

Control and the Epidemiology of Physical Health: Where Do We Go From Here?

Sheldon Cohen
Carnegie Mellon University

Syme (this volume) has eloquently argued for the potential importance of the control concept in the epidemiology of disease. He presents data from psychology, epidemiology, and sociology that support the role of control in the maintenance of health. Moreover, he convincingly argues that the control concept may provide explanations for the influence of other psychosocial risk factors such as Type A Behavior pattern, social class, and social support. I agree with Dr. Syme's basic premise. The existing evidence is provocative and indicates a need for further research to clarify the role of control in disease onset and progression. Where we disagree is on how to proceed from here. Syme proposes a generalist perspective. Specifically, he argues that it is useful at this point to accept broad definitions of both control and disease and continue to search for relations between these concepts. Alternatively, I argue that the appropriate strategy at this point is to move toward greater specificity in defining control, in defining disease outcomes, and in explaining how control would influence disease.

The thrust of my argument is that future research in this area should be driven by highly specified models of the relation between control and health that are both biologically and psychologically plausible. Epidemiologic studies are expensive and time consuming and must be designed to be cost effective. Shotgun approaches to this kind of work are neither politically nor scientifically practical. Well-specified models can maximize the economy of such work by providing guidelines for choosing appropriate samples, measures of control, measures of underlying biologic and psychologic mediation, and disease outcomes. Models that provide

convincing biologic explanations of why we would expect control to influence a disease process also provide the ammunition necessary to convince the medical community of the importance and the validity of this work.

At minimum, the development of models linking control to disease pathogenesis requires (a) distinctions between various conceptualizations of control, especially in regard to their temporal stability; (b) a strong rationale as to why we think a specific conceptualization of control should influence a disease process; and (c) an understanding of disease processes that allow specific predictions in regard to what diseases (or disease stages) should be influenced by control.

The remainder of this discussion elaborates the minimum requirements for modeling control influences on health maintenance and disease as outlined earlier. I propose a dimension on which to differentiate control and suggest both psychologic and biologic pathways that could link control to various disease processes. My intent is not to provide all distinctions between control concepts or all plausible models linking control to health or disease, but only to exemplify some reasonable approaches to the problem. Hopefully, this discourse will motivate others to develop more sophisticated and detailed models of controls relations to a range of disease processes.

DIFFERENTIATING CONTROL CONCEPTS

There are a number of dimensions on which control concepts could be distinguished (e.g., objective vs. subjective, global vs. specific, and environmental vs. personal). Clarification of the differences between various control concepts on these and other dimensions would be extremely useful in modeling the relation between these concepts and disease. However, for demonstrative purposes, I distinguish control concepts only on the basis of temporal stability. The issue here is whether a measure of control taps an enduring personal or environmental characteristic or whether it taps a short-term state. Examples of concepts that are likely to be very stable over time include locus of control and attributional style. Examples of relatively unstable concepts are self-efficacy and perceived situational control. As discussed in detail later, the plausibility of control-disease models rests to a great extent on the stability issue. In short, we need to know the minimum time that control (or lack of control) must last to influence the disease process under consideration.

WHY WOULD WE EXPECT CONTROL TO INFLUENCE DISEASE PATHOGENESIS?

This section focuses on the ways in which control may influence health and disease. The models I propose are concerned only with the causal role of control and hence do not address reserve causation (disease causing control) or spuriousness (another factor such as age or social class simultaneously influencing control and disease). The exclusion of these alternative causal pathways is not intended to reflect any hypotheses about their existence.

At the most elementary level, it can be posited that control is linked to physical disease either through its influence on health practices that increase (or decrease) risk for disease or through control triggered biological responses that affect disease onset or progression (e.g., Krantz, Glass, Contrada, & Miller, 1981). Examples of health practices that may be influenced by control and are implicated in various disease processes include smoking, diet, drinking alcohol, exercise, and sleeping. Although a number of biological systems may provide links between control and disease, neuroendocrine pathways provide the most plausible links in this case. The secretion of hormones such as cortisol, norepinephrine, and epinephrine are known to be triggered by psychological states and are suspected of playing a role in both the pathogenesis of immune mediated diseases and of coronary artery disease.

We can also ask why a particular conceptualization of control would influence the behavioral and/or biological pathways to disease. This question has led to the development of two generic models of the influence of control on disease pathogenesis (Cohen & Edwards, 1989). The main-effect model suggests that control has a beneficial influence irrespective of whether persons are under stress. The stress-buffering model proposes that control operates by protecting people from the potentially pathogenic influence of stressful events. Some conceptions of control are likely to operate through main-effect mechanisms, while others operate through stress-buffering mechanisms.

Main-Effect Model

The main-effect model postulates a general beneficial effect of control. The pathways through which such an effect could occur depends to some extent on what is controllable (e.g., information or access to material goods). Some of the mechanisms outlined later assume that control implies the ability to manipulate a specific resource, whereas others address

more general psychological consequences of having or not having control over important outcomes. As apparent in the discussion, the applicability of each mechanism depends on the specific conceptualization of control under consideration. An overall summary of possible main effect mechanisms is provided by Fig. 1.

Information-Based Models. Having control can imply access to good sources of information. Accurate information could influence health-relevant behaviors or help one to avoid stressful or other high-risk situations. Examples of beneficial information include information regarding access to medical services or regarding the benefits of behaviors that positively influence health and well-being. Appropriate information could also aid in avoiding stressful life events, or in avoiding exposure to infectious or carcinogenic agents (Berkman, 1985; Cohen & Syme, 1985).

Tangible Resource-Based Models. Control can imply access to tangible and economic services that result in better health and better health care; for example, the ability to procure food, clothing, and housing that operate to prevent disease and limit exposure to risk factors or to access health care that prevents minor illness from developing into more serious disease.

Affect and Self-Esteem Models. Feelings of control may generate generalized positive affect, a sense of predictability and stability in one's life situation, and a recognition of self-worth. These positive psychological states are presumed to be facilitative because they result in increased

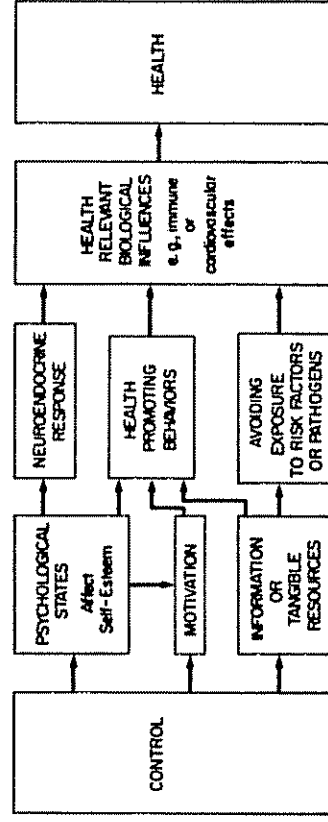


FIG. 1 Main-effect model of the psychological and biological pathways linking control to the onset and progression of disease. All indicated paths move in one causal direction. The exclusion of alternative paths is not intended to reflect any hypotheses about their existence.

access to it) reduces the evaluation of potential threat or harm, the event would be appraised as less threatening and/or harmful and hence the risk of illness decreased. A reduction in stress appraisal would be presumed to reduce negative affect, negative health behaviors, and concomitant physiological reactivity. Information about effective means of coping may also increase appropriate coping efforts.

Affect and Self-Esteem-Based Models. The ability (or perceived ability) to control a stressor often increases feelings of positive affect and self-esteem. As noted earlier, such feelings may influence health through increased motivation to perform health behaviors or through suppression of neuroendocrine responses. Feelings of control that are not specific to coping with the stressor itself may also facilitate coping with the negative affective consequences of stress, or limit generalization of negative affective or cognitive responses to other situations (Abramson, Seligman, & Teasdale, 1978; Cohen, Rothbart, & Phillips, 1976; Seligman, 1975).

Tangible Resource-Based Models. Control can imply the ability to access tangible or economic services. If such resources provide appropriate coping responses to a stressful event, they would reduce the probability of the event being appraised as threatening or harmful and hence reduce the behavioral and affective concomitants of such an appraisal. Again, the mere perception of the availability of resources may operate without actual receipt of help. Tangible resources could also help resolve specific (tangible, related) problems after a stress appraisal is made.

WHAT DISEASE PROCESSES WOULD WE EXPECT TO BE INFLUENCED BY CONTROL?

As discussed earlier, control influences on health and disease are presumably mediated by behavioral and biological factors that have been implicated in a range of disease processes. For example, negative affective responses associated with helplessness could influence cancer and infectious diseases through neuroendocrine-elicited immunosuppression (Seligman, 1975; Sklar & Anisman, 1979; Visintainer, Volpicelli, & Seligman, 1982) and coronary artery disease through neuroendocrine-elicited facilitation of coronary artery occlusion (Glass, 1977). Similarly, cigarette smoking provides a good example of a health practice that has been implicated in multiple diseases including CHD, stroke, lung cancer, and upper respiratory infections (Matarrazzo, 1982). It is clear, however, that generic models that assume psychosocial influences on susceptibility to

motivation to care for oneself (e.g., Cohen & Syme, 1985), or because they suppress neuroendocrine response (Cassel, 1976). On the other hand, it might be argued that relations between control and health are primarily attributable to those suffering from insufficient levels of control. For example, a lack of control may result in less motivation to care for oneself, or in affect mediated elevations in neuroendocrine response (Cohen, Evans, Stokols, & Krantz, 1986; Seligman, 1975).

Stress-Buffering Models

The stress-buffering model posits that control "buffers" (protects) persons from the potentially pathogenic influence of stressful events. The model assumes that stress puts persons under risk for physical disease. This risk is presumed to occur either because stress triggers neuroendocrine response, arterial flow disturbances implicated in coronary artery disease (CAD), or because behavioral adaptations to stress often include detrimental health practices (e.g., smoking and poor diet; Cohen, Kaplan, & Manuck, in press). Control operates in these models by decreasing the probability that situations will be appraised as stressful, by directly dampening neuroendocrine responses to stress, by preventing persons from adopting unhealthy behavioral adaptations, or by influencing the ability to control stress elicited emotions. A summary of stress-buffering mechanisms is provided in Fig. 2.

Information-Based Models. Control can imply having or having access to information about the nature of the potential stressful events or about ways of coping with those events. To the extent that this information (or

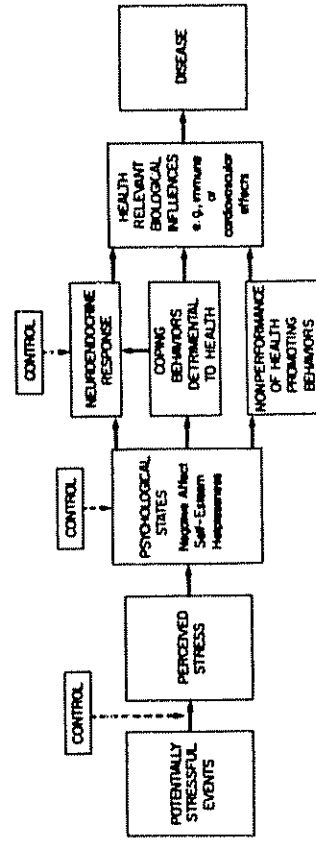


FIG. 2. Stress-buffering model of psychological and biological pathways linking control to health and well-being. All indicated paths move in one causal direction. The exclusion of alternative paths is not intended to reflect any hypotheses about their existence.

all or most diseases are gross oversimplifications. Some diseases (and disease stages) seem especially susceptible to behavioral and biological mechanisms linked to psychosocial factors such as control, whereas others are not (Cohen, 1988).

Two questions should be addressed in deciding whether a particular disease is susceptible to control influence. First, is it plausible that behavioral and biological processes presumed to be influenced by control are important precursors of the disease? For example, although modulations of neuroendocrine response and/or changes in health practices are assumed to play a role in the pathogenesis of some diseases (e.g., coronary artery disease), their influence on others (e.g., meningitis or Hodgkin's disease) is less clear. Second, is the conception of control under study temporally stable enough to provide an exposure that is long enough to influence the pathogenesis of the disease under consideration (Cohen et al., in press; Cohen & Matthews, 1987)? The answer to this question depends on the relation between the temporal stability of a particular conception of control and the developmental course of the disease under study. Plausible models assume either that (a) the concept of control under examination is relatively stable over the period of disease development, or (b) a short-term exposure to a particular level of control is sufficient to influence the disease process.

An example of the importance of temporally matching control stability and disease development is provided by the relation between control and the development of atherosclerosis. Locus of control is a temporally stable conceptualization of control, and CAD has a very long and slow course of development (clinical disease generally requiring five decades). Hence, there is a reasonable match here between stability of the control measure and the temporal characteristics of disease pathogenesis. That is, exposure to relatively lower levels of locus of control lasts over the period of disease development. Conceptualizations of control with much shorter temporal stabilities such as self-efficacy or perceived situational control would not be plausible predictors of CAD pathogenesis, although they may be plausible predictors of diseases with very short-term developments like colds and influenza.

It is possible, however, to propose plausible models of slow-developing diseases that focus on control measures with shorter stabilities. For example, we have been discussing control processes that may influence the development of atherosclerosis. As noted earlier, this disease develops over many years. However, for those with clinically significant coronary artery occlusion, a coronary event can occur in relation to a psychosocial factor lasting for a relatively short period. Consider, for example, modeling myocardial infarction (MI) incidence. An MI is a common form of heart attack caused by the death of heart tissue. MIs occur when blood

flow through the coronary arteries is interrupted and as a consequence insufficient oxygen reaches the heart muscle. Assume that persons with severe coronary artery occlusion are more likely to manifest this disease in an MI if they experience stress (e.g., Glass, 1977). A severe stressor might trigger the onset of an event, for example, by causing an artery to spasm (Cohen et al., in press). In this case, perceived control over the specific stressor, a control concept with less temporal stability, may be important if it is stable over the course of stressor exposure and operates to buffer persons from stress at the trigger point. In our example, perceived control might ameliorate the experience of stress and thus prevent the stressor from triggering artery spasm. Hence perceived situational control might protect such a person from stress-triggered disease progression.

Finally, there are plausible (but speculative) control-disease models in which short-term exposures to a particular control level triggers the onset of the development of a disease with a long (slow) developmental course. Take for example the possibility that sudden and severe breaches in control such as those often produced by severe and uncontrollable life stressors (e.g., death of a loved one) may be associated with immunologic changes that set a given disease in motion. Hence, a short-term lack of control may hit with sufficient impact to produce a dramatic but short-lived compromise in immune functioning triggering the onset of a disease like cancer that may then be self-perpetuating.

In summary, the question of whether a particular disease is susceptible to control influence depends on (a) whether the conceptualization of control under consideration affects processes that influence disease pathogenesis, (b) on the temporal stability of the control concept, and (c) on the nature and time course of the pathogenesis of the disease.

In short, temporally stable conceptions of control are plausibly related to the onset and progression of diseases with both long- and short-term pathogeneses, and less-stable control conceptions like perceived situational control are plausibly related to both diseases with short-term developments and diseases for whom stressors may act as triggers of progression or onset. Future work on diseases with plausible links to both stable and unstable conceptualizations of control is a high priority at this time. High-prevalence diseases such as infectious diseases (ranging from common upper respiratory infections to AIDS), and cancer provide opportunities in this area that have the potential for both theoretical and practical contributions.

CONCLUSION

How do we proceed from here? We can continue to examine whatever measure of control is easily available, of interest to a study collaborator or consultant, or short enough to include in an epidemiologic questionnaire.

(This comment is unfair to Dr. Syme but is closer to how things are actually done than we would like to believe.) Results from such studies can then be reviewed for consistencies and inconsistencies and a pattern may emerge. Alternatively, future research can be driven by hypothetical propositions based on plausible biologic and psychologic models of the link between control and disease. I have argued that the creation of such models requires a greater specificity in regard to the definition of control and the disease process under study.

Do we need separate models of the relation between control and disease for each individual disease and disease stage? One apparent difference between Dr. Syme's position and my own is his emphasis on general processes applying to all diseases and my emphasis on specifying relevant processes for each disease or disease stage. To some extent our differences on this point are illusory. I agree that common psychologic and biologic pathways can influence multiple disease processes, and hence it is inevitable that certain models will apply across a range of diseases. For example, elevations of secretions of the catecholamines epinephrine and norepinephrine over a prolonged period of time may influence the development of coronary artery occlusion as well as suppress immune function hence possibly increasing susceptibility to infectious and neoplastic diseases. However, not all diseases fit any single pathway model (whether behavioral or biologic). Moreover, the importance of common pathways such as the release of catecholamines varies widely among disease processes. In some cases the common pathways may be an important influence on the etiology and/or progression of a disease, in others the influence is clinically trivial. Finally, as outlined earlier, different disease processes have different temporal courses. Although conceptually mediated by the same processes, they are differentially influenced depending on the temporal stability of the control concept under consideration. In summary, some models may represent processes that influence more than one disease or disease stage, but before assuming such etiologic commonality we need to carefully consider a range of factors influencing the likelihood of a relation between specific definitions of control and specific disease processes.

ACKNOWLEDGMENTS

Preparation of this chapter was supported by a Research Scientist Development Award to the author from the National Institute of Mental Health (K02 MH00721).

REFERENCES

- Abramson, L., Seligman, M. E. P., & Teasdale, J. (1978). Learned helplessness in humans: Critique and reformulation. *Journal of Abnormal Psychology, 87*, 49-74.
- Berkman, L. F. (1985). The relationship of social networks and social support to morbidity and mortality. In S. Cohen & S. L. Syme (Eds.), *Social support and health* (pp. 241-262). New York: Academic Press.
- Cassel, J. (1975). Social science in epidemiology: Psychosocial processes and stress theoretical formulation. In E. L. Struening & M. Guttentag (Eds.), *Handbook of evaluation research* (Vol. 1, pp. 537-549). London: Sage.
- Cohen, S. (1988). Psychosocial models of the role of social support in the etiology of physical disease. *Health Psychology, 7*, 269-297.
- Cohen, S., & Edwards, J. R. (1989). Personality characteristics as moderators of the relationship between stress and disorder. In R. W. J. Neufeld (Ed.), *Advances in the investigation of psychological stress* (pp. 235-283). New York: Wiley.
- Cohen, S., Evans, G. W., Stokols, D., & Krantz, D. S. (1986). *Behavior, health and environmental stress*. New York: Plenum.
- Cohen, S., Kaplan, J. R., & Manuck, S. B. (in press). Social support and cardiovascular disease: Underlying psychologic and biologic mechanisms. In S. A. Shumaker & S. M. Czajkowski (Eds.), *Social support and cardiovascular disease*. New York: Plenum.
- Cohen, S., & Matthews, K. A. (1987). Social support. Type A behavior and coronary artery disease. *Psychosomatic Medicine, 49*, 325-330.
- Cohen, S., Rothbart, M., & Phillips, S. (1976). Locus of control and generality of learned helplessness in humans. *Journal of Personality and Social Psychology, 34*, 1049-1056.
- Cohen, S., & Syme, S. L. (1985). Issues in the study and application of social support. In S. Cohen & S. L. Syme (Eds.), *Social support and health* (pp. 3-22). New York: Academic Press.
- Glass, D. C. (1977). *Behavior patterns, stress, and coronary disease*. Hillsdale, NJ: Lawrence Erlbaum Associates.
- Krantz, D. S., Glass, D. C., Contrada, R., & Miller, N. E. (1981). *Behavior and health* (National Science Foundation's second 5-year outlook on science and technology). Washington, DC: U.S. Government Printing Office.
- Matarazzo, J. D. (1982). Behavioral health's challenge to academic, scientific and professional psychology. *American Psychologist, 37*, 1-14.
- Seligman, M. E. P. (1975). *Helplessness: On depression, development, and death*. San Francisco: Freeman.
- Sklar, L. S., & Anisman, H. (1979). Stress and coping factors influence tumor growth. *Science, 205*, 513-515.
- Visintainer, M. A., Voipicelli, J. R., & Seligman, M. E. P. (1982). Tumor rejection in rats after inescapable shock. *Science, 216*, 437-439.