from dualism. Psychologists interested in personality and cancer should follow the molecular oncology literature, and molecular oncologists should come up to speed on the relevant psychological variables.

Another specific point is that autonomy, as Eysenck presents it, should be compared to control, perceived and real. It seems to us that these constructs can be reconciled in ways that will make use of already available data on control and stress. If there are underlying psychological truths related to health status, then it would certainly help to keep our terminology consistent. Also, the Eysenck position should address the effects of a "worsening" of the personality types in addition to the positive changes or improvements that he notes as important.

As a final note, we should explain the title of this commentary. If Eysenck is right about personality type, disease state, and the implicit value of reducing disease risk, then what would a society be if people no longer possessed and exhibited these CHD-prone and cancer-prone personalities? If all the high-disease-risk behaviors and personality characteristics were attenuated, then it would be a very boring society with fewer artists, scientists, scholars, and investment bankers. Perhaps Professor Eysenck is really attempting to explain why the good die young. If he and others can figure out how to keep them around without removing what makes them "good," then he's onto something big.

Notes

The opinions or assertions contained herein are the private ones of the authors and are not to be construed as official or reflecting the views of the Department of Defense or of the Uniformed Services University of the Health Sciences.

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Personality, Stress, and Cancer: A Re-Examination

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There is considerable interest in possible relationships among personality, stress, and health. Eysenck, in his target article, suggests that there is sufficient evidence to regard personality and stress as important risk factors for cancer, equal in importance to smoking, heredity, and other physical factors. However, the model he delineated is based on a number of erroneous assumptions, and critical biological mediators, such as genetic susceptibility and health behaviors, have not been adequately assessed or controlled. Moreover, there is insufficient information on the psychometric properties of the personality inventories Eysenck and his colleagues have used to assess the different disease-prone personalities. These issues are critically discussed in this commentary.

Measure of Disease-Prone Personalities

In the target article, Eysenck discusses the Personality–Stress Inventory (Grossarth-Maticek & Eysenck, 1990) and the following six personality types it purportedly assesses: Type 1, which is the cancer-prone type; Type 2, which is the coronary heart disease (CHD)-prone type; Type 3, which combines traits from Types 1, 2, and 4; Type 4, which is the healthy, autonomous type; Type 5, which combines traits from Types 1 and 2; and Type 6, which is characterized by psychopathic tendencies along with proneness to drug addiction and AIDS. From the description and findings of Grossarth-Maticek and Eysenck (1990), it is unclear why Eysenck included the AIDS-prone personality in Type 6; the original article stated that this type includes antisocial tendencies and possibly proneness to criminal behavior and drug addiction. Moreover, the original article indicated that the probands were followed over a 13-year period; however, the first reported case of AIDS was in 1978.

The Grossarth-Maticek and Eysenck (1990) article on the construction of the Personality–Stress Inventory provided neither a theoretical rationale for the six personality types and their relation to disease-proneness nor sufficient information regarding the psychometric properties of the inventory to enable evaluation of its reliability and validity. Aside from the presentation of the test–retest reliability and the rotated and unrotated factor loadings of the principal-components analysis on the six personality types, the theoretical and empirical reasons for the items selected and retained for the different types and the internal consistency of the items within the types were not reported. Moreover, as Campbell and Fiske (1959) demonstrated, convergent and discriminant relationships between tests of certain constructs and other operational measures are fundamental to establishing the nomological network that forms the basis of construct validation. Studies examining the convergent and discriminant validity of the Personality–Stress Inventory with other mea-

sures, including measures of depression, psychopathic personality, and proneness to CHD and drug addiction, are needed.

It is also unclear from the information presented in the target and the Grossarth-Maticek and Eysenck (1990) articles whether the Personality–Stress Inventory is assessing stable personality traits as opposed to the ability to cope with stress that may be influenced by situational circumstances. Moreover, the "dynamic method" of scoring the personality questionnaire is inconsistent with the view that it is measuring stable personality traits. In the target article, Eysenck stated that the questionnaire "charts the progress of the way the individual deals with stress. Clearly, if stress is an important cause of death, then a 'D' score indicates that the individual is coping well with stress and shows psychological improvement, whereas an 'S' score shows the opposite."

Perhaps the questionnaire is assessing a behavioral pattern that could be modified by behavioral treatment. As indicated by Eysenck (1985), personality changes are difficult but that to change "a person's behavior pattern in the direction *opposite* to that characteristic of the cancer-prone personality, behavior therapy may be able to be useful in a prophylactic fashion, or to prolong life even after incurable cancer has been diagnosed" (p. 543). In short, studies on the psychometric properties of the Personality–Stress Inventory are needed before one can conclude that personality is an important risk factor for cancer and other diseases.

Biological Underpinnings of Eysenck's Model

In one of the articles that laid the foundation for Eysenck's current work (Eysenck, 1985), he suggested that "it is necessary to distinguish between acute and chronic stress, with the former *reducing* the effectiveness of the immune system, and promoting disease, while the latter may have the opposite effects" (pp. 537–538). Eysenck (1983, 1985) also suggested that chronic stress has a protective "inoculation" effect, and he cited a review of studies with rodents (Sklar & Anisman, 1981) as evidence for the protective effects of chronic stress. However, subsequent work with diverse human populations suggests that chronic stress is not protective: Chronically stressed people have poorer immune function than well-matched community controls (Kiecolt-Glaser & Glaser, 1988b; Kiecolt-Glaser et al., 1987).

Perhaps more important, however, is the notion that down-regulation of immune function is necessarily linked to cancer; in fact, there is very good evidence that this is not the case. Individuals who are severely immunosuppressed (e.g., persons with AIDS) develop only certain kinds of cancer. They do not show a widespread, undifferentiated increased incidence of all kinds of cancer, as would certainly be expected if this were the case. Fox (1981) described persuasive evidence that alterations in immune function per se are not necessarily related to most kinds of cancer.

The best evidence relating immune function concerns one particular facet of immune system function, natural killer (NK) cell activity and numbers of NK cells. Even in this case, the evidence is not as strong as it should be. For metastases, there is certainly evidence that NK cell activity is important (Levy, Herberman, Lippman, & d'Angelo, 1987); for primary tumors, however, the evidence is weak. Eysenck focuses his model on primary tumors, not metastases.

Eysenck calls for inclusion of immunological assays in prospective studies to monitor the effects of stress and therapy. However, the costs are not justified by the paucity of literature relating immune function to primary tumors.

In addition, one of the central confounds that is not described or discussed adequately is the mediating role of stress-related health behaviors. More depressed or distressed individuals are likely to engage in a variety of behaviors that have adverse health consequences, including eating and sleeping more poorly, abusing alcohol and drugs, getting less exercise, and so on (Kiecolt-Glaser & Glaser, 1988a). The only health behavior given any substantial emphasis by Eysenck is smoking, yet it is certainly not the only relevant behavior for understanding the relationships between stress and health.

Eysenck rarely distinguishes among the various kinds of cancer in his studies or in his overview. Cancer is a heterogeneous collection of conditions, not a single entity, and there are very different risk factors for different cancers (Fox, 1978). For many cancers, the single best and most reliable predictor of tumor development is one's genetic heritage (Fox, 1981). Although Eysenck says that heritability was controlled in his studies, he does not outline the procedures that were used.

Prevention of Cancer and CHD Through Behavior Therapy

Eysenck reports several behavior therapy studies in which treatment length or intensity ranged from "about 30 hr of individual treatment during the first few months after initiation of the study" to 4 hr of interviews in which contents of a "written pamphlet outlining the principles of behavior therapy as applied to better, more autonomous living, and avoidance of stress" were discussed. It is curious that such large effects would be produced by very limited therapy, behavioral or otherwise, when a relatively enduring personality style was the target for change.

Indeed, maintenance of treatment-induced behavioral change for far more circumscribed problems has been a central problem discussed at length in the behavioral literature; even in studies using much more prolonged and intense treatments, follow-up booster sessions are one of the strategies used to promote maintenance (Whisman, 1990). Eysenck himself addressed this issue some years ago (Eysenck, 1963), suggesting that continued contact with the therapist might be helpful in maintaining treatment gains. The enduring treatment effects obtained in the behavior therapy studies described by Eysenck are very different from the bulk of the literature (Whisman, 1990), particularly when treatment was limited to 4 hr; these studies certainly merit systematic replication in other laboratories.

In summary, although Eysenck's ideas have heuristic value, there are clearly several problems ranging from psychometric issues to difficulties with the proposed biological substrate. These questions need to be addressed carefully and thoroughly before the proposed relationships between personality and cancer and CHD can be accepted.

Notes

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Personality and Disease: A Call for Replication

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I am a medical statistician with a long-term interest in the epidemiology of smoking and smoking-associated diseases. Although not a regular reader of the psychological literature, I have always been on the lookout for articles suggesting a relationship of personality and stress to cancer and heart disease. Particularly where personality factors are recorded in those already suffering from disease, there are difficulties in interpreting reported associations as cause-and-effect relationships. However, although several studies have reported no significant relationship of the particular index of personality used to disease, there have been some consistent associations. The recent extensive review of Booth-Kewley and Friedman (1987), for example, highlighted a consistent relationship of coronary heart disease (CHD) to Type A personality, depression, anger–hostility–aggression, and anxiety. Although it seems clear that there are real relationships, the magnitude of the associations reported, even when significant, has often not been very large, with discussion often centering around relative risks of 2 or less between people in differing personality groups. Of course, in theory, personality indices used may not be very satisfactory in some studies, only indirectly correlating with true disease-prone personality types, so that relative-risk estimates may increase with improving indices. Even so, perhaps, before the data from Eysenck and Grossarth-Maticek, one might have thought it unlikely that any true relationship was massive, with one personality type having as much as, say, 10 times or more the risk of cancer or heart disease of another personality type. The real question of interest, it seems to me at least, is whether the article by Eysenck makes one change one's views.

The material presented in this article is remarkable in several ways. First, it is all published in the psychological literature, when one would have thought there was an enormously strong case for widening the audience and publishing the findings in the medical and epidemiological literature. Second, none of the findings are presented in the standard statistical form for results from prospective epidemiological studies. Thus, one is given cross-tables together with the occasional coefficient of association (which is of little or no value), but one is never given any relative-risk estimates. Third, the strengths of the reported associations are absolutely mammoth.

The third point is, of course, the crucial one. To illustrate it clearly, I present relative-risk estimates corresponding to the data in some of Eysenck's tables. There is in fact a problem in computing relative-risk estimates accurately from the material provided, but approximations can be calculated in two ways. One is to use standard case-control study methodology, treating the survivors as representative of the living population, and calculating the cross-product ratio ad/bc where a and b are the number of deaths from the cause of interest in the two personality groups being compared, and c and d are the number of survivors. This may somewhat overestimate relative risk if death rates are high. The other is to calculate the ratio

$$[1 - (c/(a + c))^{1/y}]/[1 - (d/(b + d))^{1/y}]$$

where y is the number of years of follow-up. The numerator and denominator are the estimated average annual death rates from the disease of interest in the two personality groups.

Tables 1 and 2, based respectively on the Yugoslav and Heidelberg studies, show estimates of relative risk by personality type for cancer, CHD, other causes, and all causes combined. Results for the Heidelberg study, being based on smaller numbers of deaths, are more variable, but the overall picture is quite clear. Types 1 and 2 (compared with Type 4) have around a 10-fold increased overall death rate in the Yugoslav study and more than a 20-fold increased overall death rate in the Heidelberg study. The relative-risk estimates are even higher for cancer for Type 1 (50-fold) and for CHD