Improving health behaviors and outcomes after angioplasty: using economic theory to inform intervention

Mary E. Charlson, John P. Allegrante\textsuperscript{1}, Paula S. McKinley\textsuperscript{2}, Janey C. Peterson, Carla Boutin-Foster, Gbenga Ogedegbe and Candace R. Young

Abstract

Patients who have been relieved of cardiac symptoms following angioplasty may not be sufficiently motivated to initiate behavior changes that can reduce risk of subsequent cardiac events. Finding an effective means to help patients modify their behavior thus presents a unique challenge. This paper describes an innovative behavioral intervention whose theoretical underpinning is \textit{net-present value economic theory}. This intervention is being evaluated in a randomized controlled trial in which all patients complete a computerized baseline health assessment of 14 cardiovascular risk factors. Each patient is presented with an individualized risk-factor profile and asked to choose risk factors for modification. In the experimental group, each risk factor is presented with a corresponding numerical biologic age value that represents the relative potential to benefit from modifying each risk factor. Risk reduction for these patients is framed as the opportunity to reduce present biologic age (the net-present value), and improve current health status and quality of life. In the control group, risk reduction is framed in the standard risk-factor approach as the value of preventing future health problems. We hope to demonstrate that economic theory is a plausible perspective from which to design interventions aimed at communicating risk and facilitating change in health behaviors.

Introduction

Data from clinical trials and statewide registries of percutaneous transluminal coronary angioplasty (PTCA) show that between 30 and 40\% of angioplasty patients experience recurrent angina, myocardial infarction (MI), death or repeat procedures by 2 years of follow-up [BARI et al., 1990; Hampton et al., 1993; Rodriguez et al., 1993; Hamm et al., 1994; King et al., 1994; Rogers et al., 1995; The Bypass Angioplasty Revascularization Investigation (BARI) Investigators, 1996; Zhao et al., 1996; Hannan et al., 1997; Writing Group for the BARI Investigators, 1997]. The high recurrence rates occur because of re-stenosis of the target lesions or because of progression of the disease that results in new coronary artery lesions. Patients who are male or who have unstable or recent onset angina have a greater likelihood of recurrence in the target vessel (Holmes et al., 1984; Blackshear et al., 1987; Rupprecht et al., 1990). In addition, patients with long or complex, Grade C lesions are at higher risk for re-stenosis than those with Grade B lesions [Hamm et al., 1994; Rogers et al., 1995; The Bypass Angioplasty Revascularization Investigation (BARI) Investigators, 1996; Writing Group for the BARI Investigators, 1997]; diabetics also...
have worse long-term outcomes than non-diabetics (Galan and Hollman, 1986; Klugherz et al., 1996; Laham et al., 1996).

Given the high recurrence rates in this population, an expert committee of the WHO has recommended that rehabilitation should be provided for all patients undergoing PTCA (Wenger, 1991). Clinicians have expressed concern, however, that PTCA patients compared to other cardiovascular patient populations may be too optimistic about their future health status. Angioplasty is a procedure that is relatively non-invasive and yields rapid symptom relief. These benefits of PTCA may, paradoxically, interfere with motivation to undertake changes in behaviors that contributed to the development of coronary artery disease (Kimble, 1998). Even when recommended changes are adopted, they are not maintained over the long term (Morocutti et al., 1999). As a result, patients who have undergone angioplasty may underestimate their risk of future coronary artery disease and the potential benefits—both in the present and future—of modifying their risk behaviors. Thus, in spite of consensus that behavior change is vital for PTCA patients, post-procedure rehabilitation efforts may prove to be especially difficult in this population. This may be the reason why risk assessment and behavioral intervention have not been reliably integrated into post-PTCA practice (Agren et al., 1989; Wright and Strang, 1997).

There is, in fact, evidence that patients who have undergone PTCA tend to be either poorly informed about health risk behaviors or unlikely actually to change their behaviors (Fletcher, 1986; Hansen, 1988; Faris and Stotts, 1990; Gaw, 1992; Gaw-Ens and Laing, 1994; Gulanick et al., 1998). For example, Gaw reported that only 50% of patients interviewed could identify specific cardiac risk factors thought to be related to their cardiovascular problems (Gaw, 1992). In a review of 19 studies, Gentz found that during the post-PTCA recovery period patients reported the need for more education about risk factors, lifestyle changes and survival management (Gentz, 2000).

While the necessity for PTCA alone could be considered a potential motivator for behavior change, patients tend not to alter their health risk behaviors without an active intervention. For example, patients 1 year after PTCA showed only small reductions in cholesterol (12 mg/dl), small reductions in body mass (0.41 kg/m²) and an increase in prevalence of smoking from 5 to 13% (McKenna et al., 1995). These researchers did report a significant increase in the proportion of patients who were engaged in moderate exercise (from 32 to 65%) and suggested that relief from ischemic symptoms after angioplasty may have enabled patients to be more active.

Thus, in light of PTCA patients’ reticence to adopt and maintain health behavior change, this population presents a unique challenge: can a more effective means of communicating future health risks and motivating PTCA patients to initiate and maintain health behavior changes be developed, and what theoretical approach should inform the design of such an intervention?

We have taken the perspective originally proposed by Allegrante and Roizen that the study of human economic behavior—specifically net-present value economic theory—holds promise as a theoretical underpinning for behavioral interventions applied to health (Allegrante and Roizen, 1998). This paper describes a randomized controlled intervention trial of PTCA patients that tests whether this approach will enhance motivation to make behavioral changes. By demonstrating that principles underlying behavior in the economic realm can serve as the theoretical basis for intervention, this project promises to link the work of behavioral psychology with that of economics.

### Previous intervention approaches

The majority of behavioral interventions for patients with coronary artery disease have targeted subjects after surgical coronary artery bypass graft (CABG) or MI (Hedback and Perk, 1987). Multi-factorial cardiac risk reduction interventions that have used cardiac endpoints generally have been successful in lowering event rates in the treatment
groups (Brown et al., 1990; Buchwald et al., 1990; Ornish et al., 1990; Schuler et al., 1992; Watts et al., 1992). In spite of this success, there is little research targeting multifactorial lifestyle behavior change in patients following angioplasty.

Most studies targeting angioplasty outcomes have either been epidemiological assessments of risk factors for re-stenosis or studies of the effects of medical but not behavioral interventions on clinical outcomes (Hollman et al., 1989; Arora et al., 1990; Benchimol et al., 1990, 1993). To date only two major randomized controlled trials have assessed the efficacy of multifactorial behavioral intervention programs for PTCA patients.

In one of these trials a Swedish team developed an intervention program targeting stress management, and changes in diet, exercise and smoking behaviors (Hofman-Bang et al., 1999; Lisspers et al., 1999). The intervention consisted of an intense residential program of health education, behavior change training and planning for future lifestyle changes. The second phase was an 11-month maintenance program with regular contacts between the patient and a nurse acting as a personal coach. As a key component of the intervention, the coaches developed an individualized program for each patient based on his or her pre-PTCA health behaviors and stress profile. Patients in the control group were asked to stay in contact with their own physician and were neither encouraged nor discouraged to modify lifestyle risk factors.

Immediately after the 12-month intervention, there were significant improvements in body mass index, cholesterol levels, exercise capacity and frequency, and dietary knowledge and habits; but there were no group differences in clinical events. At 1-year follow-up the intervention group had maintained their more positive health behaviors and had had fewer hospital admissions for cardiac events compared to the control group. There was no group difference, however, in the percentage of patients who had returned to work.

In the other study, the Stanford Coronary Risk Intervention Project (SCRIP), the intervention involved an intensive, long-term multiple risk-reduction program targeting several factors, including diet, exercise, weight loss, smoking and medications among patients with PTCA or CABG (Haskell et al., 1994). Like the Swedish group’s study, this program also featured an individualized intervention. Staff nurses and dieticians instructed participants individually on risk factors including body weight, blood pressure, cholesterol level, dietary fat intake, sodium intake, smoking and physical activity. Standard program goals were set for each risk factor and applied to all participants. Emphasis was placed on each patient in the study reaching the maximum program goal for each risk factor, while minimum and intermediate goals were applied selectively to enhance motivation and program adherence.

Risk-reduction participants returned to the clinic every 2–3 months for progress reports and clinical measures. Medical and risk factor evaluations were completed at baseline and yearly for 4 years. After 4 years patients in the treatment group had decreased narrowing of coronary arteries, as confirmed by arteriograms, and fewer hospitalizations for clinical cardiac events.

Neither of these two research teams describes specifically the theoretical bases of their programs. Both programs included intervention methods that appear to have been informed by at least two popular behavioral theories: Social Learning Theory (Bandura, 1977), and the Transtheoretical Model and Stages of Change (Prochaska et al., 1992). Some of the intervention techniques included both education about risk factors and practical training in how to achieve behavior change. Individualization, a key feature of effective behavior change programs, was also used. Hence, both these programs tailored certain features of the intervention to individuals’ risk profiles and behavior change preferences. It is unclear, however, which components of these programs were most effective and why. While the intervention effects of these studies were modest, such results are promising.

Thus, previous approaches to intervention have focused on presenting risk-factor information to patients that helps them to understand how making
behavioral changes will reduce future risk. Such approaches have not provided a means by which the patient can prioritize lifestyle changes in terms of relative risks and such approaches have failed to give the patient or his or her physician a way to place a tangible value on the change in relative risk. Given the challenges health practitioners face in convincing angioplasty patients of the need to make immediate changes to stem the progression of the disease, it would appear that such patients are in need of a new intervention approach that frames the value of behavior modification in terms of present and direct benefit.

The Healthy Behaviors Trial

The Healthy Behaviors Trial is a randomized controlled trial designed to compare the efficacy of two intervention strategies—a standard care approach versus an experimental biologic-age approach—of motivating behavioral change to improve health behavior and health outcomes (i.e., 2-year survival free from MI, stroke, Class II–IV angina or severe ischemia on non-invasive testing) among coronary artery disease patients who have undergone angioplasty with or without stents. The objective of this study is to promote behavior modification in this patient population through individualized risk profile feedback, and to test what we believe is an innovative and potentially efficacious motivational approach to risk factor education.

At baseline all participants complete a computerized health assessment that evaluates 14 cardiac risk factors, including such factors as physical activity, smoking, diet, blood pressure and medications. Table I lists the risk factors targeted for intervention. Based on the results of their assessment, all patients choose two to three factors for modification. Patient education about each risk factor in an individualized profile is framed using either a standard approach or an experimental biologic-age approach where each factor is weighted according to its relative risk. To get patients started, we provide them with behavior-change ‘tip sheets’ for each of the risks they have selected to change, as well as referrals for behavior-change programs and other community resources. Both groups of patients are followed and interviewed by telephone every 3 months for 2 years. The telephone contact provides motivational support to patients in both groups, and we use basic principles of motivational interviewing that have been developed by Emmons and Rollnick (Emmons and Rollnick, 2001) during the call to assist patients in adopting and maintaining behavioral changes. In addition, each call includes an updated assessment of interval clinical events and changes in cardiovascular risk profile, as well as an opportunity for the individual patient to change or add new risk factors. We also assess stages of behavioral change and self-efficacy during these calls. A total of 660 patients will be enrolled.

The trial is thus designed to investigate the efficacy of a novel motivational approach in which a computerized health risk assessment presents individuals with the explicit net-present value of future health actions. Using this approach, a common health risk currency, which we believe to be an important, culturally relevant metric of benefit that people can understand—biologic age—has

<table>
<thead>
<tr>
<th>Table I. Risk factors targeted for behavior change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical activity</td>
</tr>
<tr>
<td>1. increase physical activity</td>
</tr>
<tr>
<td>2. increase aerobic exercise</td>
</tr>
<tr>
<td>3. increase strength training</td>
</tr>
<tr>
<td>Smoking</td>
</tr>
<tr>
<td>4. stop smoking (or continue not to smoke)</td>
</tr>
<tr>
<td>5. increase servings of fish</td>
</tr>
<tr>
<td>6. reduce red meat</td>
</tr>
<tr>
<td>7. reduce dietary intake of cholesterol and saturated fat</td>
</tr>
<tr>
<td>8. increase fiber-rich food</td>
</tr>
<tr>
<td>9. increase flavonoid-rich food</td>
</tr>
<tr>
<td>10. increase folic acid intake</td>
</tr>
<tr>
<td>Blood pressure</td>
</tr>
<tr>
<td>11. control/reduce blood pressure</td>
</tr>
<tr>
<td>Medications</td>
</tr>
<tr>
<td>12. take aspirin</td>
</tr>
<tr>
<td>13. take β-blockers/reduce heart rate</td>
</tr>
<tr>
<td>14. take statins</td>
</tr>
</tbody>
</table>
been derived from the change in life expectancy in Kaplan–Meyer (Cox, 1972) survival curves and from studies indicating that changes in lifestyle factors, in turn, can alter survival curves. However, unlike previous first-generation health-risk appraisal and computerized risk assessments, our approach utilizes a larger database of epidemiologic evidence to arrive at an estimation of biologic age, which can then be contrasted with the patient’s chronologic age.

We believe this is worth investigating because only a few previous studies have attempted to model age-specific and population-specific benefit expectations that result from risk-factor modification. Benefit in terms of prevention of disease-related morbidity and mortality has been measured as gains in life expectancy (Tsevat et al., 1991; Welch et al., 1996; Wright and Weinstein, 1998). In particular, Wright and Weinstein found gains in life expectancy of 6.2 months from exercise and 28–34 months from quitting cigarette smoking in populations at elevated risk for cardiovascular disease (Wright and Weinstein, 1998). Roizen and Goetz have reported using the concept of what they call RealAge©, a measure of the physiologic improvement that results from intervention affecting health status, in order to translate the present value of healthy choices and motivate behavior change (Roizen and Goetz, 1998). They believe that patients respond to the idea of slowing down the aging process or becoming physiologically younger, and that this model allows both patients and physicians to make more rational health-related choices based on the preference of patients to be as young as they can be. The study described here is the first to apply the concept of biologic age to a sick population (M. F. Roizen, pers. commun.).

The experimental condition: the biologic-age approach

In the experimental group, patients receive their current biologic age based on their risk profile and are able to contrast this with their chronologic age. Patients are presented with an individualized risk-factor profile listing potential areas for behavior change, each of which carries a differential benefit for reducing biologic age. Risk reduction is framed as the opportunity to reduce one’s present biologic age and improve one’s current quality of life. For example, in an otherwise healthy 50-year-old male who has been smoking a pack of cigarettes each day for 25 years, this lifestyle risk makes his biologic age 54 years old. Thus, the net-present value of such a man quitting smoking is approximately a 4-year reduction in his biologic age.

Patients in the experimental intervention group are presented with the numerical value representing the relative potential to benefit from modifying each corresponding behavior. These patients are shown the potential impact that adopting each behavior can have on lowering their biologic age value and are then asked to choose two or three behaviors for modification in order to reduce their biologic age. In addition, patients receive standardized educational materials as do patients enrolled in the control group; however, patients in the experimental arm of the trial receive a biologic-age equivalent for each factor. Thus, although the content of the information about risk is identical for both groups, the presentation of the potential reduction in biologic age that is associated with successful modification of each individual risk factor is unique to our experimental group.

The health risk assessment used in this study is a modified version of the RealAge© computerized risk assessment developed and popularized by Michael Roizen (Roizen, 1998), and a team of scientists and software developers. RealAge© utilizes the concept of biologic age that was first introduced by Sadusk and Robbins (Sadusk and Robbins, 1968). The first-generation of health risk (or hazard) appraisals was developed by the US Centers for Disease Control in the 1970s. Later, the Carter Center, Control Data Corporation and Medical Datamation were among those who developed commercial versions of these early health risk appraisal instruments for large-scale applications in population screening. Several reports (Wagner et al., 1982; Schoenbach et al., 1987) have sought to assess the utility of health risk appraisal. Although these early health risk
appraisal instruments all used some form of biologic age as the outcome of health risks, these instruments were based on databases of relative risk of all-cause mortality that were derived largely from observational epidemiologic studies rather than randomized controlled trials on modifying risks.

RealAge© measures a large number of health risk factors, ranging from cardiovascular risk factors such as smoking, diet and exercise, to varied other risk factors, such as family health history, driving habits, social support and stressful life events, all of which can be used to predict mortality. The algorithm for calculating biologic age is based on the use of relative risk of all-cause mortality values from Kaplan–Meyer survival curves for each factor assessed. Roizen (Roizen, 1998) and colleagues (Rhodes et al., 2001; Roizen and Goetz, 1998) report having drawn these relative-risk values from randomized, controlled studies of the effects of the various risk factors on life expectancy and the benefit of changes in lifestyle factors on survival.

Similar to other health risk assessments, the RealAge© software calculates biologic age by combining data from a person’s health history and current health habits to estimate his or her risk relative to the person’s age cohort. Before an individual answers a single question, his or her biologic age is the same as his or her calendar age. The response categories for each health risk factor in the program are weighted using a relative-risk statistic drawn from published research. Each time the individual provides information regarding health status, these relative risks modify the current estimate of the individual’s biologic age.

With permission from Dr Roizen, we modified the assessment instrument for this study in several ways. First, the assessment of risks was reduced to assess only those modifiable risk factors targeted for intervention in this study, as well as relevant unmodifiable risk factors such as medical comorbidities and family medical history. Table II shows the risk factors and other variables of interest being assessed in this trial, how each variable is measured, and the behavior change goal associated with the risk factors. Second, we capped the maximum biologic age for each chronologic age by a constant conversion factor (C. McCulloch, pers. commun.). This change prevents the estimate of biologic age from being inflated in our study population due to the high incidence of prior cardiovascular events and other medical comorbidities. Third, we updated the nutritional values of all foods listed in the dietary section of the assessment using values drawn from published journal articles [e.g. (Hertog et al., 1992, 1993)] and the unpublished databases of Rimm (E. B. Rimm, pers. commun.).

The control condition: the standard care approach
At baseline, patients in the control condition complete an identical survey of cardiovascular and lifestyle risk factors as those in the experimental group. In the control group, the results of patient survey responses are compiled into an individualized list of risk factors. Patients are then given recommendations to make behavioral modifications based on this list of cardiovascular risk factors. Risk profile information is framed and presented in the standard risk reduction approach as the potential to reduce risk of future negative outcomes. For example, a patient is told that by stopping smoking, he or she may reduce his or her future risk of subsequent events. Patients are then asked to choose two to three areas of risk to target for behavioral modification. In our study, we give patients a set of standard patient educational materials tailored for each risk factor that emphasizes the future value of the specific behavioral change.

The theoretical approach: net-present value economic theory
A variety of theoretical models have guided the development of behavioral and educational interventions in clinical and community settings, including cardiac rehabilitation programs. These include the Health Belief Model (Rosenstock, 1966; Becker, 1974), Theory of Planned Behavior (Ajzen
Table II. Variables under study, measurement and behavior change goals targeted in the intervention

<table>
<thead>
<tr>
<th>Variable</th>
<th>Measurement</th>
<th>Behavior change goal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risk factors</td>
<td>ariety Measurement and Behavior change goal targeted in the intervention</td>
<td></td>
</tr>
<tr>
<td>1. overall physical activity</td>
<td>total weekly kilocalories of exercise</td>
<td>total weekly kilocalories of exercise</td>
</tr>
<tr>
<td>2. aerobic exercise</td>
<td>total weekly minutes of aerobic exercise activities</td>
<td>&gt;30 min/week</td>
</tr>
<tr>
<td>3. strength exercise</td>
<td>total weekly minutes of resistance exercise activities</td>
<td>&gt;30 min/week</td>
</tr>
<tr>
<td>4. smoking</td>
<td>total pack-years to date; current cigarettes per day; time since quitting</td>
<td>stop smoking or continue not to smoke</td>
</tr>
<tr>
<td>5. dietary fish</td>
<td>number of dietary 3-oz servings/week</td>
<td>≥1 serving/week</td>
</tr>
<tr>
<td>6. dietary red meat</td>
<td>number of dietary 3-oz servings/week</td>
<td>&lt;1 serving/week</td>
</tr>
<tr>
<td>7. dietary fiber</td>
<td>daily dietary fiber intake (g)</td>
<td>≥23.2 g/day</td>
</tr>
<tr>
<td>8. dietary flavonoids</td>
<td>daily dietary flavonoid intake (mg)</td>
<td>&gt;30 mg/day</td>
</tr>
<tr>
<td>9. dietary folic acid (folate)</td>
<td>daily dietary intake of folic acid (folate) from diet and vitamin supplements (μg)</td>
<td>600 μg/day</td>
</tr>
<tr>
<td>10. blood pressure (BP)</td>
<td>baseline: current BP in hospital chart; follow-ups: self-report BP (mmHg)</td>
<td>≤120/80 mmHg</td>
</tr>
<tr>
<td>11. low-dose aspirin</td>
<td>daily aspirin intake (mg)</td>
<td>men only: 1 aspirin (325 mg) or baby aspirin (81 mg)/day</td>
</tr>
<tr>
<td>12. heart rate</td>
<td>average heart rate based on medical chart (baseline) or self-report (follow-ups) (b.p.m.)</td>
<td>tight control: &lt; hemoglobin A1C</td>
</tr>
<tr>
<td>13. blood sugar control for</td>
<td>hemoglobin A1C level or self-report estimate of low-average-high blood sugar</td>
<td>male ≤3 mg/day; female ≤52 mg/day</td>
</tr>
<tr>
<td>diabetics</td>
<td></td>
<td>≤9 mg/day</td>
</tr>
<tr>
<td>14. dietary cholesterol and</td>
<td>daily dietary intake from all foods (mg)</td>
<td>age ≥70: CHL &gt; 160, HDL &gt;59/age ≤70: CHL &lt;180, HDL &gt;59</td>
</tr>
<tr>
<td>saturated fat</td>
<td></td>
<td>&gt;7.5 mg/day</td>
</tr>
<tr>
<td>15. serum cholesterol</td>
<td>daily dietary intake per 1000 kilocalories (mg)</td>
<td></td>
</tr>
<tr>
<td>16. dietary polyunsaturated</td>
<td>daily dietary polyunsaturated fat per day from all foods (mg)</td>
<td>male BMI ≤ 25.3/female BMI ≤ 26.99</td>
</tr>
<tr>
<td>fat intake</td>
<td></td>
<td></td>
</tr>
<tr>
<td>17. overweight status</td>
<td>body mass index (BMI) from self-report height and weight; and (for women only) whether weight has ever cycled &gt;10% within 5-year interval</td>
<td></td>
</tr>
<tr>
<td>Mediators</td>
<td>Charlson Comorbidity Index</td>
<td>none</td>
</tr>
<tr>
<td>1. comorbidity</td>
<td>single-item rating of five stages, which is obtained via interview, and is</td>
<td>none</td>
</tr>
<tr>
<td>2. stages of change</td>
<td>assessed for each of the targeted risk factor behaviors listed above</td>
<td>none</td>
</tr>
<tr>
<td>3. self-efficacy</td>
<td>single-item confidence rating for each behavior, which is obtained via</td>
<td>none</td>
</tr>
<tr>
<td>4. depression</td>
<td>CESD (Center for Epidemiologic Studies of Depression)</td>
<td>none</td>
</tr>
<tr>
<td>Outcomes (at 24-month follow-up)</td>
<td>all deaths of any cause prior to 24 months of follow-up will be documented</td>
<td>none</td>
</tr>
<tr>
<td>1. mortality</td>
<td>resting ECG; definite myocardial infarction prior to 24 months of follow-up, based on the 1968 revision of the Minnesota code</td>
<td>none</td>
</tr>
<tr>
<td>2. myocardial infarction</td>
<td>class II–IV anginal symptoms based on the Rose criteria</td>
<td>none</td>
</tr>
<tr>
<td>3. angina</td>
<td>severe Ischemia during non-invasive stress testing</td>
<td>none</td>
</tr>
<tr>
<td>4. severe ischemia</td>
<td>occurrence of a new major focal neurologic deficit that persisted more than 24 h and includes cortical blindness, persistent coma, hemiplegia, hemiparesis, aphasia, sensory changes or hemianopsia</td>
<td>none</td>
</tr>
<tr>
<td>5. stroke</td>
<td>MOS SF-36 (Medical Outcomes Study Short Form-36) Health Survey, which evaluates seven domains of function: physical, social, role, energy, mental, pain and general health</td>
<td>none</td>
</tr>
<tr>
<td>7. subsequent clinical</td>
<td>any subsequent cardiac catheterization, angioplasty/stents or coronary artery surgery during the 2 years of follow-up</td>
<td>none</td>
</tr>
</tbody>
</table>

The ‘behavior change goal’ values are based on the values used in the survey as cutoffs for ‘success’. They are embedded in the databases that guide the program in making decisions about which recommendations are generated for the individual.

CHL, cholesterol; HDL, high-density lipoprotein.
Using economic theory to inform intervention and Fishbein, 1980; Ajzen and Madden, 1986), Social Learning Theory (Bandura, 1977) and the Transtheoretical Model of Stages of Change (Prochaska et al., 1992). All of these theories are cognitive-behavioral models of behavior and each is grounded in the value-expectancy formulation of social and behavioral psychology that was originally developed by Lewin (Lewin, 1935). As useful as these theories have proved to be in guiding intervention efforts, one notable limitation is that none of the theories presents an easily understood and culturally relevant metric by which people can evaluate the utility of making behavioral changes. We believe that without such a metric, people cannot operationalize value expectancy in a clinically or personally meaningful way. It is for this reason that we have turned to a theory of economic behavior to gain new and potentially useful insight into what might better motivate health behavior changes.

The notion of economics as a potentially influential factor in behavioral motivation and behavioral change has only recently caught the interest of health scientists. Several reports have now examined economics as a factor in drug addiction (Marlatt and Kilmer, 1998; Bretteville-Jensen, 1999) and cigarette smoking (Bickel and Madden, 1999; Suranovic et al., 1999). The bulk of this work has been based on Becker and Murphy’s (Becker and Murphy, 1988) theory of rational addiction. The principal concepts from behavioral economics that have been identified by Camerer (Camerer, 1999)—expected utility and risk, exponential discounting of future costs and benefits, maximization of personal utility versus social utilities, and equilibrium—have all been shown in this work to be critical to making choices. Moreover, because such economic concepts have psychological foundations in the behavioral principles of adaptation, loss-aversion, generalized reinforcement and the human preferences for immediacy, they need to be taken into account when designing an intervention to change health behavior. Thus, integrating economic and psychosocial models could be useful to understand cigarette smoking, drug use and other health-related behaviors (Montoya et al., 2000).

At the core of Becker and Murphy’s model is the work on net-present value economic theory of the University of Chicago economist Gary Becker (Becker, 1976). Becker has demonstrated that there is convergent empirical evidence that net-present value economic theory has a strong motivational influence on individuals’ economic decisions and other behaviors. Conceptually, net-present value essentially takes the future value of a product or investment and relates it to what it is worth in present-dollar terms by discounting based on expectations of interest rate, inflation and demand. Another University of Chicago economist, Milton Friedman (Friedman, 1963), was one of the first to recognize the implications of motives and preferences in economic behavior. He suggested that with longer delays, rewards could eventually be discounted into insignificance. This means that the maximum planning horizon for rational investors would not exceed 3 years.

When these concepts are applied to other areas of decision making, they may help to explain why individuals who must make decisions about undertaking complex changes in health behavior tend to undervalue the potential future gain of the behavior change in present terms. For example, the individual who has had angioplasty and must control high blood pressure to avoid subsequent events may experience side effects of antihypertensive medication today. As a result of these immediate side effects, that individual may discount the value of controlling his high blood pressure to zero because the perceived benefit—especially in light of the more immediate side effects of treatment—is a distant benefit. Thus, it becomes critical to explain the health benefits of a recommended behavioral change in present value or present health terms. Moreover, the benefits of a medical treatment or behavior change must be perceived as being achievable in the near future term.

The novel theoretical concept in our intervention is the economic perspective on human behavior that we have chosen to take in motivating and
facilitating health behavior change. The intervention we have developed attempts to motivate behavior change by framing risk reduction as the opportunity to reduce one’s present biologic age. This approach is contrasted with the standard motivation to avoid some future risk of events. Our intervention incorporates proven concepts from well-established models of behavior change, including those of individualized feedback, stages of change and self-efficacy. Unlike other similar interventions, however, it standardizes the value of behavior change based on what economic theory has to say about what motivates people in making decisions about the future. Unlike a previous generation of health risk appraisals, we believe the metric by which we are attempting to communicate risk and the potential benefit of reducing risk—the concept of biologic age—has powerful connotations in a culture that values and reinforces the eternal search for the ‘fountain of youth’.

Figure 1 presents our representation of the logic model for the hypothesized intervention effects we expect our approach to yield. The model suggests that knowing and comparing the biologic age values attached to various health risk behaviors will be effective motivators for behavior change. The age values will demonstrate to patients in understandable terms the relative detriment of various risk behaviors and relative worth of changing them. We have hypothesized that patients will compare the potential age reduction of various health behavior changes and choose to change behaviors with the greatest payoff, i.e. those with the highest relative risk for morbidity and mortality. Patients in the control group will have no information with which to compare the potential benefit of various changes, so they are likely to choose behaviors they feel are easier to change. Overall, then, the control group’s behavior change choices are expected to be more varied and less effective. As a result, the effect of knowing one’s biologic age on clinical health outcomes will be mediated by more effective health behavior changes.

The behavior change choices and successes of both groups will be moderated by the known impact of self-efficacy, stages of change and medical comorbidities, but we expect that the biologic-age approach will significantly enhance self-efficacy and willingness to advance to an ‘action’ or ‘maintenance’ stage of change. Patients’ expectations that they can re-gain some lost vitality and feel ‘younger’ soon, as opposed to extending their lifespan at the end of life, will also serve to motivate maintenance of health behaviors longer for the experimental group, thereby mediating improved long-term health outcomes.

**Conclusion**

While numerous theoretical approaches have been utilized to change health behavior in various patient populations, the application of constructs from behavioral economics to the design of a behavioral and educational intervention in cardiac rehabilitation is novel. This is the first randomized controlled trial to operationalize an intervention approach that applies economic concepts of human behavior to a model of motivating health behavior change in a physiologically compromised population. The second-generation computerized expert-system health risk assessment that is being utilized in this work draws on established principles of health risk assessment to assess diet, physical activity, smoking and other risk factors of the patient population. Unlike previous approaches, however, it calculates and then presents individuals with the unique, explicit net-present value of their future health actions through the application of the concept of biologic age.

There are two particularly novel aspects of this calculation compared to other health risk appraisal instruments. First, the health risk assessment presents the relative-risk effects on predicted mortality in terms of current age units. For example, a 50-year-old person with several risk factors may have a current biologic age of 54. Other health risk appraisals essentially provide the same information, but present it as years added to life, expected age of death or some other metric that too often can be interpreted by the individual at risk as having consequences too far into the future to have
Using economic theory to inform intervention

Fig. 1. Logic model for the hypothesized intervention effect of biologic age on health behavior change.

Impact on his or her current health choices. Second, the biologic-age assessment also calculates the expected change in biologic age within a 3-year horizon for each risk factor, if an individual changed their risk status on that factor through behavior change. Based on net-present value economic theory (and unlike previous approaches to health risk appraisal), these short-term horizon predictions give the individual an achievable goal that has current motivational value. The net-present value approach, thus, attempts to motivate the individual to make health choices based on what economists have learned about human behavior and rational choices by taking the future gain in survival outcome and translating it into net-present years of age. It is in this way that we hypothesize that it helps motivate people by informing each individual of explicit and realistic estimates of the present value of future gains afforded by behavioral change.
We believe that this project promises to provide an empirical demonstration of the potential of net-present value economic theory as a plausible theoretical perspective from which to design interventions aimed at communicating risk, and motivating the adoption and maintenance of complex health behaviors. Such a demonstration could help build an important conceptual and scientific bridge between behavioral psychology, social cognition and learning—all of which have comprised the dominant theoretical perspectives underlying previous efforts to facilitate behavior change in cardiac rehabilitation and other health promotion programs—and behavioral economics.

Acknowledgements

The authors thank Lynn Burrell and Ray Marks for their assistance in preparing the manuscript. This work is funded by a grant from the National Heart, Lung, and Blood Institute, 1R01 HL62161, and is one of the projects of the NIH Behavior Change Consortium.

References


Received on February 14 2001; accepted on May 22, 2001