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neurotransmitters and neurohormones can mediate immune activity and that stress can influence immune response. Stress signals can activate adrenal gland secretion of corticosteroids, which in turn can suppress immune response through action on the thymus.

The now-documented fact of mutual influences among biologic systems has numerous implications. One prominent result is the emergence of a scientific basis for the hypothesized influence of the mind on physiologic functions. Identified pathways now are available to chart and explain the effects of stress, cognitive function, psychosocial events, and the like on health and disease.

However, the existence of pathways for a hypothesized event does not prove that it occurs. Similarly, our inability to cure major diseases such as cancer via the mind does not negate evidence for functional interactions between mental and immune activity.

To the extent that it reflects lack of immune competence, AIDS serves as an *in vivo* analog to a petri dish. If psychosocial factors through now-documented routes to an enhanced immune system could substantively influence health and disease, we would expect prolonged remissions and even cures among patients with AIDS. But that happy result occurs neither regularly nor even on an occasional basis.

An investigative drug that achieved the level of clinical benefit reported for psychosocial events would not be prescribed. Why do psychosocial factors fail to show therapeutic utility even in AIDS, a most fundamentally immune-regulated disease? Why have results in cancer and other serious chronic diseases been so equivocal, inconsistent, and minor in their clinical utility?

Perhaps it can be explained by the direct relationship between the strength of a factor and its demonstrability. The inability to document meaningful psychosocial influences on health may be due not to methodological barriers but to the weakness of those influences. Presumed causal links, such as those between psychosocial factors and disease, that continually elude documentation may well be illusory.

Providing Convincing Evidence for Psychological Influences on Health Constitutes a Tremendous Scientific Challenge

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Lazarus comments on the problems of providing "unequivocal" demonstrations of the effects of psychosocial factors on health. Because I find it difficult to demonstrate anything "unequivocally," I search for something a little less ambitious—convincing evidence. Convincing is, of course, in the eyes of the beholder. Moreover, what would constitute convincing evidence in one domain of health would not in another. However, there are two ingredients that I think apply in judging evidence across domains.

First, there needs to be a theory about the relation between a psychosocial variable and a specific health outcome that is plausible from sociologic, psychologic, and biologic perspectives. For example, you aren't going to convince me that stress contributes to coronary artery disease without providing a reasonable explanation of how this is possible. One might argue that stress is a contributing cause of heart disease because people smoke when stressed, because stress-elicited changes in the central nervous system alter electrical control of the heart, or because stress-elicited increases in blood levels of hormones like epinephrine and norepinephrine alter processes involved in atherosclerosis or thrombosis (Glass 1977; Cohen, Kaplan & Manuck 1992). It is also important that theories of psychosocial influences on health be sensitive to the time courses of both the psychosocial factor and the health outcome. For example, how is it that an acute stressor such as being startled could trigger a clinical event such as sudden cardiac death when coronary artery disease develops over decades? (For the answer see Cohen, Kaplan & Manuck 1992). Evidence establishing the

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plausibility of these theories often comes from studies examining effects of psychosocial factors on a proposed pathway (such as stress-elicited increases in epinephrine) and effects of a proposed pathway on a health outcome (such as elevated levels of epinephrine contributing to coronary artery disease).

Second, there needs to be a methodological mix of well-designed studies that establish the psychosocial variable as a *probable* cause of a health outcome. I use the term *probable* because in many cases it is not possible to conduct human studies that unequivocally establish causality. The methodologies used to study a relation between a psychosocial factor and a health outcome depend on characteristics of both the psychosocial factor and the outcome. However, in all cases the goal is to develop a methodological strategy that increases confidence that the psychosocial factor causes the health outcome. Single studies seldom provide sufficient evidence. Combinations of different methodological approaches and in many cases of both human and animal models are often required. The evidence becomes more convincing to the extent that results from different studies and different kinds of studies converge. The detailed technical issues involved in various methodologies are beyond the scope of this comment. However, it is worth noting that cancer, AIDS, and heart disease, diseases eliciting the most concern among those interested in mind-body relations, are the most difficult to study. This is because these diseases develop over long periods of time prior to the manifestation of symptoms (or in some cases any detectable pathology).

Health is defined by the World Health Organization as an individual's social, men-

tal, and physical well-being. My own estimation is that we are beginning to establish a reasonable core of evidence for psychosocial influences on social and mental health but know relatively little about psychosocial influences on biologically verified physical illness. For reasons suggested by Lazarus and others, establishing psychosocial factors as contributors to physical illness is a very difficult task. However, the ingredients described above as necessary for providing convincing evidence are currently being applied to the study of a number of physical illnesses, especially heart disease and infectious diseases. This work includes plausible models of how psychosocial factors might influence disease outcomes, studies of psychosocial influences on possible mediators, and epidemiologic studies implicating psychosocial factors in disease risk. I think that convincing evidence (one way or the other) will exist in regard to a number of psychosocial predictors of these diseases in the foreseeable future. (See reviews by Cohen & Williamson 1991; Matthews & Haynes 1986.)

Although I am somewhat more optimistic than Lazarus, I basically agree with him. Anyone who tells you that it is easy to demonstrate psychosocial influences on health is either ignorant or lying. Such relations may be "common knowledge" among the public, but providing "convincing" evidence for their validity constitutes a tremendous scientific challenge.

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