Perspective

Optimistic Biases About Personal Risks

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THE FACT THAT THE PUBLIC OVERESTIMATES THE HARM caused by some problems, such as toxic waste, yet underestimates the number of people harmed by other hazards, such as asthma, is now well recognized (*1*). Less familiar is the consistent, optimistic bias that exists concerning personal risks. When asked about their own chances, people claim that they are less likely to be affected than their peers (*2–7*).

This optimistic bias in "self-other" risk comparisons is easy to demonstrate. If these comparisons are not biased, claims of below-average risk will be balanced by admissions of above-average risk. In a representative sample, the mean response will be "average." Research shows, however, that the mean is usually shifted in the "below-average" direction. A random sample of New Jersey adults (*2*), for instance, yielded the following ratios of "below-average" to "above-average" responses: asthma, 9:1; drug addiction, 8:1; food poisoning, 7:1; influenza, 3:1; lung cancer, 2:1; and pneumonia, 5:1. A significant optimistic bias was found for 25 of 32 hazards in this study.

This bias in comparative risk judgments is robust and widespread. It appears with diverse hazards and samples and with different questions used to elicit the personal risk ratings (*2–7*). Optimistic biases also appear for positive events: people regard themselves as more likely than others to experience financial success, career advancement, and long life (*6*). Pessimistic biases (*8*) are rare.

Some biases occur when people compare themselves with an incorrect norm. The risk of becoming addicted to drugs really is small for most of the population, but it seems that people conclude incorrectly that their risk is far below average by comparing themselves to drug users—a salient high-risk group—rather than to people like themselves who are far more numerous.

Optimism may also arise when ambiguous risk factors are interpreted in a biased manner. People who have not tested their homes for radon gas assert that they are less likely to have problems than their neighbors (*5*). Their most frequent explanation is that their houses are well ventilated. Although high air exchange rates do decrease radon levels, it is hard to consider these explanations unbiased when individuals claiming high ventilation rates outnumber those acknowledging low ventilation rates by a ratio of 26 to 1.

There are also times when people are clearly in high-risk groups but downplay the risk or refer to risk-countering practices of little value. When Bauman and Siegel (*3*) asked gay men to rate the riskiness of their behavior for contracting AIDS, few who engaged in high-risk sex rated their own risk as high. They justified their beliefs by referring to their relatively low number of sex partners or to ineffective precautions, such as inspecting their partners for lesions or showering after sex.

In general, optimism is greatest for hazards with which subjects have little personal experience, for hazards rated low in probability, and for hazards judged to be controllable by personal action (*2*). Optimism is also strong if people think that signs of vulnerability appear early (as they think is true of diabetes, alcoholism, and asthma), so that an absence of present signs means they are exempt from future risk (*2*).

Predicting when optimism occurs does not tell us why it occurs. One idea is that optimistic biases represent attempts to shield ourselves from the fear of being harmed. Most data do not, however, support this view: life-threatening hazards elicit no greater optimism than minor illnesses (*2, 6*).

A second proposal focuses on our desire to be better than other people. Admitting that peers are less susceptible to harm can threaten our feelings of competence and self-worth (*9*). Threats to self-esteem should be particularly strong for hazards like suicide and alcoholism that are thought to be controllable; victimization in such situations is often regarded as personal weakness. This reasoning is consistent with the strong optimism-controllability correlation that exists (*2*).

A third proposal is that optimistic biases are produced by simple cognitive errors. For example, if prevention campaigns create a stereotype of a high-risk individual, people may use this as a standard and conclude incorrectly that their own risk is below average. Excessive extrapolation from the present to the future can also be viewed as erroneous reasoning. Even the association with controllability could be cognitive in origin, because we are more likely to be aware of our own efforts to control risks than others' efforts.

Still, cognitive errors do not provide an adequate explanation for optimistic biases because they do not explain why pessimistic biases almost never appear. The notion that optimistic predictions are actively constructed, rather than arising from simple mental errors, is supported by instances where reasoning is distorted to yield self-serving predictions. For example, people who think they are particularly intelligent rate intelligence as very important to career success, whereas those who think they have a good sense of humor rate this attribute as more important (*10*).

Optimistic biases in personal risk perceptions are important because they may seriously hinder efforts to promote risk-reducing behaviors. If people believe they are not susceptible to AIDS, or less susceptible than others, it may be more difficult to convince them to adopt prudent precautions. There are many positive correlations in the literature between beliefs of personal vulnerability and protective behavior (*11, 12*), but there are also situations where greater perceived susceptibility does not lead to greater action (*4, 12, 13*). There has been little research on these differences.

Although we usually think of biases as maladaptive, several authors have emphasized the benefits of illusions (*14, 15*). Optimism about personal risks is associated with less depression (*16*). Optimism about successful performance leads people to try harder on difficult tasks, so that they really do succeed more often (*14*). A general tendency to be optimistic may even have positive consequences for physical health (*17*).

The benefits of illusions, though, surely depend on the nature of the illusion and the nature of the hazard. Overly optimistic expectations about the value of low-cholesterol diets and exercise may help a heart attack victim sustain these life-style changes and be happier and more productive. But a failure to admit that our smoking, driving while intoxicated, or unprotected sex puts us at risk may keep us from making changes, and this could prove disastrous.

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Progress in Vaccines Against AIDS

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JUST AS OTHER CONCEPTUAL BARRIERS IMPEDING THE RESOlution of the acquired immunodeficiency syndrome (AIDS) crisis have been overcome—such as the nature of its etiology, a diagnostic blood test, and a drug that would have a beneficial effect on the disease—the pessimism shadowing the development of an AIDS vaccine is showing some signs of receding. This is a result of advances on several fronts including: (i) the first examples of efficacy of killed virus vaccines in various animal models, (ii) the identification of important immunological targets on the virus and the infected cell, and (iii) information that humans respond favorably to certain candidate immunogens (*1, 2*). Indeed, as a result of these and other studies, apparent obstacles are being lifted and new concepts on how to approach a vaccine are evolving. Nowhere was this more apparent than at a recent international gathering of scientists at the Annual Meeting of the Laboratory of Tumor Cell Biology (Bethesda, Maryland, 21 to 26 August 1989).

The most striking results were the animal model studies with simian immunodeficiency virus (SIV) by Desrosiers, Murphy-Corb, Gardner, and colleagues (*3–5*), as well as studies with equine infectious anemia virus (EIAV) by Montelaro (*6*). In each example, whole killed virus vaccines were able to delay the onset of disease for a significant period of time (possibly permanently, as the experiments may eventually demonstrate). Notably, protection against the respective diseases caused by experimental challenge with SIV and EIAV could be achieved even in cases where entry of the virus was not prevented. The importance of this development is underscored because it suggests that the same general rules apply to human immunodeficiency virus (HIV) vaccines as have existed for other viruses; namely, that it may not be necessary to completely block infection in order to have a successful vaccine. If some degree of infection with HIV can indeed be tolerated, a vaccine against the virus is much more within reach. It also suggests that some of the studies already performed in chimpanzees with HIV (which were considered failures because infection was not prevented) might have had a more favorable outcome had a disease end point been available.

That is not to say that one should not strive to develop mechanisms to clear the virus. Indeed, some of the examples with both SIV and EIAV apparently were also able to prevent infection (*3, 4, 6*). In this regard, an experiment by Gibbs and Salk, presented at the Vth International AIDS Conference at Montreal (*7*), achieved a surprising result when whole killed virus was administered to two chimpanzees having long-term infections with HIV; the infection appeared to have cleared within the bloodstream. Postexposure immunization in humans with the same preparation has also been studied by Salk and colleagues, although the results are still equivocal (*8*). However, long-term studies, initiated in 1986 and still in progress by Zagury, Gallo, and their colleagues, in which autologous cells expressing HIV antigens were used have resulted in clinical and biological benefit including stabilization or increase of CD4 lymphocytes in some AIDS-related complex (ARC)–AIDS patients (*9*). Taken together, these results suggest that the immune system might be harnessed to protect humans from HIV infection and perhaps to improve the course of the disease.

Although the animal model studies with SIV are of extreme importance for providing concepts and strategies for vaccine development, SIV is not HIV and subhuman primates are not the equal of humans. The SCID-Hu mouse chimeras in which components of the human immune system are able to survive for several months show some promise in this regard (*10, 11*), particularly as infection models for HIV. However, a clear demonstration that these animals can mount an immune response to the virus is lacking. Until that is achieved, their use is limited primarily to testing the protective effects of immune responses generated in other systems through adoptive transfer.

Although instructive during preclinical development, whole virus vaccines do not represent a practical avenue for a vaccine against HIV because of the possibility of infectious particles. For this reason many laboratories are using the reductionist approach which seeks to define the essential components of the virus that would confer protection. Along these lines, advances have been made in several directions including a vast array of neutralizing epitopes, T cell epitopes that are targets for cytotoxic lymphocytes, and regions of the virus envelope that can target antibodies which mediate antibody-dependent cell cytotoxicity (ADCC). In total, nearly 20 such sites have been mapped as linear epitopes (*12*).

The principal neutralizing epitope of HIV is situated within the third hypervariable region, the V3 loop of the external envelope glycoprotein (gp120). Studies by Emini and colleagues have shown that when given with the virus, neutralizing antibodies to the loop can protect chimpanzees against HIV infection (*13*). Nevertheless, the usefulness of this segment in vaccine design appeared limited

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