

Special Article

GAINS IN LIFE EXPECTANCY FROM MEDICAL INTERVENTIONS —
STANDARDIZING DATA ON OUTCOMES

JANICE C. WRIGHT, PH.D., AND MILTON C. WEINSTEIN, PH.D.

ABSTRACT

Background The gain in life expectancy is an important measure of the effectiveness of medical interventions, but its interpretation requires that it be placed in context. The interpretation of gains in life expectancy is particularly problematic for preventive interventions, for which the gains are often just weeks or even days when averaged across the entire target population.

Methods We tabulated the gains in life expectancy from a variety of medical interventions as reported in 83 published sources and categorized them according to target population and disease. We considered prevention in populations at average risk for particular diseases, prevention in populations at elevated risk, and treatments in populations with established disease.

Results The gains in life expectancy from preventive interventions in populations at average risk ranged from less than one month to slightly more than one year per person receiving the intervention, but the gains were as high as five years or more if the prevention was targeted at persons at especially high risk. The gains in life expectancy from treatments of established disease ranged from several months (for coronary thrombolysis and revascularization to treat heart disease) to as long as nine years (for chemotherapy to treat advanced testicular cancer).

Conclusions A gain in life expectancy from a medical intervention can be categorized as large or small by comparing it with gains from other interventions aimed at the same target population. A gain in life expectancy of a month from a preventive intervention targeted at populations at average risk and a gain of a year from a preventive intervention targeted at populations at elevated risk can both be considered large. The framework we developed for standardizing gains in life expectancy can be used in the interpretation of data on the outcomes of interventions. (N Engl J Med 1998;339:380-6.)

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THE gain in life expectancy is an important outcome of many medical interventions. It can help patients and physicians decide whether the benefits of an intervention outweigh its harm or help an insurance company decide whether or not to cover a new medical procedure. It can help a pharmaceutical company decide whether

a new drug is sufficiently more effective than the standard drugs to be worth marketing or help an expert panel designing guidelines for clinical practice sharpen its recommendations. Although there are well-developed criteria for assessing the quality of evidence of the effectiveness of a medical intervention (for example, the P value of a statistical test or the adequacy of controls for confounding), there is no criterion for assessing its magnitude.

It is especially difficult to establish a perspective on the gains in life expectancy from preventive interventions, because frequently only a small fraction of the recipients of the intervention actually realize any benefit, driving down the average gain. Thus, strategies aimed at preventing life-threatening diseases may appear ineffective alongside treatments for those who are already ill.

In this article, we propose that a gain in life expectancy from a medical intervention can be categorized as large or small by comparing it with gains from others of its type — that is, with other interventions aimed at the same target population. We present a comprehensive set of data on published gains in life expectancy from medical interventions, stratified according to the target population. This work is a contribution to the developing technology of calibrating and standardizing the effectiveness of medical interventions, and it can help inform a clinician's intuition or a policy maker's judgment about the importance of a life-extending preventive service or treatment.

In the field of public health, the effectiveness of preventive services is usually measured in terms of the number of cases prevented or the number of lives saved. Thus, the effectiveness of aggressive screening for colorectal cancer has been estimated to be approximately 2000 cases prevented per 100,000 persons screened.¹ This type of measurement, however, does not tell us how premature the avoided deaths would have been. For example, preventing a teenager's death from an automobile accident would be regarded differently from preventing a death from

From the Harvard School of Public Health, Boston. Address reprint requests to Dr. Weinstein at the Department of Health Policy and Management, Harvard School of Public Health, 718 Huntington Ave., Boston, MA 02115.

hospital-acquired pneumonia in a patient with end-stage cardiac disease.

By contrast, the effectiveness of medical treatments is often measured in terms of the increase in the proportion of people alive at fixed points in time — typically, changes in one-, two-, or five-year survival. Such changes can be given in relative or absolute form, which often leads to confusion. For example, a base-line mortality rate of 20 percent is reduced to 19 percent by a 5 percent reduction in relative risk, but it is reduced to 15 percent by a 5 percent reduction in absolute risk. Reporting the effectiveness of a treatment as a relative improvement is misleading, because the base-line death rate is ignored, but reporting improvements in survival rates in absolute terms still leaves some questions unanswered. Are the survivors all destined to live “normal” lives? What is the justification for focusing on a particular interval after the intervention (e.g., 5 years), given that two populations with the same chances of surviving for 5 years may, by virtue of risk factors or coexisting illnesses, have very different probabilities of surviving the first 12 months or the following 20 years? The same questions are unanswered by another common measure, the increase in the median survival time (or half-life) of the cohort, which is often used for reporting the results of clinical trials of treatments for cancer and other progressive diseases.

An argument for a new measurement — the number of people who must be treated in order to prevent one expected death or, more generally, to produce one successful outcome — has been made on the grounds that this would give the clinician an idea of how to apportion effort.² This measurement is inversely proportional to the number of lives saved and, again, does not tell us how long the survivors will live.

A much richer understanding of lifesaving effectiveness comes from comparing the full survival curves of treatment and control groups. The great advantage of the gain in life expectancy as a measure of outcome is that it is a direct measure of the shift in the survival curve caused by the intervention. Mathematically, the gain in life expectancy is the area between the two survival curves (Fig. 1). In contrast, each of the two traditional methods of measuring the effectiveness of treatments captures only one dimension of the shift in the survival curve and may even be misleading if the survival curves for the treatment and control groups cross.

There are two challenges associated with using the gain in life expectancy — one for the analyst and one for the user of the analysis. First, survival data are almost always censored, because some members of the cohort are still alive at the end of the clinical trial or observational study. A model must be constructed to extrapolate the survival curves beyond the end of the study, and the estimate of the gain in life expectancy may be very sensitive to the choice of model.

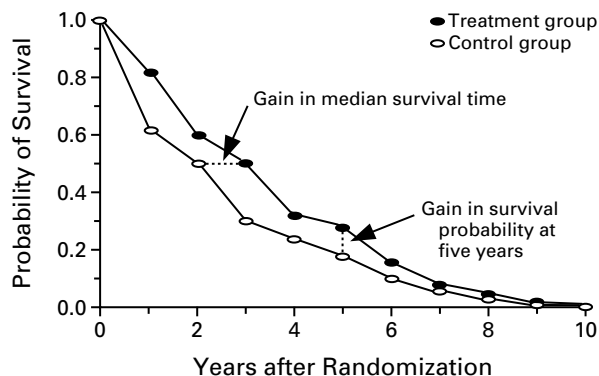


Figure 1. Hypothetical Survival Curves for a Treatment Group and a Control Group.

The life expectancy of an individual person corresponds to the area under the relevant survival curve. Thus, the gain in life expectancy from the intervention is represented by the area between the two curves. Adapted from Naimark et al.³

Second, because the gain in life expectancy is a two-dimensional measure of effectiveness, it is cognitively difficult to develop an intuitive feel for what constitutes a large or a small gain. A gain is usually thought of as a certain gain at the end of life rather than as a probabilistic gain throughout the remainder of life.³ (Often, most of the gain in life expectancy — the upward shift of the survival curve — occurs soon after the intervention.) This cognitive distortion is greater for preventive interventions than for treatments, because the base-line life expectancy is generally greater.

METHODS

Hypotheses

We began with some hypotheses about how the magnitudes of the gains in life expectancy might vary according to the characteristics of the target populations. Of the characteristics currently recorded, age, sex, and race are the primary determinants of life expectancy in the general population. In populations with risk factors for particular diseases and in populations with established diseases, these demographic factors become less important as the relative risk rises or the clinical status worsens.

The prevalence and incidence rates of the disease in the target population set upper bounds on the gain in life expectancy from a preventive intervention. Thus, a screening intervention can never lead to a large gain in life expectancy if the disease has a low prevalence, and a vaccination program can offer only a limited gain if the disease has a low incidence. Conversely, curative or palliative interventions are targeted at populations in which everyone already has the disease, so there is the potential for large gains. However, the same factor that makes the potential gain large — a poor prognosis — will often drive down the actual gain if survivors have other risks that reduce the potential gain in longevity.

Specifically, we might expect to find the following hypotheses to be true. First, the gains for older populations will be smaller than those for younger populations for several reasons: disease-specific mortality and competing risks of death increase with age, fatal complications from treatment are more likely, and there are fewer years that can be gained by averting a death.⁴ Second, the gains for women will be a little larger than those for men if the disease is not sex-specific in either occurrence or severity, because women

have lower age-specific mortality rates than men. Third, if only a few people actually benefit from the intervention (e.g., because of a low incidence of disease in the case of primary prevention or a low prevalence of disease in the case of screening), the average predicted gain will necessarily be small, even if the lives of those few people are extended by many years. And fourth, the more advanced the disease in the target population, the poorer the prognosis for the population and the greater the potential gain from treatment, but that gain will be correspondingly harder to realize. These hypotheses cannot be tested formally with our data, since we are limited to a sample of interventions for which the gains in life expectancy have been estimated in published papers. Nevertheless, they explain some of the variation in gains seen in our results.

Collection of Data

For this study, the gains in life expectancy from various medical interventions were taken directly from or were calculated from data in 83 published sources, many of which were found through a Medline search. Sources were selected if they reported gains in life expectancy or the data required for a simple calculation of gains and if they were published in English. The quality of the analysis (other than as indicated by the publication of the report in a peer-reviewed journal) was not a criterion, since our aim was to gather information on gains in life expectancy for as wide a variety of interventions as possible. We made no attempt to select the “best” article when we found more than one on the same intervention, because comparing analyses of the same or similar interventions can be valuable. We rejected some sources because the technology of the intervention has changed substantially or is no longer used.

It is rare for the primary purpose of a study to be the calculation of gains in life expectancy. The majority of the articles that yielded the information we sought were either decision analyses⁵ or cost-effectiveness analyses.⁶ Many of these analyses were appended to clinical trials or epidemiologic investigations to quantify the magnitude of a clinical benefit. Many analyses of cost effectiveness could not be used as sources, because the authors had adjusted the reported gains in life expectancy for health-related quality of life or had discounted them to present value (or both), without reporting the corresponding unadjusted and undiscounted values, as is currently recommended.⁷

Some important interventions do not appear in our study. Investigators examine interventions that are salient because they are new, because they are controversial, or both. For instance, screening for and treatment of early-stage breast cancer are currently under intense scrutiny because of the controversy over the optimal age at which women should begin periodic mammographic screening. Thus, breast cancer is prominent in our results. We were able to find the gain in life expectancy from a new drug for survivors of stroke — ticlopidine — but not the gain from the standard drug, aspirin. Our results include some interventions that are used commonly and some that are seldom used; those presented here should not be interpreted as reflecting the full range of life-extending interventions.

Some authors modeled the gains in life expectancy for the typical patient, whereas others modeled the gains for many target populations, varying age, sex, risk factors, clinical status, and occasionally, race in their models. We do not present all these subgroup analyses; rather, we report the gains in life expectancy for selected target subpopulations and indicate that the results of other analyses are available in the cited articles.

Some authors reported gains in life expectancy as point estimates, whereas others reported ranges. These ranges are sometimes formal confidence intervals or credible intervals and sometimes reflect the effect of varying a key parameter or modeling assumption in a sensitivity analysis.

We converted all the gains in life expectancy to months. The number of significant figures and decimal places varies somewhat. In cases in which the gains were very small, they are necessarily reported to as many as three decimal places, but this does not imply any judgment of greater precision. For several interventions, we

TABLE 1. PREVENTION IN POPULATIONS AT AVERAGE RISK.

| DISEASE AND INTERVENTION | TARGET POPULATION | GAIN IN LIFE EXPECTANCY (MO)* | |
|----------------------------------------------------------------------------------------------------|-------------------|-------------------------------|-----------------|
| | | MALE SUBJECTS | FEMALE SUBJECTS |
| Cardiovascular disease | | | |
| Exercise consuming 2000 kcal/wk for 30 yr ⁸ | 35-year-old men | 6.2 | NA |
| Quitting cigarette smoking ⁹ | 35-year-olds | 10 | 8 |
| Hormone-replacement therapy with estrogen only for women who have had hysterectomies ¹⁰ | 50-year-old women | NA | 13 |
| Cancer | | | |
| 10 yr of biennial mammography ¹¹ | 50-year-old women | NA | 0.8 |
| Pap smear | 20-year-old women | NA | |
| Every 3 yr for 55 yr ¹² | | | 3.1 |
| Every 5 yr for 55 yr ¹² | | | 3.2 |
| Annual fecal occult-blood test, plus barium enema or colonoscopy | 50-year-olds | | |
| Every 5 yr for 25 yr ¹ | | 2.5 | 2.2 |
| Every 3 yr for 25 yr ¹ | | 2.8 | 2.5 |
| Infectious disease | | | |
| Measles vaccine ¹³ | Infants | | 0.09 |
| Rubella vaccine ¹³ | Infants | | 0.10 |
| Mumps vaccine ¹³ | Infants | | 0.01 |
| Pertussis vaccine ¹⁴ | Infants | | 0.11 |
| Hepatitis B virus vaccine ¹⁵ | Newborns | | 0.26 |
| | Adolescents | | 0.12 |
| | Adults | | 0.03 |

*NA denotes not applicable.

calculated the gains in life expectancy from data provided in the primary sources. Generally, these calculations involved a straightforward conversion of lives saved to life-years saved per person, with life tables used to estimate life expectancy.

Owing to space constraints, the tables we present here show gains in life expectancy from only 31 of the 83 published sources. The complete set of gains, as well as the details of our methods of calculating them from the primary-source data, is available on the following Web site: www.hsph.harvard.edu/organizations/hcra/peemt.html.

RESULTS

Since we propose that a gain in life expectancy from a medical intervention can be categorized as large or small by comparing it with gains from other interventions aimed at the same target population, we present our results in tables organized primarily according to target population.

Tables 1 and 2 show data on preventive strategies, and Table 3 shows data on treatments. It is impossible to draw a clear distinction between prevention and treatment. For instance, prophylaxis against *Pneumocystis carinii* pneumonia in patients infected with the human immunodeficiency virus is, strictly speaking, a preventive strategy, but we chose to categorize it as a treatment.

In cases in which the gains in life expectancy estimated for men and women are different, both are presented. If the gain is not sex-specific, it is centered

TABLE 2. PREVENTION IN POPULATIONS AT ELEVATED RISK.*

| DISEASE AND INTERVENTION | TARGET POPULATION | GAIN IN LIFE EXPECTANCY (MO) | |
|---------------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------|------------------------------|-----------------|
| | | MALE SUBJECTS | FEMALE SUBJECTS |
| Cardiovascular disease | | | |
| Reduction of diastolic blood pressure to 88 mm Hg ⁹ | 35-year-olds with hypertension | 13 | 11 |
| | Diastolic blood pressure of 90–94 mm Hg | 64 | 68 |
| Reduction of cholesterol to 200 mg/dl (5.2 mmol/liter) ⁹ | 35-year-olds with hypercholesterolemia | 6 | 5 |
| | Cholesterol level of 200–239 mg/dl (5.2–6.2 mmol/liter) | 50 | 76 |
| Reduction of weight to ideal level ⁹ | 35-year-olds | 8 | 6 |
| | <30% over their ideal weight | 20 | 13 |
| Quitting cigarette smoking ⁹ | 35-year-old smokers | 28 | 34 |
| | ≥30% over their ideal weight | NA | NA |
| Hormone-replacement therapy with estrogen and progestin ¹⁰ | 50-year-old women with a history of coronary artery disease | NA | 11 to 26 |
| | 50-year-old women at high risk for coronary artery disease | NA | 7 to 19 |
| | 50-year-old women at high risk for breast cancer | NA | –6 to 10 |
| | 50-year-old women at high risk for hip fracture | NA | 2 to 13 |
| Cancer | | | |
| Initial office biopsy to evaluate postmenopausal bleeding, followed by dilation and curettage or hysterectomy if needed ¹⁶ | Women at high risk | NA | 6.0 |
| | 50-year-old | | 2.2 |
| Prophylactic bilateral mastectomy ¹⁷ | Women who carry <i>BRCA1</i> or <i>BRCA2</i> mutation | NA | 35 to 64 |
| | 30-year-old | | 12 to 28 |
| Prophylactic bilateral oophorectomy ¹⁷ | Women who carry <i>BRCA1</i> or <i>BRCA2</i> mutation | NA | 4 to 20 |
| | 30-year-old | | 1 to 10 |
| 50-year-old | | | |
| Infectious disease | | | |
| Hepatitis B virus vaccine ¹⁵ | 12-to-50-year-olds at high risk for hepatitis | 0.15 to 0.24 | |
| | Newborn babies whose mothers have been exposed to or have hepatitis B | 0.28 | |
| Testing of the blood supply for HIV ¹⁸ | Surgical patients | | |
| | 30-year-old | 0.27 | |
| | 50-year-old | 0.15 | |
| Preoperative autologous blood donation ¹⁹ | 70-year-old | 0.06 | |
| | Patients undergoing coronary-artery bypass grafting | 0.002 to 0.004 | |

*NA denotes not applicable, and HIV human immunodeficiency virus.

between the columns for male and female subjects in the tables.

The age of the target population is the age from which the gain in life expectancy is estimated. For instance, in Table 1, the three-month gain associated with Pap smears is the gain that can be expected for 20-year-old women who embark on a lifelong screening program; a woman who begins screening for cervical cancer at 50 years of age will increase her life expectancy by less than three months.¹²

Table 1 shows the gains in life expectancy associated with prevention in populations at average risk. In these populations, the incidence and prevalence

of disease matter enormously. For example, a program of physical exercise begun at the age of 35 years increases life expectancy by 6.2 months,⁸ and complete cessation of smoking at the age of 35 increases life expectancy by 9 months,⁹ but a decade of biennial mammography begun at the age of 50 increases life expectancy by only 0.8 month.¹¹ Even the highly effective childhood vaccines against measles, rubella, and pertussis offer gains in life expectancy of only approximately 0.1 month each.^{13,14} For the preventive interventions targeted at people at average risk, it is evident from Table 1 that a gain on the order of only a month can be considered large.

TABLE 3. TREATMENTS OF PERSONS WITH ESTABLISHED DISEASE.*

| DISEASE AND INTERVENTION | TARGET POPULATION | GAIN IN LIFE EXPECTANCY (MO) | |
|--------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------|-----------------|
| | | MALE PATIENTS | FEMALE PATIENTS |
| Cardiovascular disease | | | |
| Myocardial revascularization with coronary-artery bypass grafting or percutaneous transluminal coronary angioplasty ²⁰ | Men with coronary artery disease | | NA |
| | 1 Vessel | 1–7 | |
| | 2 Vessels | 0–8 | |
| Routine beta-blocker therapy ²¹ | 3 Vessels | 4–14 | |
| | 55-year-old men who survive acute myocardial infarction | | NA |
| | Low risk of recurrence | 1.2 | |
| | Medium risk of recurrence | 4.1 | |
| Thrombolytic therapy with recombinant tissue plasminogen activator during suspected acute myocardial infarction ²² | High risk of recurrence | 5.6 | |
| | Patients with suspected acute myocardial infarction | 15 | |
| Thrombolytic therapy with recombinant tissue plasminogen activator as compared with streptokinase ²³ | Patients with suspected acute myocardial infarction | | |
| | Inferior infarction | 0.8–3.1 | |
| | Anterior infarction | 1.2–3.5 | |
| Implantable cardioverter–defibrillator ²⁴ | Survivors of cardiac arrest with recurrent ventricular arrhythmias that do not respond to conventional therapy | 36–46 | |
| | Amiodarone therapy ²⁴ | Survivors of cardiac arrest with recurrent ventricular arrhythmias that do not respond to conventional therapy | 14–16 |
| Heart transplantation ²⁵ | Candidates with end-stage cardiac failure | 31–99 | |
| Ticlopidine as compared with aspirin ²⁶ | Patients at high risk for stroke | 0.6 | |
| Cancer | | | |
| Radical prostatectomy or radiation therapy, as compared with watchful waiting, with delayed hormonal therapy if needed ²⁷ | 65-year-old men with localized prostate cancer | 1–11 | NA |
| | Adjuvant chemotherapy ^{28,29} | Women with breast cancer | NA |
| Chemotherapy ³⁰ | Node-positive | | 3.6 |
| | Node-negative | | 7.7–11 |
| Chemotherapy ³¹ | Patients with extensive small-cell lung cancer | 6.6–8.2 | |
| | Patients with advanced non-small-cell lung cancer | 1.8–2.9 | |
| Chemotherapy ³² | Men with advanced testicular cancer | 107 | NA |
| | Autologous bone marrow transplantation as compared with standard chemotherapy ³³ | Patients with relapsed non-Hodgkin's lymphoma | 72 |
| Other | | | |
| Prophylaxis against <i>Pneumocystis carinii</i> pneumonia and toxoplasmosis ³⁴ | Patients with advanced HIV disease | 5.3 | |
| Prophylaxis against <i>Mycobacterium avium</i> complex, fungal infections, or cytomegalovirus ³⁴ | Patients with advanced HIV disease | 0.2–0.3 | |
| Elective surgery as compared with expectant management ³⁵ | 50-year-olds with symptomatic gallstones | 1.7 | 3.4 |
| Interferon therapy ³⁶ | 35-year-olds with chronic hepatitis B who are positive for hepatitis B e antigen and do not have cirrhosis | 37 | |
| Appendectomy ³⁷ | Patients with suspected acute appendicitis | | |
| | Probable | 9–31 | |
| | Possible | 2–5 | |

*NA denotes not applicable, and HIV human immunodeficiency virus.

Table 2 shows the gains in life expectancy associated with prevention in populations at elevated risk. In some cases, the elevated risk is only slightly greater than the average risk for the disease; in other cases, it is much greater. Many of the interventions shown in this table yield gains on the order of a year. For example, 35-year-old male smokers who quit smoking gain 28 months of life expectancy,⁹ and 50-year-old women at elevated risk for coronary artery disease gain 7 to 19 months from hormone-replacement therapy.¹⁰ At the other extreme, the gain from preoperative autologous blood donation is very small — about two hours.¹⁹

Table 3 shows the gains in life expectancy associated with treatment in target populations with established cardiovascular disease, cancer, or other diseases. The gains from treatment of coronary artery disease increase with the severity of the disease, but few exceed a year. Most of the cancer treatments yield gains that are much smaller than those from the three aggressive preventive interventions shown in Table 2.^{16,17} However, there are gains of several years associated with a number of the treatments shown in Table 3, such as implantable defibrillators for survivors of cardiac arrest (36 to 46 months),²⁴ bone marrow transplantation for relapsed non-Hodgkin's lymphoma (72 months),³³ and chemotherapy for testicular cancer (107 months).³²

DISCUSSION

Those who provide and pay for medical care make decisions about preventive strategies and treatments in an environment in which quantitative measures of outcome are increasingly common. By collecting and categorizing the gains in life expectancy from a wide variety of medical interventions, we have developed benchmarks for the size of the gain that can be expected in various populations, thus providing a valuable resource for those who set clinical-practice guidelines or make intervention-specific decisions about insurance coverage. Moreover, the organization of gains in life expectancy according to target population, disease, and type of intervention has established a framework that can be used for the presentation of other standardized data on outcomes.

Virtually all life-extending medical care has both positive and negative effects on health-related quality of life, and sometimes reduction in morbidity is the main outcome of the intervention, with the life-saving benefit as a bonus. Information on gains in quality-adjusted life expectancy is available from many medical cost-effectiveness and decision analyses, and could be presented systematically alongside information on gains in life expectancy. Similarly, since many of the data on gains in life expectancy and quality-adjusted life expectancy are available from cost-effectiveness analyses, cost-effectiveness ratios — measured in both dollars per year and dollars per quality-adjusted year

— could be added to our tables. Such efforts are fraught with difficulties, however, and until investigators follow reasonably uniform practices when conducting cost-effectiveness analyses, the results will be of limited value.

Although the gain in life expectancy is a richer measure of the effectiveness of “lifesaving” interventions than those used traditionally, it should not be used simplistically in clinical decision making. The reported gain in life expectancy is averaged across the target population receiving the intervention and offers no information about the distribution of the gains in life expectancy actually realized by particular patients. The mean gain may reflect a small gain for most members of a population but a very large gain for a few members who might have died prematurely without the intervention. For example, consider the triennial cervical-cancer screening program¹² shown in Table 1. The mean gain in life expectancy from screening is about 3 months for the target population, but the women whose cancers are detected preclinically actually gain an average of 25 years. Similarly, the average gains from vaccination of infants are all very small, but those whose deaths are averted gain virtually their whole lifetimes. Viewed this way, the gains of months in life expectancy from preventive interventions will often be equivalent to gains of years from medical treatments.

At the other extreme, those making decisions about the allocation of medical resources may be interested in the overall effect of interventions on the life expectancy of the whole population. A highly effective intervention will have a very small effect on the life expectancy of the population if the disease is rare. For example, the gain in life expectancy from chemotherapy for testicular cancer is about nine years for those receiving the intervention (Table 3).³² However, because this disease is so rare, the gain from making this treatment available to the man at average risk is about one hour. This gain is very small in comparison with the population-wide gains of months from the preventive interventions for coronary heart disease⁹ shown in Table 1.

The gains in life expectancy from medical interventions intended to prevent disease seem small because of the effect of averaging across a population, most members of which would never contract the disease. Our analysis establishes that a gain of a month from a preventive strategy aimed at the general population signals an important intervention.

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CORRECTION

Gains in Life Expectancy from Medical Interventions

To the Editor: Wright and Weinstein's article (Aug. 6 issue)¹ on the gains in life expectancy from medical interventions allows physicians to compare the value of different medical interventions in various at-risk populations. We have used similar data to motivate patients to exchange bad health habits for good ones. Much as Friedman found that investors discounted the gain from investments to zero when the gain was more than three years away,² we have found that patients discount the value of good health choices for a gain in life expectancy in the seemingly distant future. When we translated medical interventions, through a mechanism similar to Wright and Weinstein's, into a more immediate time frame, we were successful in motivating patients to reconsider preventive health strategies. Our unit of measurement, called RealAge, is the measure of physiologic improvement that results from an intervention affecting health. Analogous to net present value, which makes investment decisions seem rational, RealAge is the translation of increased life expectancy into net present value. The addition of five years to one's life may not be a sufficiently immediate gain to provide the motivation to reduce one's blood pressure from 150/93 mm Hg to 120/80 mm Hg; however, some patients respond enthusiastically to the idea of retarding the speed of aging or becoming physiologically younger. Thus, a 49-year-old man who controls his blood pressure, quits smoking cigarettes, or exercises may have a RealAge of 37 years.

Like Wright and Weinstein, we found that the value of immunization was just six days, but control of blood pressure, cessation of cigarette smoking, exercise, or stress control makes one more than five years "younger." Various theoretical models have been developed to explain health-related behavior, including the health belief model, the theory of reasoned action, the social-learning theory, and the transtheoretical model and stages of change.³ The model proposed by Wright and Weinstein, or a version of it transformed into RealAge measurement of physiologic changes, borrows from the Chicago school of economics to motivate patients to choose healthy behavior. We believe Wright and Weinstein's model should be expanded to include either the best data or the results of a meta-analysis on each topic. The model could further include interaction and covariance terms derived from the literature or data base to account for interdependence among predictor variables. We believe that such models will allow patients and doctors to make more rational choices to enhance health.

Michael F. Roizen, M.D.
Keith Roach, M.D.
University of Chicago
Chicago, IL 60637

Axel Goetz, M.D., Ph.D.
RealAge, Inc.
San Diego, CA 92121

Editor's note: Drs. Roizen and Roach are consultants to RealAge, Inc., and have an equity interest in the firm.

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To the Editor: In dealing with data on long-term survival for cost-effectiveness analysis, the rationale underlying methods of discounting values is that future survival is valued less than present survival.¹ Although the guidelines published in 1996 recommend reporting both discounted and undiscounted values for gains in survival,¹ in their article and on the Internet (www.hsph.harvard.edu/organizations/hcra/peemt.html) Wright and Weinstein reported only undiscounted values.

We have reported the values for discounted gains in survival for a number of anticancer treatments (together with information on gains in survival for thrombolysis in patients with acute myocardial infarction).² A reanalysis of these oncologic data with and without discounting (annual discount rate, 3 percent or 5 percent) gives the following results. The discounted gain (with the undiscounted gain in parentheses) is 1.34 (3.08) years per patient for interferon therapy as compared with no adjuvant therapy in patients with high-risk resected cutaneous melanoma,³ 0.43 (0.45) year per patient for paclitaxel plus cisplatin as compared with standard chemotherapy in patients with advanced ovarian cancer, 1.06 (3.57) years per patient for cyclophosphamide, methotrexate, and fluorouracil as compared with no adjuvant chemotherapy in patients with node-positive breast cancer (not 3.6 undiscounted months per patient, as erroneously reported by Wright and Weinstein in Table 3 of their article), 0.89 (2.18) year per patient for intraportal chemotherapy as compared with no adjuvant chemotherapy in patients with colorectal cancer, 0.37 to 0.93 (0.30 to 1.22) year per patient for interferon therapy as compared with cytotoxic therapy in patients with chronic myelogenous leukemia,⁴ and 5.99 (3.01) years per patient for autologous transplantation as compared with chemotherapy in patients with chemosensitive non-Hodgkin's lymphoma in relapse. This information supplements the undiscounted data reported by Wright and Weinstein and confirms the well-known concept that discounting introduces marked changes in survival gains.

In conclusion, we believe that gains in survival are better described by reporting both the discounted and the undiscounted values. Although the approach that considers only undiscounted data is acceptable when survival data are not censored and there is no extrapolation, the presentation of both discounted and undiscounted values is more appropriate when the analysis has a lifetime perspective and the survival data are in part experimental and in part extrapolated.

Andrea Messori, Pharm.D.
Sabrina Trippoli, Pharm.D.
Enrico Tendi, Pharm.D.
Azienda Careggi
50134 Florence, Italy

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The authors reply:

To the Editor: We thank Messori et al. for pointing out our incorrect transcription of their estimate of the gain in life expectancy from adjuvant chemotherapy for node-positive breast cancer, which is indeed 43 months, not 3.6 months, as we reported. This correction further strengthens our conclusion that gains in life expectancy tend to be higher for effective treatments than for primary or secondary prevention. We apologize for the error.

Messori et al. favor reporting discounted as well as undiscounted gains in life expectancy, citing the recommendations of the Panel on Cost-Effectiveness in Health and Medicine,¹ which one of us cochaired. These recommendations apply to cost-effectiveness analyses intended to inform resource allocations at the population level, not to decisions for individual patients. Exponential discounting may or may not be an appropriate way to measure personal preferences for future extensions of life at the individual level.² In any case, it

is incorrect to discount because of the uncertainty surrounding extrapolation of survival data. Ideally, the underlying survival curves should be made available by the investigators, who have reported the source data, so that individual utility functions for survival gains can be applied.³

The question of the most effective way to communicate information on survival gains to patients and physicians is addressed by Roizen et al., who favor a measure based on the equivalent reduction in chronologic age. Although actuarially equivalent to the gain in life expectancy, their RealAge measure places a different "spin" on the data, in an attempt to offset the often incorrect perception that gains in life expectancy accrue at the end of life. Their efforts, along with those of others who study the communication of information about risk, should be applauded. Much more needs to be learned about communicating health risks and benefits effectively, both in terms of enhancing an understanding of them and in terms of promoting informed health decisions by patients and physicians.

Milton C. Weinstein, Ph.D.
Harvard University
Boston, MA 02115

Janice C. Wright, Ph.D.
63 Houghton Bay Rd.
Wellington, New Zealand

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