.D.
HARALD HANSLUWKA, PH.D.
ALAN D. LOPEZ, PH.D.
1211 Geneva 27, Switzerland World Health Organization

1. Cancer increases in developed countries. WHO Wkly Epidemiol Rec 1985; 17:125-9.
2. Cancer in developed countries: assessing the trends. WHO Chron 1985; 39:109-11.
3. Cancer as a global problem. WHO Wkly Epidemiol Rec 1984; 59:125-6.
4. Parkin DM, Stjernswärd J, Muir CS. Estimates of the worldwide frequency of twelve major cancers. Bull WHO 1984; 62:163-82.
5. Gu XY, Chen ML. Health services in Shanghai County: vital statistics. Am J Public Health 1982; 72:Suppl 9:19-23.

6. Stjernswärd J, Stanley K, Eddy DM, et al. Cancer control: strategies and priorities. World Health Forum 1985; 6(2):160-4.
7. Eddy DM. Setting priorities for cancer control programs. JNCI 1986; 76: 187-99.
8. Control of oral cancer in developing countries: a WHO meeting. Bull WHO 1984; 62:817-30.

To the Editor: Although I agree with Bailar and Smith that some success claims may be exaggerated and some future goals unrealistic and I support increased efforts to prevent cancer, I believe that their article presents an unfairly bleak picture of the effort against cancer.

The inevitability of death should, one feels intuitively, create some linkage among death rates for various diseases. From 1964 to 1979, death rates (age-adjusted to the 1970 population) for heart disease and stroke (together, nearly 50 percent of all deaths) fell 25.9 and 42.2 percent, respectively, while cancer mortality increased 6.9 percent.[1] Perhaps falling cardiovascular mortality has "unmasked" cancer mortality, and persons who formerly died with cancer now die of cancer. In this context, a small percentage increase in cancer mortality may well represent progress.

Inevitable mortality also makes it unrealistic to expect the prevention of eventual death; at best, only premature death can be prevented or reduced. From this perspective, progress has been made against cancer: Age-specific cancer mortality decreased from 1964 to 1979 for all 5-year age groups below the age of 50 (Table 1).[1] Although such deaths represent a small fraction of total cancer mortality, the decrease represents an important reduction in the number of premature deaths.

Table 1. 1979 Age-Specific Mortality Rates (per 100,000) for All Cancers in the Total Population and Percentage Changes since 1964.*

Age Group	Rate	Change	Age Group	Rate	Change
0–4	4.5	−47.1	45–49	129.0	−3.8
5–9	4.8	−32.4	50–54	231.2	+2.5
10–14	4.2	−30.0	55–59	355.4	+5.8
15–19	5.5	−28.6	60–64	539.3	+13.0
20–24	7.0	−23.9	65–69	708.5	+6.3
25–29	10.4	−26.2	70–74	928.7	+12.9
30–34	17.4	−29.6	75–79	1227.1	+21.5
35–39	33.0	−23.4	80–84	1414.2	+16.3
40–44	66.5	−15.1	>85	1434.7	−2.2

*Data from *Vital Statistics in the United States.*[1]

The data excluding lung cancer (Table 2)[2] suggest that, except for nonwhite males, cancer mortality among nonsmokers decreased or remained constant from 1962 to 1977. The cervical-cancer data and the data on rectal as compared with nonrectal colon cancer illustrate the benefits of treatment when early detection is possible. The differences in changes in mortality according to race and sex for total cancer and nonrectal colon cancer suggest that some of the limitation of progress against cancer is due to failure to apply knowledge, not failure to develop or discover it.

Although Bailar and Smith imply that emphasis on prevention will yield large results, more cautious expectations are in order. If, as the authors state, lung cancer is the best example of the failure of treatment, it is also the best example of the failure of prevention (especially among women). We have known for 22 years how to prevent this disease. Just as treatment has had limited success because of the failure to discover a "cancercillin," so too gains in prevention may be limited in the absence of a "tumovax." In such a case, prevention will require the compliance of an asymptomatic population, always difficult to achieve. A balanced approach is needed: research on prevention and on new methods of cure, and — what is most likely to yield rapid results — improved implementation of current knowledge and research on improved early detection. The two last-mentioned capitalize on the limited but real progress in cancer treatment.

Table 2. 1977 Age-Adjusted Mortality Rates (per 100,000, Adjusted to the 1970 Population) for Selected Cancers, According to Race and Sex, and Percentage Changes since 1962.*

Site	White Female	Nonwhite Female	White Male	Nonwhite Male
	rate (% change)			
All	133.0 (+0.3)	148.1 (+5.0)	207.8 (+13.6)	258.2 (+32.3)
Lung	17.7 (+179.9)	17.1 (+187.9)	67.6 (+61.4)	80.9 (+100.2)
All except lung	115.3 (−8.7)	131.0 (−3.1)	140.2 (−0.6)	177.3 (+14.5)
Cervix	3.5 (−53.4)	9.6 (−47.7)	—	—
Colon (not rectum)	16.4 (−5.4)	16.4 (+28.1)	20.7 (+18.3)	18.5 (+41.8)
Rectum	3.0 (−39.5)	3.0 (−34.5)	5.2 (−34.6)	5.5 (+3.0)

*Data from McKay et al.[2]

David A. Mann, M.D.
10 Colonial Village Dr.
Arlington, MA 02174

1. Vital statistics in the United States. Vol. 2 (Mortality, Part B.) Rockville, Md.: National Center for Health Statistics, 1979 and 1964.
2. McKay FW, Hanson MR, Miller RW. Cancer mortality in the United States: 1950–1977. Bethesda, Md.: National Cancer Institute, 1982. (NIH publication no. 82-2435.)

To the Editor: I have the good fortune to belong to the class of oncologists that is dealing with a subset of cancer wherein success is real; in pediatrics cure is the norm. That claim can be demonstrated by the measure that Bailar and Smith advocate — the decline in mortality from cancer in children in the general population.* The results achieved by pediatric oncologists represent a major contribution to the decrease of all cancers in patients under the age of 30 years, as acknowledged by Bailar and Smith. Even in pediatrics, however, the very success rate makes the current approach to cure through large-scale clinical trials less effective. Furthermore, the victory is often a Pyrrhic one, since the threat of second malignant neoplasms is ever increasing, and serious physical, developmental, and psychosocial iatrogenic sequelae are inevitable.

Prevention is always better than treatment of established disease, though physicians can treat quite effectively, both empirically and symptomatically. Whatever success the war on cancer has had has been achieved through objectively verified empiricism. Prevention cannot be approached as readily that way. One has to understand in order to prevent. A great deal of research on prevention is in fact research on causation. Much prevention can be accomplished when cause and effect are understood, as in the case of lung cancer and smoking. Preventive efforts without an understanding of cancer's causes are either too broad or miss the mark entirely; as a result they are costly and often ineffective.

Large-scale funding of research on therapy has not been a uniform failure, although the areas of success contribute only a small percentage to the improvement of treatment of all cancers. However, it either has failed or has become less promising in every area of success achieved. A rethinking of funding priorities is indeed in order. I strongly urge the redistribution of funds to favor once again fundamental research into the malignant process. In that way we will be able to prevent an understandable disease.

Jan van Eys, Ph.D., M.D.
The University of Texas
M.D. Anderson Hospital and
Houston, TX 77030 Tumor Institute at Houston

*Miller RW, McKay FW. Decline in US childhood cancer mortality: 1950 through 1980. JAMA 1984; 251:1567-70.

To the Editor: As a medical oncologist familiar with the barrenness and futility of so many published studies of cancer treatment (including some conducted by national cooperative groups), I found much with which I could agree in Bailar and Smith's article. Despite the undoubted successes of chemotherapy in the treatment of particular tumors, such as lymphomas and breast cancer, the claim "We are winning" is hardly justified by the facts. The source of this inappropriate emphasis on treatment seems to me to be the American health care community's excessively pragmatic stress on curing disease whose pathogenesis is still not understood. But shifting the emphasis to prevention, as these authors propose, reflects the same ill-directed pragmatism. Aside from lung cancer, how many neoplastic diseases can be considered preventable on the basis of present knowledge?

The authors are also in error, I believe, in equating recent progress in the elucidation of the molecular genetics of cancer (i.e., oncogenes) with previous fads in this field. This work has given us the first specific data on the biochemical abnormalities of neoplastic cells. The national approach to cancer should be redirected not to yet another blind, empirical enterprise, however popular, but to basic research, which alone holds out the hope of real progress.

ALBERT S. BRAVERMAN, M.D.
Downstate Medical Center–
Brooklyn, NY 11203 State University of New York

To the Editor: Recently, Bailar and Smith[1] and DeVita[2] faced off on the progress we have made in the war against cancer. In their article in the *Journal,* Bailar and Smith stated that mortality figures indicate we have not made progress, whereas DeVita, current director of the National Cancer Institute (NCI), pointed to the success of clinical research in advancing survival.[2] This could be a case of "where you sit is where you stand."

Perhaps they both have their facts right but not their interpretations. There are several obstacles to preventive activities, to the rapid dissemination of research results, and to the use of appropriate state-of-the-art treatment. The U.S. government is a major culprit in this problem, with inappropriate reimbursement policies and the lack of a coordinated NCI program to communicate information to primary care physicians.

Reimbursement for preventive activities is appalling. Breast mammography, Hemoccult tests, and the Pap test are not regularly reimbursed. Federally funded research may have proved their efficacy, but no one told the reimbursers. Reimbursement by third-party insurers and the diagnosis-related-group (DRG) payment program are altering the patterns of treatment, pushing chemotherapy treatment out of the hospital, for example, and establishing serious barriers to the treatment of adult patients with leukemia. Recently, the Prospective Payment Assessment Commission recommended that the adult leukemia DRGs be recalculated and increased on the basis of information provided by the Association of Community Cancer Centers, but action has yet to be taken. New forms of therapy are also facing an uphill battle for reimbursement, despite their efficacy and cost effectiveness. For example, VP-16 for small-cell lung cancer has been denied by Blue Cross–Blue Shield in some parts of the country, since the research on which its use is based is still considered "experimental" (e.g., not listed on the package insert).

At NCI over the past decade, only four programs have allocated any money for "technology transfer," most aimed at tertiary care community hospitals. Since a great many decisions in cancer therapy are made by primary care physicians in rural areas and since "the first decision in cancer management most often determines whether the outcome will be successful,"[3] this inattention to ensuring that physicians are aware of the importance of prevention and of diagnostic and staging procedures is a serious concern.

We spend 10 percent of the gross national product on health care bills, billions for important basic and clinical research, 25 percent of the current NCI budget for research on prevention, and less than 2 percent of the NCI budget to get the word out to the people who manage 85 percent of the care. As if to emphasize this lack of interest in the providers of care, the recent appointments to the National Cancer Advisory Board completely ignored the need for community-physician representation. Is it so surprising that we are not affecting the bottom line?

WILLIAM M. DUGAN, JR., M.D.
Indianapolis, IN 46202 Methodist Hospital of Indiana

LEE E. MORTENSON, M.S., M.P.A.
Association of Community
Rockville, MD 20852 Cancer Centers

1. Bailar JC III, Smith EM. Progress against cancer? N Engl J Med 1986; 314:1226-32.
2. Bailar NEJM article "irresponsible, purposefully misleading," ignored facts, DeVita tells NCAB. The Cancer Letter. May 23, 1986:1-2.
3. Rubin P. Clinical oncology: a multidisciplinary approach. 6th ed. Rochester, N.Y.: American Cancer Society, 1983:vi.

To the Editor: Bailar and Smith concluded from total mortality data that we are losing ground against cancer. Their method may be dominated by the numerous deaths at advanced ages, thereby masking progress in terms of prolonged survival of patients or of delayed onset or prevention of incident cases. Therefore, a complementary approach, free of these problems, might employ years of potential life lost (YPLL) before age 65.[1] This summary measure highlights the leading causes of death among younger persons (Wise RP, et al.: unpublished data).

Application of this YPLL measure to mortality from all causes between 1979 and 1984, with use of the 10 percent current mortality sample from the National Center for Health Statistics, revealed a 12.8 percent decrease in the rate of YPLL per 1000 persons under the age of 65. This reduction in mortality at younger ages was broadly based, with decreases in the YPLL rate for 10 of the 12 leading causes during this period (Fig. 1).

The decrease in the YPLL rate for cancer was 4.1 percent. However, the YPLL rate for cancers other than lung cancer fell faster, decreasing 6.2 percent during this interval. Most of this progress was due to the reduced death rate from cancer (excluding lung cancer) in 45-to-54-year-olds, which decreased from 126.7 to 117.6 per 100,000 persons. In striking contrast, the lung cancer mortality rate in the group 55 to 64 years old increased (146.5 to 156.1 per 100,000), thus reducing the overall improvement for cancer. Bailar and Smith's emphasis on tobacco's role in causing increased cancer is clearly appropriate.

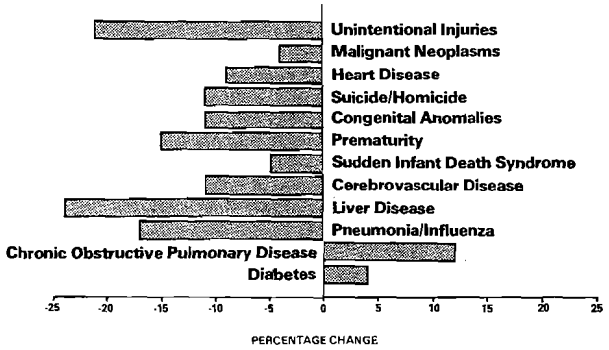

Figure 1. Percentage Change in the YPLL Rate for the Ranked Leading Causes of Premature Mortality in the United States, 1979 to 1984.

The percentage change is calculated as (1984 rate − 1979 rate) × 100/1979 rate. The YPLL rate is the number of years of potential life lost before age 65 per 1000 persons under age 65. Causes of mortality are defined and ranked in conformity with data from *Morbidity and Mortality Weekly Report.*[2] (Source, Centers for Disease Control: unpublished data.)

These data are consistent with a general trend among Americans to live longer that is due to progress (defined in terms of reduced mortality before age 65) against a wide array of diseases, not including chronic obstructive pulmonary disease and diabetes mellitus. As deaths from causes that compete with cancer decrease, more people are at risk of death from malignant conditions. With the reduction of mortality from cardiovascular disease, for example,[3] a larger proportion of deaths at older ages may now be due to cancer.

In addition, some increment in cancer death rates at older ages could also be due to prolonged survival of patients with cancer, which must also be credited as "progress against cancer." Again, the YPLL method would be more likely to reflect such progress than total mortality.

In summary, we suggest that the public health struggle against cancer should also be evaluated in terms of lengthened survival.

The views expressed in this letter do not necessarily reflect any policy or official position of the Food and Drug Administration.

R.P. Wise, M.D., M.P.H.
Center for Drugs and Biologics
Rockville, MD 20857 Food and Drug Administration

1. Changes in premature mortality — United States, 1983-1984. MMWR 1986; 35:29-31.
2. Estimated years of potential life lost before age 65 and cause-specific mortality, by cause of death — United States, 1984. MMWR 1986; 35:365.
3. Thom TJ, Epstein FH, Feldman JJ, Leaverton PE. Trends in total mortality and morbidity from heart disease in 26 countries from 1950 to 1978. Int J Epidemiol 1985; 14:510-20.

The above letters were referred to the authors of the article in question, who offer the following reply:

To the Editor: We welcome this interest in the accomplishments and future course of cancer research. We can comment here on only four points raised by these letters.

A cancer-research program that does not reduce overall death rates is not successful, whatever its other accomplishments. Thus, mortality data are critical, despite short delays from diagnosis to death and further short delays in publication. Cancer research was a major activity long before the National Cancer Act of 1971, and most cancer deaths occur within two to three years after diagnosis. (The main exception, breast cancer, accounts for only 9 percent of all cancer deaths.) Major improvements in survival by 1981 should be clearly visible in the 1983 data now available; they are not there.

If the evaluation of progress against cancer should not reflect our failure to contain the rise in deaths from lung cancer, neither should it reflect the fall in cancer of the stomach, cervix, and perhaps other organs for which recent trends are largely unrelated to treatment. We know of no good argument for any omissions at all, and certainly not for lung cancer alone.

Two letters point to percentage drops at younger ages that are larger than percentage rises at older ages, but the pattern is reversed for absolute risks. For the evaluation of progress, a fall from 25.1 per 100,000 to 17.3 (the 31.1 percent decrease at ages 30 through 34) is fundamentally much smaller than a rise from 597.6 to 722.7 (the 20.9 percent increase at ages 65 through 69), even after weighting for population size.

Should progress be measured according to years of life lost before age 65? To do so implies that some lives are worth less than others and that some should not be counted at all. Evaluation should not be based on years of life lost without full public understanding and acceptance of its implications.

The ugly fact remains that overall cancer mortality is rising; if one omits cancer of the lung, stomach, and cervix, it has been essentially unchanged for some 35 years. This cannot be explained away as a statistical artifact, obscured by the clear evidence of progress here and there, or submerged by rosy rhetoric about research results still in the pipeline. And it cannot be ignored.

We repeat our call for a full inquiry into the past, present, and future course of cancer research.

John C. Bailar III
Boston, MA 02115 Harvard School of Public Health

Elaine M. Smith
Iowa City, IA 52242 University of Iowa Medical Center

MORE ON OPEN-CHEST CARDIAC MASSAGE AFTER CARDIAC ARREST

To the Editor: The results of the prospective clinical trial of open-chest cardiopulmonary resuscitation (CPR) reported by Geehr and his associates (May 1 issue)[1] are important, but predictable from data on animals.[1-3] Previously published clinical and animal studies have shown that open-chest CPR provides better hemodynamics than closed-chest compression.[2-5] It remains uncertain, however, whether improvements in hemodynamics result in improved resuscitation and long-term survival. We approached this question in an animal model of cardiac arrest and found that after 15 minutes of closed-chest massage, open-chest CPR improved resuscitation, as compared with continued efforts by closed-chest techniques.[2] However, there was no improvement in resuscitation by open-chest CPR after 25 minutes of closed-chest massage, in spite of improved hemodynamics with open-chest massage.[3] The clinical implications of our study are that open-chest resuscitation will improve survival only if it is begun within 15 to 20 minutes of the onset of cardiac arrest, but will not improve survival if begun after 25 minutes.[3] Geehr et al. have confirmed this latter finding in humans.[1] Further clinical studies are needed to determine if the earlier application of open-chest massage will improve survival.

We believe that open-chest CPR will have a limited but definite role in the treatment of patients in cardiac arrest. Of all the proposed alterations of the standard technique of CPR investigated by our group and others, open-chest CPR results in the most dramatic hemodynamic improvement. Despite this, three important questions remain: Are there newer techniques of closed-chest CPR that are more effective than standard CPR? What feaures can tell us whether continued efforts with closed-chest CPR will be ineffective? Will open-chest cardiac massage, when applied early (within 15 minutes of arrest), improve resuscitation and result in improved long-term survival without neurologic defects?

We agree with Geehr and his associates that open-chest CPR is not appropriate as a "last-ditch" effort in patients with arrest times of 25 minutes or longer. Such use will only reinforce the negative impression that many physicians now have about open-chest CPR. The exact role of open-chest CPR needs to be determined by appropriate clinical trials firmly based on good experimental data.

Arthur B. Sanders, M.D., Karl B. Kern, M.D.,
Kenneth L. Raessler, M.D., Charles Otto, M.D.,
and Gordon A. Ewy, M.D.
University of Arizona
Tucson, AZ 85724 College of Medicine

1. Geehr EC, Lewis FR, Auerbach PS. Failure of open-heart massage to improve survival after prehospital nontraumatic cardiac arrest. N Engl J Med 1986; 314:1189-90.
2. Sanders AB, Kern KB, Ewy GA, Atlas M, Bailey L. Improved resuscitation from cardiac arrest with open-chest massage. Ann Emerg Med 1984; 13:672-5.
3. Sanders AB, Kern KB, Atlas M, Bragg S, Ewy GA. Importance of the duration of inadequate coronary perfusion pressure on resuscitation from cardiac arrest. J Am Coll Cardiol 1985; 6:113-8.
4. Del Guercio LRM, Feins NR, Cohn JD, Coomaraswamy RP, Wollman SB, State D. Comparison of blood flow during external and internal cardiac massage in man. Circulation 1965; 31:Suppl I:I-171–I-180.
5. Bircher N, Safar P. Comparison of standard and "new" closed-chest CPR and open-chest CPR in dogs. Crit Care Med 1981; 9:384-5.

To the Editor: We must be careful not to draw unjustified conclusions from the interesting report of Dr. Geehr and his colleagues.

after menopause. Women whose cancer is diagnosed when they are entering menopause at 54 have a better prognosis than either 54-year-old women who are several years past menopause or 49-year-old menopausal women. The prognosis of middle-aged women with breast cancer probably depends jointly on the chronological relation of diagnosis to menopause and the age at diagnosis.

SIDNEY KLAWANSKY, M.D., PH.D.
Whitaker College,
Cambridge, MA 02139 Massachusetts Institute of Technology

1. Rochman H, Conniff ES, Kuk-Nagle KT. Age and incidence of estrogen receptor positive breast tumors. Ann Clin Lab Sci 1985; 15:106-8.
2. McCarty KS Jr, Silva JS, Cox EB, Leight GS Jr, Wells SA Jr, McCarty KS Sr. Relationship of age and menopausal status to estrogen receptor content in primary carcinoma of the breast. Ann Surg 1983; 197:123-7.
3. Klawansky S, Fox MS. A growth rate distribution model for the age dependence of human cancer incidence: a proposed role for promotion in cancer of the lung and breast. J Theor Biol 1984; 111:531-87.
4. Adami H-O, Malker B, Holmberg L, Persson I, Stone B. The relation between survival and age at diagnosis in breast cancer. N Engl J Med 1986; 315:559-63.
5. Mohle-Boetani J, Grossner S, Malec M, Whittemore AS. Survival advantage among patients with breast cancer diagnosed at 45 to 49 years of age. N Engl J Med 1986; 315:587.
6. MacMahon B, List ND, Eisenberg H. Relationship of survival of breast cancer patients to parity and menopausal status. In: Forrest APM, Kunkler PB, eds. Prognostic factors in breast cancer: proceedings of the First Tenovus Symposium, Cardiff, 12th-14th April, 1967. Edinburgh: E&S Livingstone, 1968:56-64.
7. MacMahon B, Worcester J. Age at menopause. Rockville, Md.: National Center for Health Statistics, 1966:1-20. Vital and health statistics. Series 11: Data from the National Health Survey, no. 49.

The above letters were referred to the authors of the article in question, who offer the following reply:

To the Editor: Dr. Daniell suggests that there is a relation between age at diagnosis and the pattern of tumor dissemination. A predominance of visceral spread in older women might contribute to a shorter survival. However, since no curative treatment is available for patients with any type of systemic dissemination, differences in survival time should not translate into a more favorable long-term survival and a higher cure rate among patients with distant metastases first demonstrated at nonvisceral sites. The findings of Daniell thus do not explain the Swedish survival data.

Professor Lindegård has seemingly misunderstood our methods. Our analyses were based on patients available for follow-up only. A bias due to lack of follow-up in patients who emigrated after having had a breast cancer diagnosis reported to the Cancer Registry is unlikely. This hypothesis would require (1) that such women constituted a considerable proportion of all patients, (2) that they were unevenly distributed among the age groups, and (3) that their prognosis differed considerably from that of patients in the same age group who were available for follow-up. None of these requirements is sufficiently met by our data. The differences in the prevalence of breast cancer between Swedes and immigrants have no bearing on our conclusions. They are reasonable in view of the differences in age-specific incidence rates between Sweden and the low-risk and intermediate-risk countries from which most immigrants came.

Gore and Pocock suggest that the "clinical profile" acts as a confounder in the analysis of age–survival relations. Clinical covariates are complex measures, dependent on the biologic features of the tumor, the tumor–host relations, and the temporal progress of tumor growth and dissemination. Our primary aim was to analyze the biologic properties in relation to age. It seems far from clear whether adjustment for "clinical profile" is preferable in this context; it might decrease the power of the analysis without any meaningful gain in internal validity.

Our claim that age has an effect on relative survival beyond the 10th year of follow-up was based not on cumulative rates, but on the persistent difference in annual hazard rates and the consequent continued change in difference between age groups even later than 10 years after diagnosis. This finding probably cannot be refuted by data from the relatively small Western General series, which have been grouped into only three categories.

We do not agree with Dr. Klawansky that hormone-dependent and hormone-independent tumors constitute two distinct populations. Receptor concentration is a continuous — not a dichotomous — variable, and many observations indicate that its clinical correlates behave accordingly. Dr. Klawansky's hypothesis that hormone dependence is related both to age at diagnosis and to survival is reasonable from several points of view, but it does not account for the relations between age and survival observed in our study, which were more pronounced and longer lasting than the difference in survival prospects between patients with receptor-rich and those with receptor-poor tumors.

Our conclusion that "biologic mechanisms other than the hormone dependence of the tumor" may account for the relation between age and survival has been erroneously interpreted to mean that the mechanisms are necessarily of a "nonhormonal" nature. For instance, hormonal regulation of the metastatic process would be a possible explanation for the findings.

HANS-OLOV ADAMI, M.D.
LARS HOLMBERG, M.D.
S-751 85 Uppsala, Sweden University Hospital

BIRGITTA MALKER, B.SC.
National Swedish Cancer Registry
Stockholm, Sweden National Board of Health and Welfare

INGEMAR PERSSON, M.D.
Uppsala, Sweden University Hospital

MORE ON PROGRESS AGAINST CANCER

To the Editor: Letters from Bitran[1] and Mann[2] (Oct. 9, 1986, issue) in response to the article by Bailar and Smith[3] (May 8, 1986, issue) imply that prevention is unlikely to have an important impact on mortality from cancer, at least with respect to lung cancer. Much evidence suggests the contrary. Physicians can certainly do more with regard to promoting healthier practices by their patients,[4] but the potential rewards of prevention extend beyond the practitioner's office. Specifically, community health promotion studies have demonstrated a beneficial impact on a wide range of health behaviors (including smoking)[5]; work-site health promotion efforts have shown beneficial effects in hypertension control,[6] exercise,[7] and other health behaviors[8]; and school health education programs offer another promising venue for promoting healthier life styles.

Prevention can work and has. Those seeking progress against cancer might well heed the example of the undisputed progress against cardiovascular disease, which has resulted in a decline in mortality of approximately 25 percent in the United States from 1964 to 1979.[9] One analysis ascribes more than half the decline to changes in life style.[10]

The comment by Bitran regarding rising incidence rates of lung cancer among both white men and women is incorrect. The incidence of lung cancer among white males actually declined by 4 percent between 1982 and 1983[11] — a result of the decline in the prevalence of smoking among males, from 52 percent in 1963 to about 35 percent in 1983.

Although biomedical research is essential to progress in both treatment and prevention and much remains to be learned about the cause of many cancers, present knowledge warrants a stronger attack on the preventable cause of approximately one third of all cancer mortality — cigarette smoking. The failure to reduce the prevalence of smoking further speaks less to the efficacy of prevention than to our failure to fund and promote it adequately.

DONALD H. GEMSON, M.D., M.P.H.
Columbia University
New York, NY 10032 School of Public Health

1. Bitran JD. Progress against cancer? N Engl J Med 1986; 315:963.
2. Mann DA. Progress against cancer? N Engl J Med 1986; 315:966.

3. Bailar JC III, Smith EM. Progress against cancer? N Engl J Med 1986; 314:1226-32.
4. Gemson DH, Elinson J. Prevention in primary care: variability in physician practice patterns in New York City. Am J Prev Med 1986; 2: 226-34.
5. Puska P, Nissinen A, Tuomilehto J, et al. The community-based strategy to prevent coronary heart disease: conclusions from the ten years of the North Karelia Project. Annu Rev Public Health 1985; 6:147-93.
6. Foote A, Erfurt JC. Hypertension control at the work site: comparison of screening and referral alone, referral and follow-up, and on-site treatment. N Engl J Med 1983; 308:809-13.
7. Blair SN, Piserchia PV, Wilbur CS, Crowder JH. A public health intervention model for work-site health promotion: impact on exercise and physical fitness in a health promotion plan after 24 months. JAMA 1986; 255: 921-6.
8. Fielding JE. Health promotion and disease prevention at the worksite. Annu Rev Public Health 1984; 5:237-65.
9. Vital and health statistics in the United States. Vol. 2. (Mortality, Part B.) Rockville, Md.: National Center for Health Statistics, 1979 and 1964.
10. Goldman L, Cook EF. The decline in ischemic heart disease mortality rates: an analysis of the comparative effects of medical interventions and changes in lifestyle. Ann Intern Med 1984; 101:825-36.
11. Horm JW, Kessler LG. Falling rates of lung cancer in men in the United States. Lancet 1986; 1:425-6.

To the Editor: Although Bailar and Smith minimize the importance of incidence rates, an examination reveals that increases in cancer rates cannot be ascribed exclusively to tobacco use. Overall cancer incidence has increased sharply, at the rate of 1 percent annually since 1970 and at similar rates as that for lung cancer alone.[1] Incidence rates have also risen sharply for cancers unrelated to smoking, including breast and colon cancer and acute adult nonlymphatic leukemia; only about one third of the overall increase is due to respiratory cancer. For some cancers, the rising incidence rates contrast with constant mortality, as increases appear to have been offset by small improvements in treatment. Moreover, substantial recent increments in mortality rates of cancers not related to smoking have been masked by focusing on overall mortality data alone.[2] For example, from 1979 to 1983 there were marked increases in mortality rates among blacks for cancers of the prostate, lymphatic–hematopoietic system, and breast; lung cancer accounts for, at most, half the recent increase in cancer mortality among black males. Similarly, for the entire U.S. population from 1979 to 1983, there were major increases in cancer-associated mortality rates for cancer of the lymphatic–hematopoietic system and in the category of "all other causes," including malignant diseases such as soft-tissue sarcoma; there were also increases for melanoma and for cancers of major sites such as the colon from 1969 to 1976,[1] although new site categorization by the National Center for Health Statistics makes it difficult to obtain recent comparative data.

Furthermore, there is substantial evidence that occupational and community exposures to carcinogens are major factors in increasing lung cancer rates that cannot be attributed exclusively to smoking. Lung cancer rates are higher among blacks than whites, although a smaller proportion of blacks have ever smoked and black smokers smoke less than white smokers.[3] Increasing proportions of lung cancers are adenocarcinomas, which are either weakly related or unrelated to smoking.[4] Rates for cancers other than lung cancer that are associated with high relative risks among smokers, such as cancer of the buccal cavity, are declining.[3] Geographic patterns of lung cancer closely follow those of petrochemical and other industries and have recently shifted to reflect their migration to the south-central United States, and studies have also incriminated community exposure to atmospheric emissions from industries manufacturing or using industrial carcinogens.[5] High lung cancer rates that are largely insensitive to smoking levels have been identified among various occupational groups.[6] Also, case–control studies have demonstrated an occupational cause in 30 to 40 percent of lung cancers in Norway and Italy.[7]

There is ample support for the growing perception that we have lost the war against cancer and that national policies and priorities, including those of the major concerned institutions — the National Cancer Institute and the American Cancer Society — must be redirected from currently imbalanced and overly optimistic emphasis on chemotherapy and treatment into more promising areas of prevention and control.

Samuel S. Epstein, M.D.
Chicago, IL 60680 University of Illinois Medical Center

Montreal, PQ H3C 3P8, Canada Joel Swartz, Ph.D.
University of Quebec at Montreal

1. Pollack ES, Horm JW. Trends in cancer incidence and mortality in the United States, 1969–76. JNCI 1980; 64:1091-103.
2. Age adjusted death rates for 72 selected causes by color and sex, 1979-1983. Washington, D.C.: National Center for Health Statistics.
3. Davis DL, Bridbord K, Schneiderman M. Cancer prevention: assessing causes, exposures, and recent trends in mortality for U.S. males, 1968–1978. Teratogenesis Carcinog Mutagen 1982; 2:105-35.
4. Wagoner JK, Infante PF, Bayliss DL. Beryllium: an etiologic agent in the induction of lung cancer, nonneoplastic respiratory disease, and heart disease among industrially exposed workers. Environ Res 1980; 21:15-34.
5. Epstein SS, Swartz JB. Fallacies of lifestyle cancer theories. Nature 1981; 289:127-30.
6. Axelson O, Sundell L. Mining, lung cancer and smoking. Scand J Work Environ Health 1978; 4:46-52.
7. Kjuus H, Langård S, Skjærven R. A case-referent study of lung cancer, occupational exposures and smoking. III. Etiologic fraction of occupational exposure. Scand J Work Environ Health 1986; 12:210-5.

The above letters were referred to the authors of the article in question, who offer the following reply:

To the Editor: We heartily agree with Gemson that prevention works. We also note, however, that the recently reported small decreases in the incidence of lung cancer are not yet reflected in age-adjusted death rates.[1] Although a drop in mortality from lung cancer will surely follow the fall in incidence, increased efforts to curtail smoking will cause it to come sooner. This triumph of prevention, when it occurs, will far outweigh all the advances in treatment since 1950.

Epstein and Swartz state that cancer incidence rates have been rising sharply, at an annual rate of 1 percent since 1970. We said in our article why we have reservations about the meaning of trends in incidence rates, and more recent data from the National Cancer Institute indicate a smaller increase — about 5 percent for the nine years from 1975 through 1984.[1] Also, there may be a problem in using the 1970 data because they refer to a population base that is different from that of the 1975–1984 rates. Of course, there would be no basis for complacency even if the incidence rate had been declining, and Epstein and Swartz correctly note that the trend (if any) for all cancers combined may mask contrary trends for some specific kinds of cancers.

The 1975–1984 data[1] show welcome and statistically significant decreases in the reported age-adjusted incidence of cancer of the stomach, cervix, and uterine corpus (and unspecified uterine cancer), and of leukemia (all types), as well as significant increases in cancer of the colon–rectum, liver, lung–bronchus, breast, prostate, testis, urinary bladder, and kidney, and in non-Hodgkin's lymphoma and melanoma of the skin.

Epstein and Swartz comment only on the apparent increases in the incidence of some cancers, as well as the clear increases in cancer-associated mortality in black males and in some kinds of cancer in the whole population. This is the converse of the tendency of the National Cancer Institute and others to emphasize the positive.[2,3] We are glad of the support of Epstein and Swartz in promoting a shift of emphasis toward cancer prevention, but we believe that arguments for such a shift must include a strictly evenhanded and objective reading of the situation as a whole. The rate of progress against cancer is discouraging enough even when all the successes are included. The overall rate of death from cancer is rising, and if lung cancer is excluded, the rate has been essentially unchanged for a decade. We see no evidence of any recent change in these patterns. The need for a comprehensive external review of the

entire U.S. national cancer program is more acute now than it was at the time our paper was published.

John C. Bailar, III
Boston, MA 02115 Harvard School of Public Health

Elaine M. Smith
University of Iowa
Iowa City, IA 52242 College of Medicine

1. Sondik EJ, Young JL, Horm JW, Gloeckler LA, eds. Annual cancer statistics review. Washington, D.C.: National Cancer Institute, December 1986.
2. National Cancer Institute. Update. December 8, 1986.
3. Greenwald P, Sondik EJ, eds. Cancer control objectives for the nation: 1985–2000. National Cancer Institute Monograph no. 2, 1986.

"I ACCUSE" THE *JOURNAL* OF UNFAIR JOURNALISM

To the Editor: On January 13, 1898, in an open letter to President Félix Faure on the Dreyfus case, Émile Zola started each paragraph with the words "J'accuse" (I accuse). In this letter I continue this practice.

I accuse the *New England Journal of Medicine* of not providing an equal forum for the debate of issues. Fair journalism calls for equal coverage.

I accuse the *Journal* of publishing, without a balancing view, the article by Bailar and Smith that concluded "we are losing the war against cancer."

I accuse the *Journal* not merely of publishing such controversial views but of issuing press releases to help publicize them. The resultant flood of secondary articles reached the deserts of Arizona.

I accuse the *Journal* of burying balancing views such as those of DeVita (from the National Cancer Institute) and Korn (from Stanford University) (Oct. 9, 1986, issue)* among a welter of letters to the Editor. Neither their views nor any other balancing views have permeated these lay hinterlands.

I accuse the *Journal* of thoughtless weighting of views. Concurrent opposing views should be sought for simultaneous publication. Press releases should be balanced from the start.

I accuse the *Journal* of potentially jeopardizing health care and research in the United States by such practices. I look forward to your rebuttal.

Frederick Hecht, M.D.
Southwest Biomedical
Scottsdale, AZ 85251 Research Institute

*DeVita VT Jr, Korn D. Progress against cancer? N Engl J Med 1986; 315:964.

Editor's reply: The *Journal* rarely publishes countervailing opinions on controversial articles in the same issue with the original article. Our usual practice is to rely on our Correspondence section for expressions of opposing views, and that is what we did in the case of the Bailar and Smith article.

Because we received so many letters, both pro and con, we published more than the usual number and devoted five pages in our October 9 issue to that purpose. Far from "burying balancing views," as Dr. Hecht claims, we included among the 12 published letters no less than 6 that were critical of the Bailar and Smith article, including the letter from DeVita and Korn.

In view of their strong criticisms of the Bailar and Smith article, DeVita and his colleagues were invited to submit a full-length article presenting their view of the subject, but they declined.

As for the "balanced" press releases Dr. Hecht demands from us, he needs to know that the *Journal* issues no press releases of any kind. We don't believe that is our job. We let the media decide whether any of our articles are newsworthy and how they are to be interpreted to the public.

On this subject (progress against cancer), as on any other controversial health topic, we remain open to responsible views from all sides. The Bailar and Smith article was unsolicited. We do not set the agenda, nor do we influence what our contributors choose to say. If Dr. Hecht can think of a better way for us to deal with scientific debate, we would welcome his suggestions.

Arnold S. Relman, M.D.

HERPES SIMPLEX VIRUS INFECTIONS

To the Editor: In their excellent review of the clinical spectrum of herpes simplex virus (HSV) infections, Corey and Spear (March 13, 1986, issue)[1] neglected to discuss the less commonly acknowledged HSV infections involving just the skin. Although cutaneous involvement by HSV is associated with concomitant lesions of the mucous membranes or a disseminated HSV infection, it may also occur alone as an initial manifestation of an HSV infection. We recently evaluated a patient with cutaneous involvement by HSV that occurred without known exposure to the virus or other identifiable risk factors usually associated with HSV infections.

A 23-year-old woman had a vesicular eruption on the left knee that was associated with generalized malaise, fever, paresthesias, and inguinal adenopathy. Five weeks earlier she had had a second-degree burn on the same site and had intermittently "picked off" the eschar. The vesicular lesions resolved without therapy in about 10 days. Six months later a similar eruption developed on the same site; the patient sought medical care and was treated with oral cephalosporins for a presumed cellulitis. The lesions resolved in about seven days. After a third outbreak, the patient was seen at the University of Connecticut Health Center and was found to have clusters of vesicles on an erythematous base (Fig. 1), with associated tenderness and localized erythema. A Tzanck smear revealed

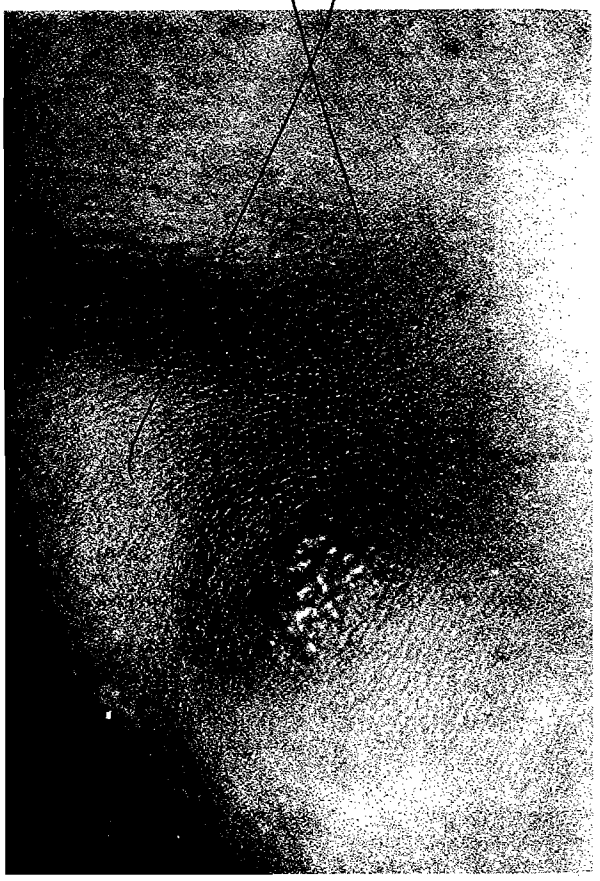

Figure 1. Cutaneous Herpes Simplex Virus Type 2 Infection of the Left Knee.