Population Risk, Actual Risk, Perceived Risk, and Cancer Control: a Discussion

Howard Leventhal, Kim Kelly, Elaine A. Leventhal

Given the difficulty of converting population-based estimates of cancer risk into precise statements of individual risk, it is not surprising that (*a*) individual differences in risk perception are at best poorly correlated to the best available determination of "actual risk" and to behaviors to prevent and detect and treat cancer, and (*b*) success in bringing perceived risk into line with actual risk has been limited. These inconsistencies are of concern because individual perceptions of risk are thought to be important motivators of action for the prevention and early detection and treatment of cancer. Following the reviewer's suggestion that risk perceptions are readily influenced by contextual factors, we suggest examining risk perception in a self-regulatory framework in which both risk judgments and motivated action are products of underlying representations of cancer and the self. Self-assessments of risk may access only a part of the data necessary for motivation, whereas motivation to sustain action calls on a larger number of concrete features of the database (symptoms, time loss, consequences). Studies of cancer risk perception can make a major contribution to our understanding of processes involved in self-appraisals and self-management to maximize well-being and to avoid catastrophic disease. [Monogr Natl Cancer Inst 1999;25:81–5]

How do we expect studies of risk perception to help with the control of cancer? To answer this question, it is useful to define the role of "risk" in the ongoing practice of cancer control. For the epidemiologist, risk refers to the probability or likelihood of the occurrence of cancer in specific populations within a given time frame *(1)*. The epidemiologist's definition of risk also involves the identification of factors associated with, and hopefully causally related to, different probabilities of the occurrence of cancer. The biologist searches for inherited and somatic (environmental) genetic factors responsible for failure to control cell division. The objective is to identify the mechanisms (i.e., molecular and physiological processes) underlying specific risk factors. Risk is defined at this level by identifying genetic markers for mutated genes or familial markers (family history) indicative of vulnerability to cancer. The clinician attempts to translate these different sources of information into a probability or risk statement for the individual patient by matching the factors in the patient's risk profile that overlap with the factors defining risk in both population and biological studies. Each approach represents efforts to define "actual" risk. At this time, neither the epidemiological nor the biological data alone or in combination are sufficient to answer the clinician's or patient's question: "Precisely what is the probability that (this specific patient/I) will contract a particular type of cancer at a given point in time, i.e., at 50, 60, or 70 years of age?" In short, there is considerable ambiguity in translating epidemiological and biological knowledge into estimates of actual risk for specific individuals in specific time frames, even with such individualistic risk indicators as those derived from Gail modeling *(2)*.

The presumed value of knowing actual risk is that it can facilitate the control of cancer by encouraging preventive action and early detection and treatment for individuals at high risk. Some routes for cancer control appear to bypass direct communication with at-risk individuals and focus on population risk. For example, establishing and enforcing public policies that reduce exposure to environmental carcinogens can reduce risk for entire populations, as can employment and work rules that require risk-reducing behaviors and penalize risky behaviors. Individual behavior, e.g., participation in screening and preventive actions, can also be influenced by mass media reports of cancer in public figures and by the decision of medical practitioners to introduce screening into annual examinations, the latter involving compliance in the absence of a personal decision. Each of these routes involves influencing behavior. Even efforts that do not directly target at-risk persons must affect behavior by influencing policy makers, and in our political system, this means influencing voters, their elected representatives, and the bureaucracy empowered with enforcing policy. In sum, while these routes change behavior of those at high risk, the means by which they do so, e.g., policy changes and integrating screening into annual medical examinations, do not require a change in perceived risk.

The importance of perceived risk, therefore, is its presumed significance as a motivator of behaviors to prevent, detect, and manage cancer in cases other than those involving public policy or medical or employment requirements for compliance. It is assumed that perceptions of risk are related to motivation to act and to action, and that increasing the match between perceived risk (beliefs) and actual risk (reality) will encourage individuals to initiate and maintain preventive and treatment behaviors at a level that is appropriate to their actual risk and its source. As the papers by McCaul and Tulloch *(3)* and Rothman and Kiviniemi *(4)* in this monograph so ably indicate, all is not well with this assumption. First, it is clear that it is no easier to bring perceived risk in line with estimates of actual risk than it is to extrapolate from epidemiological and biological data to the actual risk of cancer for a given person at a specific point in time. That creating a match of an estimated actual risk to perceived risk is difficult is attested to by data from genetic counseling in which substantial effort in one-on-one settings falls short of creating

Affiliations of authors: H. Leventhal, K. Kelly, Institute for Health, Health Care Policy and Aging Research, Department of Psychology, Rutgers University, New Brunswick, NJ; E. A. Leventhal, Department of Medicine, Robert Wood Johnson School of Medicine, University of Medicine and Dentistry of New Jersey, New Brunswick.

Correspondence to: Howard Leventhal, Ph.D., Rutgers State University of New Jersey, Institute for Health, 30 College Ave., New Brunswick, NJ 08901 (e-mail: howardl@rci.rutgers.edu).

See "Notes" following "References."

perfect matches *(5)*. Second, only a weak relationship exists between perceived risk and behaviors to prevent cancers or to detect and to treat cancer. In our brief commentary, we address issues respecting the empirical support for these generalizations, and then we suggest a framework that can help us to examine both the factors responsible for the match or mismatch of perceived and actual risk and the discrepancy between perceived risk and preventive behaviors. The framework that we propose 1) indicates that the weak association between perceived risk and behavior is to be expected and 2) provides a path for dealing with this expectation in both research and practice.

Major Issues in Risk Perception

Weak Association Between Actual Risk, Perceived Risk, and Cancer-Control Behaviors

McCaul and Tulloch *(3)* review a previously published meta-analysis by McCaul et al. *(6)* that makes clear that the association between measures of perceived risk of cancer and screening behaviors was statistically significant but small at best: an effect size of $r = .16$ between measures of risk and mammography for the 19 studies considered. Family history of breast cancer and breast symptomatology fared better, showing moderate effect sizes for relationship to mammography, .33 for family history (19 studies) and .30 for breast problems, e.g., symptoms. Two sets of factors suggest that these modest effect sizes should be expected. First, the dependent variables under study, e.g., screening behaviors such as mammography and prostate-specific antigen (PSA) tests, and so forth, are not solely controlled by individual volition and, therefore, would not necessarily reflect the individual risk perceptions. As McCaul and Tulloch *(3)* suggest, people see doctors for routine, annual and work-related medical examinations and may be given or asked to take a screening test that they do to fulfill their perceived obligation as a patient. As O'Connor et al. *(7)* indicate in this monograph, however, the rise of consumerism, the movement toward evidence-based medicine, and clinical trials reporting the outcomes of various cancer-control procedures may increase the pressure toward patient involvement in decision making. Indeed, patients may be surprised to find themselves under increased pressure to make decisions, such as decisions with regard to PSA testing, that were formerly left to their physicians. Individuals may be uncomfortable with this new responsibility and believe that they lack sufficient scientific knowledge to fill this decision-making role. The change may encourage sharing information with family members and friends leading to social inputs encouraging screening, once again bypassing risk perceptions. None of these factors, however, is stable. Thus, shifts in the cultural and medical-practice landscape can alter the balance between perceptions of risk and other factors with respect to specific screening and treatment behaviors.

In addition, personal experience with cancer-control procedures, such as mammography, PSA tests, colonoscopy, and the like, can affect how these practices are perceived and can influence the magnitude of their relationship to perceived cancer risk. Women who find mammography embarrassing, uncomfortable, and painful may be less likely to undergo screening. Furthermore, if women believe that some risk results from the available preventive, detection, and treatment options, e.g., belief that radiation from mammography increases risk, screening may be refused regardless of their perceived breast cancer risk. Whereas these beliefs may affect the relationship of perceived risk to practices recommended by medical practitioners, they may not affect individual readiness to engage in alternative or complementary practices. Thus, perceived risk may predict other, nonmedically recommended actions. Investigators may be looking to the wrong outcomes when studying the behavioral consequences of perceived risk.

Second, on the independent variable side, Rothman and Kiviniemi *(4)* indicate risk perceptions are notoriously variable. Judgments of risk change depending on the response format, e.g., verbal, numerical, or ladder, and part of the variability arises because people have a variety of difficulties understanding numerical probabilities [e.g., *(8)*]. Furthermore, as Rothman and Kiviniemi *(4)* point out, the success of efforts to change perceived risk is poor. People do not understand differences between risk over a given time frame and risk as a cumulative estimate, and they treat identical risk ratios differently, depending on the numerical values in which they are expressed, e.g., 1/10 versus 10/100, the latter of which appears to convey greater risk. The variability of risk judgments reduces their value as predictors of subsequent, behavioral outcomes.

Bringing Risk Perceptions in Correspondence With Actual Risk

What can be said about the possibility of bringing risk judgments into line with actual risk and stabilizing these judgments to improve their relationship to selected, behavioral outcomes? The review by Rothman and Kiviniemi *(4)* in this monograph strongly suggests that risk judgments are highly sensitive to contextual factors; we suspect variability in response to context is highly dependent upon the participants. Many examples can be cited; e.g., people who are healthy and less well informed or have not committed to a decision are likely more influenced by context. Perceptions of risk are inflated when physically healthy recipients of risk information are encouraged to think about their own family history of disease and environmental exposures that cause risk *(9)*. Also, the greater the ease of recall of personally relevant risk-increasing factors, the greater the perceived risk, and the greater the ease of recall of risk-reducing factors, the lower the perceived risk *(10)*. Sensitivity to contextual factors increases the variability in risk judgments and lowers their power as predictors of behavior. The data suggest, however, that sensitivity to context varies across issues and populations. For example, the summary of decision aids by O'Connor et al. *(7)* in this monograph makes clear that more than 70% of patients ". . . who have a stated preference at baseline . . ." are uninfluenced by the relatively intensive exposure to information involved in the use of decision aids for choice of treatment. By contrast, half of the undecided are influenced by information and decision aids. The degree to which contextual risk perceptions are more or less modifiable depends on the issue (death from cancer, air pollution, or car accident), the participant population (healthy, newly diagnosed, or advanced stages of disease), and the method of influence.

An Approach to Risk Perception for Research and Practice in Cancer Control

Uncritical acceptance of the two main points raised by the reviewers, i.e., the weak relationship of risk perceptions to behavior and the difficulty of bringing perceived risk in line with actual risk, could lead to premature rejection of risk appraisal

and behavioral approaches for controlling cancer. Indeed, the many factors that our reviewers identify as responsible for these two, general conclusions suggest that future research and practice may prove fruitless if they proceed on the assumption that risk perceptions are stable, have a causal relationship to overt behavior, and must match actual risk to be effective tools for cancer control. Rejecting both the stability and causal properties of perceived risk should not be interpreted to mean that risk judgments are invalid; perceived risk and readiness for action may indeed vary from situation to situation and moment to moment, but they may do so in understandable ways. The key issue is whether we can generate a model for understanding risk perceptions that will be usable for both research and practice.

Judgments as Self-Appraisals

If a woman is asked to evaluate her overall health status or risk of breast cancer, she can reflect on her family and personal medical history and her current physical and psychological function and can then make a self-appraisal as though she were an external observer *(11)*. She could report her judgment of her health status or perceived risk of breast cancer on a verbal or numerical scale. Poorly worded questions and unclear definition of scale points will lower the quality of these data. Reducing these "contaminants" to zero or removing the aforementioned contextual factors causing variability in risk perceptions will not change an essential fact: Self-reports of risk and other similar judgments may be absolutely fine predictors of health outcomes and have absolutely *no* causal relationship to behavior. Self-assessments of health on 5-point scales are excellent predictors of mortality over 5- and even 20-year time frames *(12)*, yet there is no evidence that these assessments are causes of health or risk behaviors, and there is no reason to assume that they cause mortality. Thus, we might identify a group of 65-year-old women who judge their risk of breast cancer in the decade ahead as greater than one in 20, higher than the population risk of women their age, and the future may confirm the correctness of their judgments. However, these judgments may have absolutely no implication or causal relationship to activities to control breast cancer, and only the superstitious would believe that these judgments cause breast cancer. Some of the observations on which these personal risk judgments are based may be reflections of causal variables; others may not (Fig. 1). For example, a high frequency of cancer in the family may reflect an underlying genetic cause; however, emotional distress may directly increase perceived risk but have no relationship to any of the processes causing breast cancer. In short, the observations affecting judgments of risk and health status may or may not be reflections of factors causing the outcomes to be predicted by the judgment *(13)*.

Judgments as Reflections of Motivation and as Causes of Motivation for Action

A covert, mental evaluation or an overt, expressed judgment of health status or risk can represent, therefore, an abstract assessment of the self that is stripped of motivational significance. Neither the covert appraisal of risk nor its overt expression has motivational power; motivation for self-protective action resides in the significance or meanings that underlie these appraisals. The belief system underlying motivation has at least three interrelated belief systems as illustrated in Fig. 1: (*a*) the representation (or beliefs) about the disease, i.e., its identity (label and symptoms), time line (age of occurrence), consequences, per-

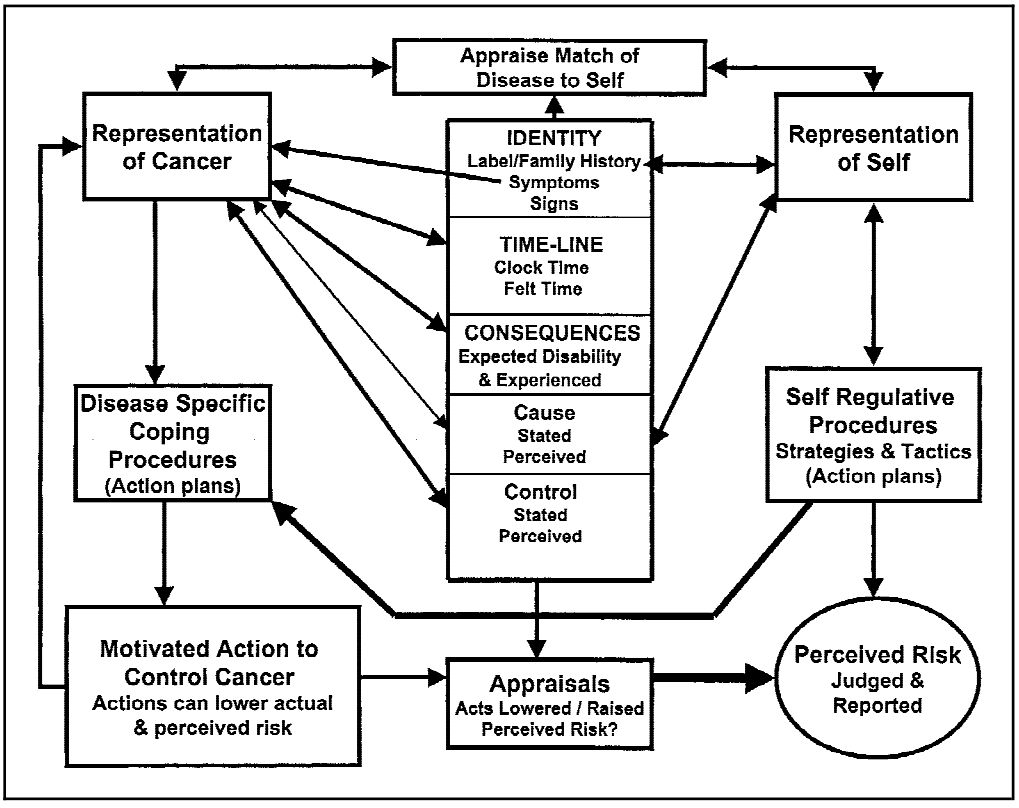

Fig 1. Common-sense perceptions of illness threats. Variables in five domains (identity, time line, consequences, cause, and control) define people's common-sense representations of illness threats. The overlap between the factors defining the representation of cancer (left side of diagram) and those defining the representation of the self (right side of diagram) establishes the self-relevance of the disease. The factors contributing to risk perception, e.g., family history of disease and beliefs about cause, may differ, however, from those needed to generate motivation, e.g., symptoms, time lines (time of disease onset within expected life span), consequences, and beliefs about control. Self-regulation procedures (e.g., general beliefs such as conservation of energy, reducing stress, diet, exercise, etc.) will maximize well-being, and longevity will affect perceived risk and moderate disease-specific coping procedures. Correspondence between perceived risk and motivated action to control cancer will increase with greater overlap between factors involved in the representation of cancer and the self, and this increase can result in a more congenial relationship between general, self-regulative procedures and medical recommendations for cancer control. For example, the general belief that "attacking problems early will facilitate positive outcomes" is more compatible with mammography and prostate-specific antigen testing than are beliefs that a vegetarian diet and avoidance of artificial substances, e.g., medications, are important for well-being and longevity.

ceived causes, and beliefs about its susceptibility to control *(14);* (*b*) the representations of the self, e.g., self-identities, time line (expected years to mortality), causes (factors affecting vulnerability to disease), control (belief in ability to regulate environmental causes), and consequences (perceptions of changes in the self in coming years); and (*c*) disease-specific and self-relevant beliefs about the procedures for prevention, treatment, and recovery (e.g., risks and pain from surgery and radiation therapy). For example, a risk judgment may predict motivation to prevent and control cancer if the factors underlying it include observations of family history of disease and personal behaviors (high-fat diet, smoking) that indicate risk (i.e., the risk is personal), perceived consequences that are painful and distressing, causes that are controllable (i.e., the behavioral components are perceived as changeable), and a time line such that these factors are likely to cause cancer within the individual's lifetime. The representation of the illness and the relationship of this representation to beliefs about the self are the database for the risk judgment and the source of motivation for action. However, the factors necessary for forming a judgment of risk are not identical to those for creating motivation. Risk judgments can be based on family history of disease and the presence of somatic symptoms that create worry and feelings of vulnerability *(15,16).* If other factors critical for motivation are absent, e.g., the time line for the disease and self (e.g., "Cancer won't strike before I die of heart disease"), consequences (e.g., "I can survive breast cancer if I get it"), and the relationship of its consequences to personal values (e.g., "It will not disrupt my family relationships"), judged risk will not be related to risk-reducing behavior.

Action is not solely a matter of the representation of the disease and the self; it is also affected by representations about available procedures for self-protection. Beliefs about the self and strategies for self-protection (self-regulatory procedures) are important moderators of action. People who believe that "I am what I eat" and "Toxins in my food may cause me to get cancer" will control intake to lower risk. If they believe stress causes cancer because, "Stress makes me feel ill," avoiding stress and conserving resources will be selected as routes to control risk. Action will be directed by these self-regulative strategies and associated, specific beliefs about particular procedural tactics for self-protection; e.g., green tea destroys toxins or reducing work commitments will lower stress. Medically recommended actions, e.g., early detection via mammography and treatment via surgery, will be subject to the same appraisal process. Overt action will take place if the procedures recommended for prevention are perceived as relevant to the perceived cause, as acting before the expected onset (time line) of the disease, and as doable within the framework of the individual's resources *(17),* and if they fit the individual's generic, self-regulatory strategies.

A risk judgment unconnected with the set of factors necessary for motivated action will have but weak relationship to behavior unless specific efforts are made to link the judgment to those factors. In short, it is not enough to connect risk perceptions to actual risk: Actual and perceived risk must be connected to the tripartite system (i.e., representations of disease, self, and procedures) underlying behavior. Decision aids and counseling are designed to meet these objectives—i.e., to connect perceptions of disease and treatment risks and treatment outcomes to the self system. The degree to which counseling succeeds is likely to depend on the way in which it addresses the factors involved in the consequences, time frame, causes, control, and identity (symptoms, pain, etc.) of disease; the procedures for disease management; and the relationship of the two sets of beliefs to representations of the self, i.e., personal values and identities, time lines, self-regulatory strategies, etc., discussed in detail in this monograph by O'Connor et al. *(7).* Furthermore, it is critical to remember that each of these sets of factors, e.g., somatic signs of presence of risk, time frames, consequences, and control, is multilevel. That is, each is represented in abstract, propositional forms of language and numbers, in concrete somatic sensations that stimulate fear of cancer and acquired images of family members and friends who suffered with cancer, and in yet more primitive or fundamental forms respecting the vigor of the physical self and its resistance to calamitous disease. The above conditions will ensure the formulation of a specific plan of action, e.g., a scheduled time for preventive action and a place for action, and adherence to a behavior recommended for cancer prevention.

The contributors to our symposium revealed the two major issues arising from studies of risk perceptions in the framework of cancer control: their weak relationship to behaviors to detect and control cancer and the difficulty of improving the correspondence of risk perceptions to actual risk, assuming actual risk can, in fact, be defined. In exposing the wide range of factors underlying these problems, our panelists exposed the contradictions in current data and, more importantly, the vigor and potential for increasing our understanding of risk perception and self-protective action by the theoretical and empirical analysis of perception of risk of cancer. Kurt Lewin is often quoted as saying, "Nothing is so practical as a good theory" *(18).* The growing body of investigations on the behavioral aspects of cancer risk and cancer control suggests a variant of Lewin's quotation, i.e., "There is nothing so useful for good theory as the careful analysis of a practical problem."

REFERENCES

(1) Ahlbom A, Norell S. Introduction to modern epidemiology. 2nd ed. Chestnut Hill (MA): Epidemiology Resources; 1990.

(2) McTiernan A, Gilligan MA, Redmond C. Assessing individual risk for breast cancer: risky business. J Clin Epidemiol 1997;50:547–56.

(3) McCaul KD, Tulloch HE. Cancer screening decisions. Monogr Natl Cancer Inst 1999;25:52–8.

(4) Rothman AJ, Kiviniemi MT. Treating people with health: an analysis and review of approaches to communicating health risk information. Monogr Natl Cancer Inst 1999;25:44–51.

(5) Kelly K, Leventhal H, Baran J, Schwalb M, Toppmeyer D, Much J, et al. Perceived risk and distress in individuals receiving counseling for BRCA1 and BRCA2 mutations. Poster session presented at the annual meeting of the Society of Behavioral Medicine; 1999 Mar; San Diego (CA).

(6) McCaul KD, Branstetter AD, Schroeder DM, Glasgow RE. What is the relationship between breast cancer risk and mammography screening? A meta-analytic review. Health Psychol 1996;18:183–94.

(7) O'Connor AM, Fiset V, DeGrasse C, Graham ID, Evans W, Stacey D, et al. Decision aids for patients considering options affecting cancer outcomes: evidence of efficacy and policy implications. Monogr Natl Cancer Inst 1999;25:67–80.

(8) Shiloh S, Sagi M. Effect of framing on the perception of genetic recurrence risks. Am J Med Genet 1989;33:130–5.

(9) Weinstein N. Why it won't happen to me: perceptions of risk factors and susceptibility. Health Psychol 1984;3:431–57.

(10) Rothman AJ, Schwarz N. Constructing perceptions of vulnerability: personal relevance and the use of experiential information on health judgments. Pers Soc Psychol Bull 1998;24:1053–64.

(11) Bem DJ. Self-perception theory. Adv Exp Soc Psychol 1972:6:2–57.

(12) Idler EL, Benyamini Y. Self-rated health and mortality: a review of twenty-seven community studies. J Health Soc Behav 1997;38:21–37.

(13) Benyamini Y, Leventhal E, Leventhal H. Self-assessments of health: what do people know that predicts their mortality. Res Aging 1999;21:477–500.
(14) Leventhal H, Leventhal EA, Contrada RJ. Self regulation, health, and behavior: a perceptual-cognitive approach. Psychol Health 1998;13:717–33.
(15) Easterling DE, Leventhal H. The contribution of concrete cognition to emotion: neutral symptoms as elicitors of worry about cancer. J Appl Psychol 1989;74:787–96.
(16) Cunningham LLC, Lauren LC, Andrykowski MA, Wilson JF, McGarth PC, Sloan DA, et al. Physical symptoms, distress, and breast cancer risk perceptions in women with benign breast problems. Health Psychol 1998; 17:371–5.
(17) Bandura A. Self-efficacy: toward a unifying theory of behavioral change. Psych Review 1977;84:191–215.
(18) Hovland CI, Janis IL, Kelley HH. Communication and persuasion. New Haven (CT): Yale University Press; 1953. p. 2.

NOTES

Supported by Public Health Service grant AG03501 from the National Institute on Aging, National Institutes of Health, Department of Health and Human Services.