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The Connection Between Clinical Health Promotion and Health Status

A Critical Overview

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ABSTRACT: The clinical practice of health promotion is based on at least four assumptions: (a) that behaviors increase the risk of certain chronic diseases, (b) that changes in behaviors can reduce the probability of risk of certain diseases, (c) that behavior can be easily changed, and (d) that behavioral interventions are cost effective. Although data support most of these assumptions, the strength of the support is much weaker than is assumed by many psychologists. Thus, the expected health benefits from behavioral programs may not match the enthusiasm espoused by some health psychologists. A major problem in this field is that outcomes are frequently not conceptualized in relation to health. Health status is therefore suggested as the focal point for conceptualizations of health psychology.

Health psychology, behavioral medicine, and behavioral health appear to be the most rapidly growing specialty areas for behavioral scientists (Matarazzo, 1980, 1984). Many of the new health psychologists are specializing in research, and an apparently larger number are offering behavioral medicine and health psychology services. Health promotion is directed toward community and individual programs to promote life-styles that maintain and enhance health (*Healthy People*, 1979). The term *clinical health promotion* describes the offering of health promotion services for fees by individuals who claim to be uniquely trained to provide them. Despite the growth of the field, clinicians and researchers seem not to be communicating well with one another, and it may be a good time to stop and reconsider the expected potential benefit of this emerging area of specialization. This article focuses on some problems in the clinical practice of health promotion. My comments are less relevant for basic research.

I recently learned of a phone conversation in which a psychologist contacted an endocrinologist to announce that diabetes could be "cured" through stress inoculation training. Upon questioning, the endocrinologist was told that plasma glucose level (an indicator of diabetes control) could be affected by stress. The psychologist, who apparently was looking for referrals, knew little else about diabetes. He was unable to specify the role of stress as a cause of diabetes, a regulator of diabetes, or an outcome of the condition. Furthermore, the physician was unable to determine how stress inoculation training would cure diabetes or what role it played in the development of diabetic complications. At about the same time, a San Diego clinical psychologist placed an ad in a local newspaper stating that genital herpes could be controlled through psychotherapy. A symposium at the Western Psychological Association focused on the cost effectiveness of health promotion in the work place. It was concluded that behavioral interventions are highly cost effective, yet no cost-effectiveness analysis was performed in any of the reported studies.

Although these examples are somewhat extreme, they underscore some of the concerns about this rapidly developing specialty. On the other hand, behavioral science clearly has a major role to play in health care (Matarazzo, 1982). Behavioral specialists have been enthusiastically welcomed as participants in health care, and many of their accomplishments have been impressive (Krantz, Baum, & Singer, 1982). On the other hand, psychologists are relatively new to health fields and often ignore the well-established literature that may stimulate greater reflection upon their activities. As an enthusiastic supporter of health psychology and behavioral medicine, I am pleased to see the field developing and thriving. Yet, the art should not precede the science. The rationale behind many applications in health psychology depends on a series of as-yet unverified assumptions.

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Health—The Missing Element

A missing element in the conceptualization of health psychology is the definition of *health*. The conceptualization and measurement of health are rarely discussed in health psychology literature.[1] However, there is an extensive literature on health status with contributions representing medicine, sociology, public health, economics, policy science, statistics, and many other disciplines (Berg, 1973). The National Center for Health Statistics has even established a Clearing House on Health Status Indexes, which provides regular publications on new developments in the field.

Health status is the only reasonable focal point of clinical health interventions. All participants in the health care system have as their goal extending the duration of life and improving the quality of life. These concepts are operationalized in several measures (Bergner, Bobbitt, & Pollard, 1976; Chen, Bush, & Patrick, 1975; Kaplan & Bush, 1982; Kaplan, Bush, & Berry, 1976, 1978). Variables often studied by health psychologists are only important in relation to health status. For example, stress is important because it may affect health. Similarly, lack of exercise, cigarette smoking, high sodium diets, and red meat consumption have been shown to be risk factors for poor health outcomes, but these risk factors should not be confused with the outcomes. They are only important because they bear probabilistic relationships to outcomes. However, heart disease is far from perfectly predicted from known risk factors. This is illustrated rather dramatically in the Pooling Project, a major effort that pooled results from the six best-known prospective studies on prediction of heart disease. Within a defined 10-year period, only about 10% of men with two or more risk factors developed coronary heart disease. Fully 90% of men with two or more risk factors did not develop problems, and more than 58% of those who developed heart disease had only one risk factor or did not have any of the known risk factors (Inter-Society Commission for Heart Disease Resources, 1970). Risk factors provide some probabilistic information but account for a limited proportion of the variance in predicting health outcomes.

In a similar vein, a study that focuses on coping strategies is of little value unless we know that these strategies mediate health. However, few investigations make any attempt to link measured variables to health outcomes. Those that do tend to use rather primitive conceptualizations of health status (DeLongis, Coyne, Dakof, Folkman, & Lazarus, 1982). Baum, Grunberg, and Singer (1982) review psychological, behavioral, and physiological measures of the stress response without even mentioning standard measures of health status.

Developing a scientific approach to health promotion requires some operational definition of health. The problems in developing such definitions are widely discussed in the health literature (Chen, Bush, & Zaremba, 1975; Fanshel & Bush, 1970; Hulka & Cassel, 1973; Stewart, Ware, Brook, & Davies-Avery, 1978). Although there are many difficulties in developing an approach to health measurements, there is also agreement that progress is being made (Jette, 1980; Keeler & Kane, 1982; Sullivan, 1966; Torrance, 1976).

Many different indicators have been used as general health outcome measures. The most suitable are those capable of combining mortality and morbidity. Morbidity (illness) might be best assessed through its effects on the quality of life (Kaplan, 1984). Measures should combine different types of morbidity (Sullivan, 1966) so that both benefits and consequences of treatment can be simultaneously assessed. As Mosteller (1981) noted in his presidential address to the American Association for the Advancement of Science, death rates (mortality) are too crude to measure the efficacy of surgery because many surgical benefits are aimed at improved quality of life. Surgery also poses risks of lowered quality of life in addition to death. Several measures have been proposed to provide comprehensive summaries of the impact of treatment on mortality, morbidity, and the quality of life.

Once health status is defined, we can progress to examining its mediators. Mediators of health status include cognitive and social variables such as coping and social support; environmental factors such as pollution, noise, and exposure (Cohen, Evans, Krantz, & Stokols, 1980); and immune responses (Biondi & Pancheri, 1984). In addition, disease states may also be conceptualized as mediators of health status. Diseases are important because they affect the quality of life and mortality. Cancer is a concern because it affects current quality of life or the probability that quality of life will be affected or premature death will occur sometime in the future. If cancer had no effect on quality or duration of life, it would be of little concern. There may be considerable variability in the way the same disease affects function or quality of

[1] I should note that Stone (1982) conceptualized health psychology very broadly. I recognize the diversity of roles played by health psychologists, and for the purposes of this discussion, I will focus more specifically on roles played by many health psychologists and behavioral medicine specialists—those involving direct patient contact. The discussion will exclude some of the more indirect links between psychology and health. For example, I will not review the very important work on doctor–patient communication (DiMatteo & DiNicola, 1982; Stone, 1982) because its focus on physician behavior is one step removed from outcome. However, once scientific methods for evaluating doctor–patient interactions have been established (see DiMatteo & DiNicola, 1982), we must then place physician behaviors in context by calibrating their effects on health status. If they do not affect patient health status, they are superfluous.

life. For many chronic diseases, there is no medical or surgical cure. However, the major function of health care is to improve or maintain functioning (often through alleviating symptoms). A remedy for a disease would be of little value if remediating the disease did not improve the quality of life or change the probability that quality of life would be affected sometime in the future. Some psychologists have attempted to define health by scaling clusters of disease names (Siverhus, Penrod, Leventhal, & Linz, 1982). These efforts, however, tell us little about the quality of life concerns that are crucial for both patients and physicians.

Many health promotion specialists measure knowledge change, coping, or even blood chemistries (cholesterol and triglycerides) assuming that these variables reflect health. In the following sections, I will argue that these and other assumptions in the clinical practice of health promotion are not uniformly supported by research data. As a result, we are in danger of losing credibility by overenthusiastically announcing the expected benefits of our services. Because the majority of work in health promotion is in the area of heart disease prevention, I will draw most heavily upon research on cardiovascular disease.

Assumptions

Instead of linking their activities directly to health status, many health promotion practitioners make assumptions (usually reasonable ones) about the causal relationship between behavior and health. In this section I will review evidence relevant to four basic assumptions that underlie many of the interventions in health promotion. These assumptions are (a) that specific behaviors create risks for serious illnesses, (b) that changes in risk factors cause changes in health status, (c) that behavior can be easily changed, and (d) that behavioral programs are cost-effective.

Assumption 1

Among the four assumptions to be considered, the belief that behavior is a risk factor for serious illness is most clearly supported by the evidence (Matarazzo, 1982). The evidence is overwhelming that smoking is causally related to lung cancer and alcohol use is related both to diseases of the liver and traffic fatalities. Thus, the Institute of Medicine of the National Academy of Sciences (1982) implicated the role of individual behavior in the cause and maintenance of many disease states.

Although smoking and alcohol use are two behaviors that are clearly related to the development of chronic diseases, much of our current work is directed toward the primary prevention of heart disease through dietary and exercise modifications. In these areas, the assumption that behavior is related to outcome has less empirical support. One of the earliest and perhaps most important statements on the effect of diet in the development of atherosclerosis was presented by Keys and Anderson (1955). Their review suggested that a "luxury" diet with a high percentage of calories from animal fat produced a greater probability of death due to cardiovascular disease. Animal fat contains high concentrations of cholesterol, and dietary factors were believed to elevate cholesterol and cholesterol-bearing lipoproteins in the blood, resulting in atherosclerosis. These diseases, it was believed, could be deterred by reducing the amount of cholesterol in blood serum. Doing so, however, requires an understanding of the causal mechanisms for high serum cholesterol. Many of us have naively assumed that cholesterol in food becomes cholesterol in blood. This is not true; for instance, Keys and Anderson showed that dietary cholesterol has little or no effect on serum cholesterol within the ranges of the ordinary human diet. They also noted that dietary cholesterol has little effect on the development of atherosclerosis. However, human and animal studies did demonstrate that total fat in the diet is predictive of cholesterol levels. Epidemiologic studies, such as the famous Framingham prospective study, have consistently demonstrated small but statistically significant relationships between high serum cholesterol levels and development of heart disease. However, these studies have not shown the association between consumption of saturated fats and high serum cholesterol levels (Kannel, Castelli, Gordon, & McNamara, 1971). Stallones (1983) reviewed six American epidemiologic studies and one British study on dietary characteristics and ischemic heart disease mortality. Within each of these populations, those who developed ischemic heart disease did not consume more calories, more fat, or more cholesterol than those who remained well. In fact, the only differences observed were in the opposite direction. Current investigators are actively trying to gain insight into this problem through dietary manipulation (Carmody, Fey, Pierce, Connor, & Matarazzo, 1982; Matarazzo et al., 1982).

Perhaps less discussed in the psychological literature is the linkage between genetic patterns and cholesterol levels. The great majority of serum cholesterol is produced endogenously. In other words, the body makes its own cholesterol independent of dietary intake. As dietary cholesterol decreases, the body tends to make more in order to compensate (Steinberg, 1979). The most serious cases of hyperlipidemia (excessively high levels of blood lipids and lipoproteins) most likely result from an inherited metabolic disorder (Steinberg, 1979). Cases of hyperlipidemia are usually classified according to a scheme developed by Fredrickson and Levy (1972). This scheme was later adopted by the World Health

Organization and includes six distinct forms of hyperlipidemia. In most cases, these disorders are linked more closely to genetic problems than they are to dietary patterns. In other words, levels of serum cholesterol are regarded as primarily influenced by genetic or constitutional makeup and influenced to a lesser extent by dietary factors (Steinberg & Grundy, 1978).

Debate over the role of diet in lowering high levels of serum cholesterol is ongoing. The majority of the evidence from animal studies, metabolic ward studies, and epidemiologic studies identify a low-fat diet as a potential remedy for hyperlipidemia (Kohn et al., 1978). However, the mechanism by which dietary habits influence blood levels is still not understood (Steinberg, 1979). Furthermore, dissenters continue to argue that there is no relationship between dietary factors and serum cholesterol levels. For instance, some analyses of the Framingham heart study have failed to show significant relationships between diet and serum cholesterol (Gordon, Kannel, & Halperin, 1979). Nichols, Ravenscroft, Lamphiear, and Oslander (1976) measured 24-hour diet recall on a sample of 2,000 persons participating in the Tecumseh heart study and found that serum lipid levels were unrelated to dietary practices.

In the early days of the Framingham heart study, it was argued that total serum cholesterol was the best single predictor of heart disease. Later it was suggested that cholesterol attached to low-density lipoprotein was the best single predictor. This was revised to argue that cholesterol attached to high-density lipoprotein was the best predictor. In the next statement, it was the ratio of high- to low-density lipoprotein. The most recent statement argues that cholesterol in any density is not one of the strongest predictors of heart disease (see Avogaro, 1984, for a review of the chronology). A major Italian study of survivors of myocardial infarctions showed that 75% of the heart attack victims had normal lipid–cholesterol patterns (Avogaro, 1984). Thus, contrary to several decades of consensus, new evidence is beginning to raise new questions about the cholesterol–heart-disease connection.

In summary, the weight of the evidence does suggest that high levels of serum cholesterol cause atherosclerosis. Some evidence further suggests that dietary factors play a role in high serum cholesterol levels. However, the role of dietary factors is probably small in relation to genetic factors. Most epidemiologists believe that diet accounts for a small but significant portion of the variance.

Assumption 2

The second assumption often made by health promotors is what we call the *dynamic* assumption. If there is a correlation between behavior and disease, then modifying the risk-related behavior will reduce the incidence of disease. Although this assumption seems quite plausible, the evidence for it is inconclusive at this point. With regard to smoking, there is convincing evidence that heavy smokers are well-advised to stop smoking (Surgeon General, 1980). The evidence on accidents is obvious and deserves little discussion. Teenagers who drive at high rates of speed while under the influence of alcohol will certainly have lower probabilities of accidents if they modify these behaviors.

Much of the current work in health promotion is directed toward modifying dietary and exercise patterns in order to prevent heart disease. Although this is a worthy effort, it is important to recognize that we have little evidence that these efforts will be effective. At least eight prospective studies have been reported in the literature. The New York Coronary Club study (Christakis et al., 1966) did demonstrate a positive effect of dietary change, but the study did not include a control group. Leren (1966) conducted a prospective study in Norway and found a significant positive effect of dietary change in very small cohorts. Dayton and Pearce (1964) implemented dietary change in the Veterans Administration Hospital in Los Angeles and found significant reductions in coronary deaths. However, these effects were only in younger men and those with initial high levels of plasma cholesterol. Miettinen, Turnpeinen, Elosuo, Paavilainen, and Karvonen (1972) also reported a positive trend in their cross-over design study of Finnish patients. However, their work has been criticized because the population sample changed, and the results were not statistically significant. In sum, at least four studies have reported that dietary changes reduce the incidence of deaths due to heart disease. However, in each of these studies, there was an unexpected finding for total deaths: Mortality averaged over all causes was not affected by the experimental dietary interventions. Reductions in deaths due to heart disease are associated with increases in deaths from other causes—in most cases cancer. This has led some to speculate that low cholesterol diets may actually cause cancer (Pearce & Dayton, 1971). Although this hypothesis is doubtful, it is certainly worthy of further investigation (Cambien, Ducimetiere, & Richard, 1980; Kark, Smith, & Hames, 1980).

At least four studies have reported negative results with regard to changes in dietary cholesterol and deaths due to heart disease. These include a study by the Medical Research Council (1975) and a large international study by Keys (1970). The large and extravagant Keys study reported negative effects for all except those in the youngest age group. Perhaps the most interesting of all studies reported to date is the Coronary Drug Project (Coronary Drug Project Research Group, 1970). The approach in this project was to use a drug known as clofibrate, which can

demonstrably lower serum cholesterol levels. It was argued that this was an important study because the drug produced greater decreases in serum cholesterol than might be expected through diet. Despite reductions in cholesterol levels achieved through clofibrate, consequent reductions in heart disease were not statistically significant. However, the drug may have had toxic side effects, and the study may not have had enough statistical power.

More recently, the National Heart, Lung, and Blood Institute spent $115 million on the Multiple Risk Factor Intervention Trial (MRFIT), which compared groups given or not given counseling to reduce a variety of cardiac risk factors. Although the experimental group showed some modest reduction in risk scores, so did the control group (Neaton et al., 1981). Upon long-term follow-up, heart disease death rates in the two groups did not differ significantly (MRFIT Research Group, 1982).

Because of the inconsistent picture that emerged from the early studies, the National Heart, Lung, and Blood Institute decided to conduct the Coronary Primary Prevention Trial, which was completed after nearly a decade in early 1984 (Lipid Research Clinics Program, 1984a). This randomized experimental clinical trial assigned high-risk men to either a placebo or cholestyramine, which is a drug that is known to significantly reduce serum cholesterol levels. Long-term follow-up was conducted over a 10-year period to determine differential death rates due to heart disease in the two groups. Cholestyramine was successful at lowering cholesterol by an average of 8.5% in the experimental group. Those in the treatment group experienced 24% fewer heart disease deaths and 19% fewer heart attacks than the placebo group. As in other studies, differences between the groups in total mortality were not statistically significant. However, mortality from causes other than heart disease in the treatment group was not attributed to toxic side effects of the medication. Those taking the medicine experienced more accidental or violent deaths than those randomly assigned to the placebo. One of the most important results was that within the treatment group there was a significant relationship between compliance with the advice to take the medication and the development of heart disease. This suggests that the treatment did indeed cause health benefits (Lipid Research Clinics Program, 1984b).

Although the results of the Coronary Primary Prevention Trial are very impressive, it is important to consider them in light of most health promotion efforts. Upon publication of the trial results, many magazines and newspapers attributed the results to diet. In fact, the program did not use diet to control cholesterol levels, because both treatment and placebo groups received the same diet. In the planning stages of the trial, diet had been considered as the intervention rather than medicine. However, preliminary analyses revealed that the expected effect of diet would not be detected, even in a large experimental trial.

Most health promotion efforts are directed toward those who are currently well. The Lipid Research study only included those above the 95th percentile in serum cholesterol. The results tell us very little about the benefits of diet or cholestyramine for those who do not already have significantly elevated cholesterol. Even for those at high risk, the results may be difficult to understand. Although there was a 24% reduction in mortality in the treated group, the actual percentage of patients who died is similar in the two groups. In the placebo group, there were 38 deaths among 1,900 participants (2%). In the cholestyramine group, there were 30 deaths among 1,906 participants (1.6%). Cholestyramine costs about $150 per month (Kolata, 1984). It is a grainy substance with an unpleasant taste that is taken with liquid several times each day. Those with high cholesterol levels must ask themselves whether they would be willing to pay $150 each month for 7 to 10 years for an unpleasant medication that will reduce their chances of dying from 2% to 1.6%.

It is important to reemphasize the effect upon total mortality in these studies. Most of the studies demonstrate that the intervention reduces serum cholesterol and other variables believed to mediate heart disease. In some of the studies, there were actually reductions in deaths due to heart disease. However, when there were reductions in deaths due to heart disease, there were increases in deaths from other causes. Our objective is to extend the duration of life or to improve the quality of life. The successful intervention studies show only changes in the causes of death. It is somewhat unsatisfactory to leave life expectancy unaffected while only influencing the reason listed on a death certificate.

A similar pattern emerges for the risks of obesity. Obesity is a serious risk factor for several important diseases, including heart disease. As a result, we attempt to influence all individuals to reach their ideal body weight or even go below it. Much of the effort is directed toward those who are between ideal body weight and 20% excess. Considerable evidence suggests that the chances of heart disease increase with degree of obesity (Hubert, Feinleib, McNamara, & Castelli, 1983). However, there is very little evidence in terms of total mortality that it is a disadvantage to be moderately overweight. In fact, total mortality rates are the lowest for those between −14% and +15% of ideal body weight (Society of Actuaries, 1959). For reasons not well understood, the mortality rate is actually higher for those in excess of 15% below the ideal body weight (Bray, 1972). Andres (1980) reviewed 16 studies on the relationship between obesity and total mortality and found no evidence that moderate obesity

is a health risk. He suggested that increased cardiovascular deaths found among the moderately obese are compensated for by decreased deaths from other causes. We must caution that the causal ordering for the relationship between low body weight and high mortality is not understood. Low body weight may be a risk, but it is equally as likely to be the result of an ongoing disease.

Even if behavior change is proven effective, we must consider the strength of the effect. Several years ago, the Heart Foundation recommended that Americans cut their egg consumption in half in order to lower cholesterol levels and prevent heart disease. Using data from the Framingham heart study, Vaupel and Graham (1980) performed a series of calculations to estimate the impact of adhering to these recommendations. According to their calculations, cutting egg consumption in half (from an average of five eggs per week to two and one half eggs per week) would, even according to the most optimistic estimates, increase average life expectancy by only 10 days. Totally eliminating eggs from the American diet would increase mean life expectancy for Americans by only 20 days. According to Framingham data, a 48-year-old man has a 60% probability of reaching his 60th birthday. If he ate two dozen eggs per week and completely eliminated them at age 48, the chances of his reaching his 60th birthday increase to 60.5%. Of course, eggs are only one component of a high-cholesterol diet, and there is little risk in developing programs to reduce egg consumption. However, the impact of these programs on life expectancy and morbidity may not be dramatic.

In summary, most physicians and health promotion specialists believe that it is reasonably probable that modifying behaviors associated with the development of heart disease would change the incidence of these frightening conditions. However, current evidence does not always support this position, and it is important to consider the support for different positions in this ongoing debate.

Mortality due to heart and circulatory diseases has decreased more than 30% in the last 30 years. More than 60% of the decrease has occurred since 1970 (Levy & Moskowitz, 1982). Health promotion specialists have proclaimed that the decreases in cardiovascular deaths correspond to warnings by the American Heart Association and to changes in the American life-style. However, others also want to take credit for this decline. Alternative explanations include an increase in the use of cardiopulmonary resuscitation, improved intensive care units, improvements in surgical methods for treating heart disease, and better emergency medical services. Future research should help separate the contributions of these various services (Levy & Moskowitz, 1982).

Assumption 3

One of the major contributions of behavioral medicine is expertise in modifying behavior. Influencing health behavior is a natural point for intervention by psychologists. Social psychologists have developed a detailed and impressive literature on social influence. Behaviorally oriented clinical psychologists have demonstrated that their methods are the most successful approaches known for the management of difficult problems such as obesity. Nevertheless, recent evidence suggests that some health behaviors are very recalcitrant. For instance, Leventhal and Cleary (1980) reviewed the literature on the effectiveness of smoking cessation programs and found that the overall performance was somewhat disappointing. In controlled studies, the probability of influencing a smoker to stop is 20%–30% (Hunt & Matarazzo, 1973). Foreyt et al. (1982) reviewed the evidence for the long-term effectiveness of weight-loss programs. They found that most programs are effective in the short run, but very few have been shown to be effective in achieving long-term maintenance of weight loss. Carmody, Senner, Malinow, and Matarazzo (1980) examined drop-out rates from physical exercise rehabilitation programs for cardiac patients and found that the drop-out rate represented a downward-sloping, negatively accelerating curve. The first 3 months were a critical period during which a substantial number of the participants left the program and presumably stopped exercising. Those who remained in the program appeared to achieve substantial benefits. This suggests that motivation to complete the program is an important variable and may make a substantial contribution to outcome. Schachter (1982) studied smoking and weight loss in a retrospective interview study of members of a psychology department and residents of a small beach community. He found that substantial numbers of individuals had stopped smoking or lost weight on their own without the help of a program. Those who had participated in a formal program were actually less likely to be successful in achieving these behavior changes. Schachter suggests that those who enroll in formal programs may be least capable of effecting changes on their own.

This should not be taken as an indictment of behavioral programs. In fact, they are the most successful of any known approaches to health habit modification. Against this background, it is instructive to consider the efficacy of behavioral approaches to long-time habit change in order to temper our enthusiasm. The Stanford Heart Disease Prevention Program (Meyer, Nash, McAlister, Maccoby, & Farquahar, 1980) has been one of the most successful large-scale behavior change programs initiated to date. Residents of three small California communities participated in the study. In one community, an intensive

media campaign was used. In a second community, intensive instruction was used in addition to the media campaign for some residents. The third community served as a control. The results suggested that there were changes in knowledge as a function of the media campaign, and intensive instruction produced even greater increments in knowledge gained. There was also a small but statistically significant effect for changes in cardiovascular risk scores. However, these changes were explained primarily by changes in smoking behavior (Leventhal, Cleary, Safer, & Gutmann, 1980). Changes for other health risk characteristics were quite modest. Another major intervention study is the MRFIT (1982). In this study, one group of participants received a special intervention designed to modify a variety of cardiovascular risk factors. A comparison group of participants received usual care and follow-up. In comparison to predicted rates, both groups showed some modest decline in cardiac risk factors and in heart disease mortality. However, mortality differences between these two groups were not statistically significant. Although members of the experimental group appeared to benefit, the same trends were observed among subjects in the control group.

In sum, the literature demonstrates that behavioral interventions may have modest rather than strong effects. Further, the long-term success rates for most interventions tend to be disappointing. Many investigators have reported that long-term adherence rates tend to be low and drop-out rates tend to be high. For example, Kentala (1972) conducted one of the best controlled long-term follow-ups of cardiac patients participating in a rehabilitation program. He found that only 39% were still participating after 5 months, and at the end of 1 year, only 13% of the original participants were still enrolled. We can expect behavioral programs to be effective for modifying health habits. However, we must be aware that these programs have limitations and that the long-term efficacy remains to be demonstrated.

Assumption 4

In order for an innovative approach to be utilized, we must demonstrate that it is equivalent or superior to alternative methods for dealing with the same problem. Our recent study showing that patients benefit from a 15-minute preparation for a sigmoidoscopy examination (Kaplan, Atkins, & Lenhard, 1982) has been criticized because the short interventions had only a small but statistically significant impact. One psychologist suggested that we use six 1-hour sessions to prepare the patients for the 2-minute examination. Faced with this choice, anxious patients might prefer the single administration of a tranquilizer.

To evaluate innovative approaches to health care properly, we need cost-effectiveness studies. All treatments involve some costs. First, there are direct costs, such as the fees paid to providers and the costs of hospitalization, medication, and laboratory tests. In addition, there are indirect costs, such as the costs of traveling to obtain care, the costs of time lost from work, and the costs for family members and friends (e.g., costs associated with a wife's taking time off from work to care for her husband). Finally, there are a variety of other costs, such as time costs, that is, the time taken to participate in treatment.

Assessing the effectiveness of health care interventions is also very difficult. As will be discussed later, establishing the effectiveness of a program requires the quantification of its benefits and risks. Some economists argue that benefits should be assessed in terms of the number of dollars a program saves. However, this approach has not been popular among social and health care scientists because it tends to favor programs that benefit the rich. We have argued previously (Kaplan & Bush, 1982) that the most reasonable approach to the assessment of effectiveness is to express the benefits of health programs in terms of the years of life they produce. The years of life figure can be adjusted to take into consideration diminished quality of life. For example, a program that improves quality of life by 10% (or .1 units) on a 0–1.0 scale for 10 years would produce the equivalent of 1 life year. Using a general health outcome measure, it is possible to make comparisons between very different types of health programs.

The conclusion that many types of preventive health services are cost effective often is not supported when exposed to this sort of analysis. For example, many public policies are advocated because they will prevent the loss of lives. In particular, the Occupational Safety and Health Act (OSHA) was designed to create health standards for the work place that would prevent occupationally related deaths and diseases. Yet in some cases, the costs to prevent an accidental death may be unusually high. For instance, benzene, a common component of paint and unleaded gasoline, is known to have a weak significant causal relationship with leukemia. Among 470 male workers exposed to benzene in the work place at a DuPont paint factory in Camden, South Carolina, between 1950 and 1952, 8 had died of cancer by 1975 as opposed to 4 who would have been expected to die of cancer by chance. The level of exposure for these men was 20 parts per million (ppm). The OSHA consultants studied the dose response curve for animal and human studies and concluded that a safe level of exposure to benzene would be as low as 0.2 parts per million. Using a series of somewhat complex calculations, they concluded that the cost of implementing a 2.0-ppm standard would be $3.5 million per life saved. For a 1.0-ppm standard, the costs would be about $29 million for each additional cancer death

averted, and for the 0.2-ppm standard, the costs would be approximately $169 million for each cancer death prevented (Mendeloff, 1980). Graham and Vaupel (1981) examined the costs per death prevented in 57 programs. They found that the cost of preventing a single death ranged from $170,000 to $3 million. The less expensive programs tended to be items such as air bags in automobiles, which came in at less than $300,000 per fatality prevented. Most of the very high-cost programs were related to environmental protection and occupational safety.

To date, most of the highly cost-effective programs have been in the area of primary prevention and screening. For instance, Weinstein and Stason (1976) have found that early detection and treatment of hypertension produces the equivalent of a life year at between $10,000 and $23,000 (in 1984 dollars). Bush, Chen, and Patrick (1973) found that screening of neonates and dietary treatment for PKU produces a well life year at about $7,500.

It must be noted that there are many alternative methods for cost-effectiveness analysis. In addition to the difficulties of determining the effects of programs, assignment of costs is complex and often controversial (Kaplan & Bush, 1982). Nevertheless, the Office of Technology Assessment (1979, 1980) and others (see Kane & Kane, 1982) have considered these problems, and most analysts agree that this sort of process is needed to set priorities and decide between very different approaches to health care.

The major problem faced by health care administrators is that there are many attractive alternatives. Resources are limited, and some choices must be made. Health care costs have grown to consume nearly 10% of the gross national product, and it has become imperative to use scarce resources in the most efficient manner. Cost-effectiveness analysis provides a common metric that can be used to compare alternative uses of resources.

The challenge for health psychologists will be to demonstrate that their services fall within the reasonable range of cost effectiveness. Despite some imperfections in the method, this sort of analysis can be a valuable heuristic that can help us decide which services are productive and which are not. For example, Weinstein and Stason (1976) carefully evaluated the expected benefits of a program designed to increase compliance with therapeutic regimens. Using data from Haynes et al. (1976), they estimated that a behavioral program could improve adherence to antihypertensive medications by 50%. Their next step was to estimate the health benefits of increased compliance using data on the relationship between medicine consumption and blood pressure and the relationship between blood pressure and health status. Then they estimated the cost effectiveness of the behavioral program and demonstrated that even an expensive program is worthwhile because the cost-effectiveness ratio is lower with the program than without it. The cost-effectiveness analyses depend on many assumptions, and it can be shown that varying these assumptions impacts on the cost-effectiveness ratio. Weinstein and Stason (1976) varied the assumptions and found that the compliance program remains cost effective across most assumptions. In other words, assumptions influence the cost-effectiveness estimate, yet under most assumptions the conclusion remains the same: Programs that improve adherence to antihypertensive medications are a wise use of resources.

In other cases, small variations in assumptions can have a major impact on the estimated value of a program. One example comes from estimates of the value of programs for the primary prevention of heart disease. It has become a cliche that it is much more expensive to treat a disease than to prevent it, but there have been few analyses of prevention programs. One exceptionally thoughtful monograph by Berwick, Cretin, and Keeler (1980) systematically explored the cost effectiveness of various options for the prevention of heart disease in children by lowering cholesterol. The approaches included targeted screening and dietary intervention for high-risk children, school programs, and mass media campaigns.

Although it seems advisable to target children for early intervention in heart disease prevention programs, the analysis was greatly affected by assumptions about alternative uses of money. In the cost-effectiveness literature, this is known as the discounting problem. Cost-effectiveness analysis usually requires the discounting of outcomes. The reason that we discount is that the money we have now can be invested. For example, $100 today will become more than $100 next year if it is put in a bank or a wise investment. Hence, if we are purchasing a future benefit, it makes sense to value it at the expected rate that the money would be worth at the time the benefit is delivered. Suppose, for example, that a treatment to prevent a condition was available at a cost of $100. However, the condition would not occur for a year, and we are given the choice of paying for the treatment now or a year from now. Most people would choose to pay later, or they would expect a discount if they paid now.

The choice of discount rate for future benefits can have a major impact on the cost-effectiveness estimates for preventive programs. In the Berwick et al. (1980) study, a program for screening school-aged children for high cholesterol and changing their diets was given an estimated cost per life year saved of approximately $2,400 for males and $1,600 for females if costs and their effects were discounted at the rate of 5% per year. However, the benefits occur many years later than the treatments. Thus, applying a higher discount rate for benefits makes achievement

of the program's objectives appear much more expensive. For instance, using a 10% discount rate for the same program shows the cost effectiveness to be $83,500 for males and $105,000 for females to produce a well year. Most health psychologists have not given consideration to discounting problems. Instead, they simply consider the costs of their interventions and the benefits achieved. Doing so leads to strong overestimates of the benefits of a program. For instance, in the case of the screening for cholesterol and treatment through diet, the costs per life are only a few hundred dollars when no discount to future benefits is applied (Berwick et al., 1980).

Cost-effectiveness estimates are often distrusted because they tend to vary as a function of different assumptions. However, by simulating the cost-effectiveness ratios under a variety of assumptions, it is possible to obtain a range and confidence intervals. In many cases, a program looks cost-effective under a wide range of assumptions, and in others, programs appear as poor bets under any assumptions. Some analyses are sensitive to specific assumptions. Primary prevention programs to prevent heart disease in children, for instance, are sensitive to assumptions about discounting. Even though assumptions affect cost-effectiveness estimates, the effect of varying these assumptions can be explicitly included in the model.

Successful health care providers must be mindful of their objectives. In health care, we should attempt to provide the best possible care at the lowest possible cost. The best possible care is defined as that which has the most beneficial overall effect on health status. This combines both efficacy and side effects. Health care can be defined very broadly, because many variables influence health outcomes. Medical care is one type of intervention that may affect outcomes, but this is true of many other interventions, such as providing safe highways, a clean environment, and so on. All of these approaches are designed to increase the duration of life and improve its quality. Considering costs, risks, and economic benefits of treatments will be necessary to successfully integrate psychological services into the complex and diverse health care system. Excellent discussions of the methodological problems involved in these analyses are available (Office of Technology Assessment, 1979, 1980; Weinstein & Stason, 1977).

To summarize this section, there are many links in the chain between intervention and outcome. The assertion that behavioral interventions are a worthwhile health service requires at least four separate assumptions. First, we must assume that behavior is a risk factor; second, we must assume that changing risk factors produce changes in health; third, we must assume that behavior can be changed; and fourth, we must assume that behavioral interventions are better alternatives than other medical, social, and policy interventions. At each step, there are many important and unresolved research questions. Despite the complexity of the issues, a realistic appraisal of the potential of health promotion programs demands an appreciation of current research literature and the unfinished research agenda.

Conclusions

This overview of the emerging field of health promotion is not intended to be negative. I agree with Matarazzo (1982) and many others who proclaimed that health psychology, behavioral health, and health promotion are among the most important and exciting opportunities that professional psychology has encountered in a long time. Many of the major research questions are unanswered, and the opportunities are plentiful. However, progress is likely to be slow. Professionals from many academic disciplines have become involved in health care, and others have proclaimed that they have the answer to the perplexing problems. However, despite heroic treatment, technological advances, and widespread involvement of many groups in health care, current life expectancy for adults has changed very little since the turn of the century. Most of the change in the life expectancy is attributable to decreases in infant mortality.

Many current activities in health promotion are supported by the best available empirical evidence. Programs to prevent common problems such as heart disease should be continued. My only concern is that we recognize the complexities of the problems and the general absence of definitive evidence on the relationship between behavioral interventions and disease prevention. There is no quarrel with health promotion, only with the promotion of health promotion.

Health psychology is destined to have an impact on roles played by psychologists. Its impact on health will be evaluated as measurement technologies emerge and more data become available. A realistic appraisal of our contribution requires an identification of our objectives, the integration of psychological and other biomedical research, and the willingness to collaborate. The history of health care suggests that if we change the world, we will change it in small increments.

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