Health Risk Appraisal: the Estimation of Risk

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HEALTH RISK APPRAISAL IS A METHOD AND A TOOL that describes a person's chances of becoming ill or dying from selected diseases. The procedure generates a statement of probability, not a diagnosis.

Identifying the variables known to influence individual risk, quantifying their effect and interaction, and constructing algorithms to estimate risk are fundamental to risk appraisal. The accurate estimation of risk presents a challenge to both research workers and clinicians. Existing risk-appraisal instruments have been reviewed elsewhere, for example, by Hettler and coauthors (*1*) and in a 1978 survey of health promotion conducted by the Washington [D.C.] Business Group on Health. We treat here the methodological and research issues relating to the current state and future development of risk estimation.

This article (which was supported by contract No. 282-78-0174 with the Office of Health Information, Health Promotion and Physical Fitness and Sports Medicine of the Public Health Service) is adapted from a background paper prepared for the National Conference on Health Promotion Programs in Occupational Settings. *The authors are with General Health, Inc. Dr. Goetz is Vice-President for Research and Development, Ms. Duff is Vice-President for Programs, and Dr. Bernstein is President. Tearsheet requests to Axel A. Goetz, MD, PhD, General Health, Inc., 1046 Potomac St., N.W., Washington, D.C. 20007.*

Risk Estimation

If one can accurately estimate a person's risk of (*a*) getting specific diseases, both physical and mental (for example, having a myocardial infarction), (*b*) dying from certain diseases (for example, from breast cancer), and (*c*) dying within a defined period (for example, within 10 years), then it is appropriate to ask of what use these estimates are. To adequately answer this question, it is essential to recognize the underlying hypotheses that require substantiation.

Hypothesis 1. Given a particular disease with a known incidence and for which there are identified risk indicators, a change in the prevalence of these risk indicators in the population will result in a change in the incidence of the disease.

There are two versions of hypothesis 1: the full-benefit assumption and the partial-benefit assumption.

Full-benefit assumption: The change in disease incidence resulting from a change in the prevalence of a risk indicator will reflect the full benefit from this change (for example, a nonsmoker and a just-stopped smoker will have the same risk of myocardial infarction).

Partial-benefit assumption: Only partial benefit is derived from a change in the prevalence of a risk indicator (for example, a nonsmoker has less risk of a heart attack than a just-stopped smoker).

Hypothesis 2. Giving people information about their own risk will lead to actions perceived as, and directed at, reducing risk.

Neither hypothesis 1 or 2 has been fully tested. Most recent investigations have focused on hypothesis 2 on the assumption that hypothesis 1 is valid.

The Stanford Heart Disease Prevention Program (*2*) led to reduction in the prevalence of two specific risk indicators—smoking and high cholesterol levels. What is not known is the degree to which the risk of myocardial infarction and stroke per se was reduced in the study population. In the North Karelia Project in Finland (*3,4*), a decline was reported in stroke incidence and myocardial infarction following diet change, lowered blood pressure, and a reduction in cigarette smoking. Detailed analysis of these data may allow some preliminary tests of the partial-benefit assumption versus the full-benefit assumption. The National Heart, Lung, and Blood Institute's Multiple Risk Factor Intervention Trial (*5*) may also yield data useful for deciding among the alternative versions of hypothesis 1. To date, little is known about the effects on disease incidence of changes in certain behaviors and characteristics.

Although hypothesis 1 remains unsubstantiated, pursuit of programs designed to reduce the prevalence of risk indicators in defined populations seems prudent. Hypothesis 2 can only be tested if risk estimates can be conveyed to individuals. However, little is known about the most appropriate methods for transmission of such information. Our purpose here is not to present evidence in support of risk appraisal as a tool for behavioral change but rather to address the present state of risk estimation.

Brief History of Risk Estimation

The concept of risk is an ancient one. Consultation with the Delphic Oracle or prediction of the health of inhabitants of a city from a survey of the city's topography as described by Hippocrates are both early forms of risk assessment. The 18th and 19th centuries provide many examples of increasingly sophisticated studies identifying risk indicators. For example, in 1775 Sir Percival Pott described the association between chimney sweeping and scrotal cancer ("the soot wart"). These observations, however, were fortuitous, not the result of deliberate epidemiologic studies to identify risk indicators. The deliberate search for predictive relationships between risk indicators and the incidence of disease and death and the quantification of the relative risk attributable to these indicators have only recently become possible. This new capability in risk estimation can be attributed to several developments:

1. New statistical and mathematic techniques that support the study of the relationships beween predictive variables and outcome events in health have become available. In the Framingham study (*6*), for example, multivariate logistic models are used, based on the method of Walker and Duncan (*7*), to identify characteristics related to the prognosis following myocardial infarction or angina pectoris.

2. The widespread availability and decreasing cost of computers have made it possible to analyze great quantities of information from large research studies. With modern computer technology, powerful statistical methods can be applied to the analysis of large amounts of data. The computer also supports vital information storage and retrieval functions in the management of longitudinal studies.

3. Studies that lead to the determination of relative risk are expensive. They have been made possible only by the increased availability of public funds for epidemiologic research.

4. Recent advances in understanding the pathophysiology and biochemistry of disease processes have revealed risk indicators, such as high-density lipoprotein and histocompatibility antigens.

Risk Estimation Versus Diagnosis

Medicine traditionally focuses on the diagnosis and treatment of disease. The patient's medical history, physical examination, and laboratory tests are designed to provide clues, signs, and symptoms which, when considered together, will suggest a diagnosis. The intent is the immediate detection and identification of disease. Risk estimation calls for a different approach to information gathering—the collection of data that will permit outcomes to be anticipated over a much longer time frame, one measured in years, if not in decades.

Blood pressure can serve as an example of the distinction between the information traditionally gathered to assess physical status and the information required to estimate risk. In conventional teaching, a blood

pressure of 120/80 mm Hg is considered normal. A blood pressure higher than 140/90 is defined as borderline hypertension and one higher than 160/95, as hypertension. A person who has a blood pressure lower than 140/90 is considered healthy. Diastolic blood pressure is regarded as more important than systolic.

From the perspective of identifying persons at risk of coronary heart disease and stroke, the systolic blood pressure value alone is sufficient for estimating the contribution of the blood pressure level to the risk of these diseases. And what is more important, at levels of systolic blood pressure considered within the healthy range by most clinicians, risk may be substantially elevated, as the chart shows. A person with a systolic pressure of 140 may have a considerably higher risk than someone with a systolic pressure of 110 (*8*).

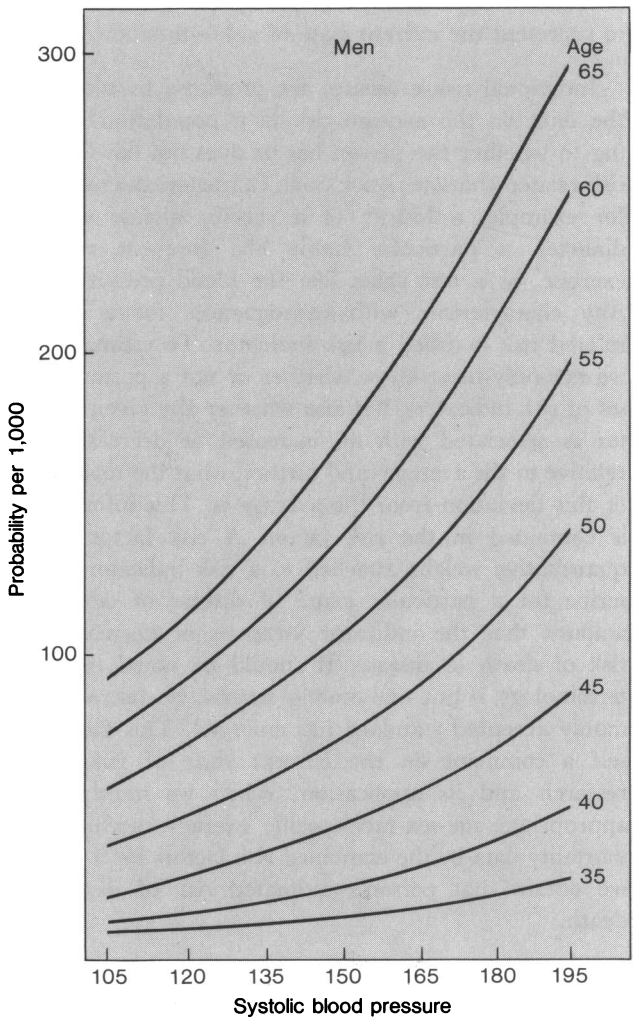
But systolic blood pressure is not by itself a sufficient basis for estimating the risk of cardiovascular disease and stroke. Variables including age, cholesterol levels, cigarette smoking status, type A personality (*9*), high-density lipoprotein levels, and energy expenditure rates through exercise also contribute to cardiovascular disease risk. The interrelationships among these variables are complex and require computations that cannot be performed in most clinical practice settings.

This complexity is one of the reasons that risk assessment and both individual and aggregate group risk profiles have until now been rarely used in clinical settings. This infrequent clinical use, however, is no longer a function of technical or informational deficiencies. Risk can be estimated, and those involved in guiding people toward healthier lives and those having the responsibility for maximizing the health of the employed need to expand their practice patterns to include an accurate estimation of risk.

To incorporate risk estimation into clinical practice, health professionals will have to gather additional information above and beyond the routine medical history. An expanded knowledge of risk indicators and risk factors, as well as computer support facilities, will be required.

The past successes of medicine have been in the prevention and cure of acute illnesses. The value of the public health approach to these conditions has been demonstrated by the reduction of infectious disease through immunization and environmental sanitation. Chronic diseases, which have their acute expression in the final stages, but may begin early in life, are the major challenge of the future. A shift toward their control began in the late 1940s with the Cancer Control Program's use of the Papanicolaou smear to detect early cases of cervical cancer (*10*). Today, screening for existing disease in its early stages is widespread (*11*). Robbins and his associates moved from their experience in the Cancer Control Program to develop an area they called prospective medicine. Their aim was to devise a method that would allow the practicing physician to identify patients at risk—a step that would come before screening for the signs and symptoms of disease. Robbins and Hall's Health Hazard Appraisal, formulated in 1968 (*12*), was the first attempt to estimate mortality risk quantitatively, and it remains the basis for most of the health risk appraisal tools available today. Although the knowledge is not yet reflected in many of the available health appraisal systems, much has been

Probability at 18-year followup that low-risk male subjects in Framingham Study will get cardiovascular disease within 8 years, by systolic blood pressure (mm Hg) at specified ages



NOTE: Low-risk Framingham subjects have a serum cholesterol level of 185 mg per 100 ml, do not smoke, exhibit no glucose intolerance, and have no left ventricular hypertrophy by electrocardiogram.

SOURCE: Reference 8.

learned about the values of risk factors and their interaction, and these advances have helped to improve the art of risk estimation.

General Approach to Risk Assessment

Suppose we were to estimate a person's risk of death within the next 10 years. Not knowing anything about the person, our best estimate would be the average risk regardless of age, race, sex, or any other characteristics that the person might have. This estimate, although expressed in a precise figure, would leave a large amount of uncertainty. If, however, we knew, say, the person's age, this uncertainty would be greatly reduced, but the estimate still would not be very useful. The more we know about a person, the better we can estimate that person's disease and death risks—up to the point at which we have asked all the questions that we know are related to risk. To the extent that a risk appraisal instrument approaches this point, we can consider it to represent the current state of risk estimation.

Individual risk estimates are produced by modifying the data on the average risk in a population according to whether the person has or does not have certain risk-related characteristics. Such characteristics might be, for example, a history of a certain disease such as diabetes, a particular habit like frequent vigorous exercise, or a test value like the blood pressure level. Any characteristic with consequences for a health-related risk is called a risk indicator. To estimate risk, we not only must know whether or not a person has a set of risk indicators, but also whether any given indicator is associated with an increased or decreased risk relative to the average, and further, what the magnitude of this deviation from the average is. This information is contained in the risk factor. A risk factor is the quantitative weight attached to a risk indicator to describe for a particular cause of disease or death the amount that the indicator increases or decreases the risk of death or disease. It should be noted that this terminology is but one among several. So far, no commonly accepted standard has emerged. This fact is itself a comment on the current state of risk-related research and its application. When we multiply the appropriate age-sex-race-specific average morbidity or mortality data by the combined risk factors for a person, we obtain that person's estimated risk of disease or death.

What Risk Estimation Is and Is Not

Implied in the risk estimation method just described is a comparison of the person whose risk we are estimating with groups of persons who in the past have shared the same risk indicator or set of risk indicators. Also implied is the assumption that the presence of the same set of risk indicators then and now has the same health consequences. It is difficult to assess the validity of these assumptions. We are, therefore, limited to predictions of risk in a statistical sense. Risk estimation is not the prediction of a person's future medical history. Even if two persons had an identical set of risk indicators, their fate might be vastly different because of variables that have not as yet been captured in the risk indicator set, such as environmental exposures presently not known to have consequences for risk, pathogenic processes underway, or differences in genotypes. Although with increasing knowledge about risk indicators and risk factors, the range of possible health outcomes for a person can be assessed with ever greater precision, estimates of risk still only give the odds or likelihood of an event such as a myocardial infarction occurring in a group of people with or without a certain set of characteristics (such as smoking, average weight, or low blood pressure).

If we are to make use of the growing knowledge reflected in the epidemiologic and biomedical literature and to obtain more precision in risk estimation, the risk indicators and risk factors used in our risk-assessment instruments must continually be updated. To maintain the precision achieved, the data bases for morbidity and mortality incidence must also be brought up to date regularly.

Mortality and Morbidity Data Bases

To update the data bases for average age-sex-race-specific mortality, Geller and Steele (*13*) suggest a 10-year cycle, since they found relatively little change in mortality rates in U.S. vital statistics data from 1960 to 1970. Moreover, they argue that the changes found were partly due to the random variations that one is likely to see in counts for 1 year only. To cope with such random variation, these authors suggest that death rates be averaged over a 3-year period centered around the years of the decennial census of the U.S. population. It is a matter of opinion whether the errors incurred by a decennial update are tolerable for major causes of death, with rates showing a significant trend upward or downward over time.

A number of authors, for example, Geller and Steele (*13*) and Imrey and Williams (*14*), note the geographic variation in mortality rates and propose to consider area-specific mortality data bases. Such expansion of the data base should await exploration of the proportion of regional variation that is accounted

for by differences in the prevalence of risk indicators. Geographic variation in certain cancers and in liver cirrhosis, for example, may in large measure be due to geographic differences in alcohol consumption. Such variation in habits would be captured through the risk factors modifying average mortality data. If, at the same time, the effect of such variation in habits was represented in the regionalized data base, considerable error could be introduced, because the same effect would be accounted for twice.

Much more problematic than average mortality data is the data base for morbidity incidence. Few assessment instruments are concerned with morbidity risk because the criteria for the definition of cases usually have not been agreed upon and, also, mechanisms for recording cases are often absent. However, at least for some causes, the assessment of morbidity risk is feasible. The Third National Cancer Survey (*15*) is a case in point. For cardiovascular disease risk, the Framingham study has provided incidence data for coronary heart disease, atherothrombotic brain infarction, intermittent claudication, and a number of other diseases.

For most other causes of morbidity, only fragmented data exist, approximating to varying degrees the data required for risk estimation. The extent to which morbidity incidence as reported, for example, in hospital discharge data or in the Health Interview Survey of the National Center for Health Statistics can serve as surrogates in the assessment of disease risk must be examined further.

Davies (*16*) outlines as follows some of the steps involved in the translation of data as reported in the literature into a coherent and internally consistent set of risk indicators and factors:

Guidelines for Estimating Risk Factors

1. Prognostic Characteristics
   a. For selected disease, search the literature for all evidence of prognostic characteristics.
   b. Identify those which have quantitative data on relative risks.
   c. For those with relative risks, find an estimate of incidence in general population by age, sex, race, if possible.
   d. Select prognostic characteristics with adequate quantitative data.
   e. Eliminate any that duplicate or are secondary to a primary characteristic.
   f. Sort into "independent" classes and mutually exclusive categories (e.g. age, smoking category).
2. Derivation of Risk Factors
   a. Convert relative risks to Risk Ratios with lowest risk being 1.0.
   b. Convert Risk Ratio to Risk Factor.
   c. Indicate where assumptions, interpolation, smoothing, averaging or extrapolation are used.
   d. Identify published sources.
3. Combining Independent Risks
   a. Search for evidence of interaction (association) between "independent" prognostic classes.
   b. If none of significance exist, follow probability theory.

To illustrate this approach to estimating risk factors, an example is taken from General Health's Personal Health Profile (Washington, D.C., 1979). This example outlines the generation of risk factors for one risk indicator related to cardiovascular disease (coronary heart disease CHD), namely, high-density lipoprotein (HDL) plasma concentration.

A literature search turned up numerous studies, carried out in a variety of populations in different countries, showing a consistent pattern of a strong inverse relationship between the HDL level and the incidence and prevalence of coronary heart disease.

Incidence rates for coronary heart disease are reported for different levels of HDL in a recent study (*17*). The data for both men and women can be fitted well by a simple regression equation that allows for interpolation of fine intervals of HDL concentration, for example, in increments of 5 mg per 100 ml. From these incidence rates, relative risk (RR) values can be computed for each HDL concentration interval, setting RR = 1.0 for concentrations $\geqq$ 75 mg per 100 ml.

To transform the RR values into risk factors for multiplication of the average incidence data, the proportion (p) of the population at risk for each concentration interval must be known. These data have been summarized for the Cooperative Lipoprotein Phenotyping Study (*18*).

Given n HDL concentration intervals, the risk factor F_i associated with each interval can be computed:

$$F_i = \frac{RR_i}{\sum_{i=1}^{n} RR_i p_i} \qquad ; i = 1, 2, \ldots, n$$

This formula represents the general case of the procedure outlined by Robbins and Petrakis (*19*).

The appropriate factor for multiplication of average CHD incidence can now be obtained if a person's HDL level is known. Before combining the HDL risk factor with other risk factors for coronary heart disease, the degree to which the HDL risk factor interacts with these other factors must be known. Rhoads and associates (*20*), Castelli and associates (*21*), and Gordon and associates (*17*), for example, have supplied evidence on the degree of independence of HDL from other CHD risk factors. The study by Gordon and associates shows the largest amount of variance in a CHD risk factor shared by HDL to be 2 percent (squared

correlation coefficient) for cardiovascular risk indicators included in the Personal Health Profile. High-density lipoprotein concentration is, therefore, considered an independent contributor to CHD risk, and its associated risk factor is multiplied by the combined risk factor for the other CHD risk indicators.

Translating Risk Data Into Risk Factors

It is hardly surprising that difficulties arise in updating health risk appraisal instruments when such updating is based on data from studies conducted and reported for other purposes. To arrive at a risk factor estimate, it is necessary to commit oneself to numbers and procedures that often are more definite than the research results upon which they are based. Assumptions have to be made when actual data would be better, but they are not available. Particularly uncomfortable decisions have to be made in combining risk factors for a given cause of illness or death. Epidemiologic studies frequently are restricted to a consideration of only a small set of risk indicators for a given cause and neither allow for inferences about the risk attributable to each indicator nor present data on the degree to which the indicators involved are correlated or interacting. Even in those studies in which the data collected on risk indicators are comprehensive, the methods of analysis generally used are univariate or bivariate, whereas multivariate analysis would be necessary to account for the interrelationships among variables.

Pooling the results of studies concerned with the same causes is difficult if independent variables are measured imprecisely or in noncomparable ways (for example, alcohol consumption measured by drinks per week versus ounces of alcohol per week or being alcoholic versus being nonalcoholic). Few studies indicate what proportion of their study populations are characterized by a risk indicator or combinations of risk indicators. The generally available sources of statistical information cover only a small subset of the required data on the proportions of the overall population that are in each risk indicator classification. Yet, as we have already shown, such information is essential to the translation of relative risk data into risk factors.

Risk Estimation and Mental Status

Compared with the estimation of the morbidity and mortality risk from physical disease, the current state of risk estimation in the field of mental health is primitive. In the field of physical health, as we have seen, identification of the risk indicators is well advanced—even though identification of the size of the effect and interaction of these indicators is not so far along. In the study of mental health, on the other hand, the processes of defining mental illness and of identifying the risk indicators associated with its various forms are still underway.

Distribution of mental illness. As noted, to estimate the risk of morbidity, we need population-based, age-specific incidence data and data on the nature, distribution, and effect of the risk indicators for the disease in question. Before the risk of becoming mentally ill can be estimated, however, some agreement must be reached on what mental illness is. Fundamental problems exist regarding its definition, description, and measurement. Data based on cases defined in terms of admissions to psychiatric treatment are clear in operational terms (*22*), but they have limited usefulness in incidence estimates, in part because treatment rates vary with the availability of facilities and with public attitudes about their use (*23*).

There have been various approaches to the definition of mental illness. One trend has been to study the presence or absence of signs and symptoms and to attribute diagnostic significance to clusters of signs and symptoms. The other principal approach has been to study functional competency or impairment. Neither approach is entirely satisfactory. With the exception of alcoholism, mental deficiency, and organic brain syndrome, agreement among psychiatrists on the application of current diagnostic classifications is unacceptably low (*24*). The lack of systematic use of standardized measures and the consequent difficulties arising from the results greatly hinder the development of a functional approach to the definition of mental illness.

A critical problem in the study of mental illness in the population has been the failure to develop some baseline data on what mental health is as opposed to mental illness (*25*). Even in the best population-based self-report data, information about the reliability and predictive validity of the measures used is lacking. For example, consider such data as answers to a question about whether the respondent "had or felt near a nervous breakdown" that was posed in the HANES (Health and Nutrition Examination Surveys) of 1960–62 and 1971–75, conducted by the National Center for Health Statistics, and the responses to Dupuy's General Well-Being Schedule (*26*), which was given to a population sample of 6,900 adults in the 1971–75 HANES.

Risk indicators for mental illness. It has been shown that a wide array of biological, social, and cultural

variables may be associated with an increased vulnerability of groups or individuals to mental illness. Mental illness has been found to be differentially associated with social variables, including age, sex, race, and social class. However, both the strength of these associations and their etiological implications remain unclear. A consistent result in numerous studies of race, for example, Rabkin and Streuning (*27*), is that race must be considered in combination with other variables such as income to account for the variance in the prevalence rates for mental illness. Although consistent results on the inverse relationship between the prevalence of mental illness and social class enable us to make some general statements about risk, those statements alone (even supposing the methodological problems were solved) are very limited in their application to risk estimation, since they divide people into such large and undifferentiated risk pools. A greater understanding of cultural variables and the variables that mediate the effects of social class would permit more individualized risk estimates.

The impact of a wide array of social changes on stress and illness has received considerable attention in the literature in the past 20 years. Stressful life events have been shown to play a precipitating role in the onset of physical and mental illness (*28*), but as Rabkin and Streuning point out in their review of the literature, "recent life events account for perhaps 10% of the variance in illness onset, and often considerably less. In practical terms then, life-events scores *taken by themselves* are not effective predictors of the probability of future illness" (*29*).

The effects on a person of exposure to stress would appear to be mediated by several groups of variables. Rabkin and Streuning consider these in three broad categories: (*a*) characteristics of the stressful situation, (*b*) individual biological and psychological attributes, and (*c*) characteristics of the buffering social-support systems available to the person.

Existing evidence suggesting that both social losses such as bereavement (*30*) and social isolation (*31*) increase individual vulnerability to mental and physical illness is convincing. It is the actual strength of that association, rather than its validity, which is now in question, since its strength is what determines risk.

In summary, we are of the opinion that at this time there is insufficient information in the literature to support an estimation of a person's risk of becoming mentally ill. Although there have been indications of an increase in prevalence rates for mental illness across broad social factors and in association with various mediating social and psychological variables, data regarding the strength and interaction of such associations are almost nil.

The aim of most contemporary risk-appraisal instruments is not to estimate the risk of mental illness. Rather the approach has been a normative one—to provide a person with information as to how he or she compares with others on selected dimensions considered to be related to mental health. Thus, several appraisals include a version of Holmes and Rahe's Stressful Life Events Schedule (*32*). The Personal Health Profile enables the user to compare himself or herself with the national norms on the General Well-Being Schedule, which were derived from HANES, and also to make comparisons with similar persons on measures of social support and coping style.

The question as to what is known about estimating a person's risk of becoming mentally ill poses a challenge to today's research and practice in mental health and psychiatry. Consideration of risk in relation to mental status strongly suggests that prospective inquiry and preventive practice should be directed at meeting this challenge.

Outlook for Risk Estimation

The rapid progress of epidemiologic and biomedical research will no doubt help increase the scope and validity of risk estimation techniques. Benefits from research will be maximized if the information needed to improve risk estimation is clearly delineated.

By becoming research tools themselves, health risk appraisal instruments can contribute greatly to the knowledge base for risk estimation. Responses to appraisal instruments can be aggregated to produce large data matrices with several hundreds of variables for many thousands of persons. Such aggregation will permit the definition of a wide variety of subpopulations and the identification and study of risk-related variables. Prospective study of the persons in such a data base, combined with the analysis of data on their morbidity and mortality and health service utilization over time, would significantly increase the contribution of risk-appraisal data bases to the art of risk estimation.

Whether identification of the populations and individuals at risk, coupled with programs that are known to decrease the prevalence of risk indicators, will in fact yield the dividends of healthier, more productive people and reduce expenditures related to illness, disability, and premature death is another, often controversial,

issue. As more risk estimation and risk reduction are carried out, we must make sure that data to answer these questions are collected and analyzed. Once rigorous evaluative studies are carried out, resource allocation based on predictable outcomes will become possible. Such efforts are long overdue.

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