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The Multiple Linkages of Personality and Disease

Howard S. Friedman
University of California, Riverside

Abstract

Associations between personality and health and longevity are increasingly well-documented, but the causal inter-connections are often much more complex than originally anticipated. Multiple causal pathways may operate simultaneously as the individual travels an idiosyncratic route across the lifespan. Therefore, a straightforward model of personality, immunity, and disease may never be established and validated, because it overlooks other key elements of the causal processes. Psychoneuroimmunology research may profit from closer integration into the broader conceptual understandings of personality and health, using a new lifespan epidemiological personality approach.

Keywords

personality; psychoneuroimmunology; longevity; lifespan mortality risk; conscientiousness

Understanding of individual differences and disease has come a long way since the early focus on syndromes like the coronary-prone behavior pattern. Not only have clear associations been documented, but there remains little doubt that modern conceptions and findings from personality psychology provide valid and useful new insights into the nature of health and health maintenance. In particular, two important phenomena have emerged, but are generally overlooked. The first is that multiple causal linkages between personality and disease may be simultaneously operating across long periods of time. The second is that understanding of individual differences in disease may often be of equal or greater importance than is understanding the general causes of disease.

Looking broadly, a curious observation has emerged from the research literature on personality, immunity, and disease. There is now substantial evidence that individual differences (in humans) in stress reactivity, chronic stress, coping, cognitive styles, emotionality, and so on are associated with a host of physiological responses and homeostasis, including a variety of immunity-related states (Cacioppo & Berntson, 2007; Kemeny, 2007). And there is of course little doubt that disruptions of the immune system affect susceptibility to and recovery from disease. Yet there is shockingly little evidence for a straightforward model of psychological dysfunction, consequent immune disruption, and subsequent disease (Segerstrom & Miller, 2004). For example, there is very thin direct evidence of susceptible persons suffering significant stress such as divorce, coping poorly, showing immune impairment, and developing rheumatoid arthritis or leukemia or pulmonary disease, as compared to a well-substantiated traditional medical model of persons being exposed to and infected by HIV, showing immune disruption, and then developing AIDS and other diseases. In fact, it may be the case that such

Dr. Howard S. Friedman, Department of Psychology, 900 University Ave., University of California, Riverside, CA 92521. Howard.Friedman@ucr.edu 951 827-3672.
Howard S. Friedman, Department of Psychology, University of California, Riverside, CA 92521. Howard.Friedman@ucr.edu

a straightforward model of personality, immunity, and disease will never be established, because it overlooks other key elements of the causal processes.

Advances in animal (non-human) studies are making progress in finding animal analogs to human individual differences, which go beyond the traditional attention to genetics, temperament, and early deprivation. For example, they may use reliable behavioral coding to capture social structure and traits like sociability, while taking psychophysiological measures and hormonal assays (Capitanio, Mendoza, & Bentson, 2004; Weinstein, Capitanio, & Gosling, in press). The parallel question then arises as to the extent to which such studies can capture the full range of complex phenomena that emerge as links between personality and human health.

Conceptual Progress in Understanding Personality and Disease

Modern notions of biopsychosocial homeostasis began with the psychophysiological models of Claude Bernard (1880) and the sfight-or-flight" discovery by Walter Cannon (1932). Psychosomatic approaches began in earnest soon after. For example, in classic work beginning in the 1930s', Dunbar (1955) and Alexander (1950) described conflicted patients developing ulcers, asthma, heart conditions and so on. But the concepts and assessments (often psychoanalytic case studies) were empirically unsupportable and psychometrically deficient (Friedman & Adler, 2007). In the one of the first well-designed studies, in the late 1940s, a number of medical students at Johns Hopkins University were assessed in terms of their biological and psychological characteristics, categorized as either slow and solid, rapid and facile, or irregular and uneven. The last were more likely later to develop a serious medical disorder (Betz & Thomas, 1979).

Systematic study of the association between behavior patterns and heart disease began in earnest in the 1950s, with a subsequent explosion of research (thousands of studies) of Type A behavior (Chesney & Rosenman, 1985). Importantly, persons who did not show Type A characteristics were not independently conceptualized, but simply called Type B. The paradigm eschewed the usual scientific practice of establishing construct validity by showing that (1) the assessment is related to what it should theoretically be related to (convergent validation) and (2) the assessment is not related to what it should not be related to (discriminant validation) (Campbell & Fiske, 1959). This purposeful disregard of construct validity and psychological theory did not make the phenomenon more objective and simpler to study (as was intended), but, on the contrary led to endless meandering and unspecified studies, a sad lesson still important for current research in this field. Yet by inspiring so much empirical research, the flawed Type A idea helped the establishment of a much more comprehensive approach.

One conceptual advance that followed was to place such issues into a more valid scientific framework. For example, the constructs termed "disease-prone personalities" and "self-healing personalities" (Friedman 1991/2000/1998; Friedman & Booth-Kewley, 1987) directed research away from associations of single-predictors to single-outcomes (like Type A and heart disease); instead, there was simultaneous attention to multiple predictors and multiple outcomes. There was also concern with rigorous, multi-dimensional conceptualization and assessment. With this attention to broader personality and overall well-being, research generally found that similar sorts of individual differences were simultaneously relevant to a wide array of health outcomes (Friedman, 2007; Smith & Gallo, 2001).

The Multiple Causal Linkages Between Personality And Disease

The necessities of medical treatment and the exigencies of research design often conspire together to eliminate study of one of the most important elements of understanding health, namely pathways across long periods of time. Medical care is mostly geared to treat acute

illnesses or acute manifestations of chronic diseases rather than long-term prevention of disease and promotion of health. (Immunizations are a significant exception to this general rule.) Similarly, research designs, for reasons of cost, feasibility, and practicality, must usually focus on treating disease or on secondary prevention (measures taken once a problem begins to develop, such as interventions to reduce high blood pressure), rather than on primary prevention (measures designed to prevent illness from developing in the first place). Thus, certain sorts of more complex causal linkages tend to be under-explored or even totally overlooked. The area of stress physiology unfolding over time is one in which animal research holds promise but is under-utilized (see Koolhaas, Boer, & Buwalda, 2006).

Such matters are made worse by the organization of research and treatment by disease. Some physicians and researchers are experts in and are funded to study heart disease, while others study AIDS or diabetes, and still others deal with drug abuse or smoking or depression. Researchers of psychoneuroimmunology (PNI) are, in theory, better positioned to avoid such limitations because of a primary focus on basic processes. Yet even here it is unusual to see long-term studies of initially healthy people, and subsequent stress, immune processes, behavior, and multiple disease outcomes, including mortality risk. Longevity is arguably the best single measure of health (and indeed expected life-span is used for that reason in cross-national epidemiological comparisons of health).

With detailed study of personality and health and longevity, however, we are led to research not only on psychoneuroimmunology and stress, but also on unhealthy habits, social relations, and reactions to varying life paths, and then see how the pieces fit together in the whole person and across time. The study of personality and health also draws much-needed attention to the individual's selection and creation of health-relevant environments (Friedman, 2000). It matters whether a person's cells or organs are uninfected and functioning well, but only because these affect the (whole) person. It is the *individual* who lives a unique life path, becomes ill or stays well, and lives long or dies prematurely.

It turns out that there are a variety of valid causal linkages between personality and health, each with its own subsets and variations. A frustrating fact of life is that it is often true that more than one linkage is simultaneously causing an observed association; but most study designs cannot detect multiple causal linkages. In other words, many health outcomes are multidimensional and are multiply or complexly determined; the phenomena thus push us to the limits of model construction and research design and analysis. This state of affairs is especially worrisome because a key goal is usually to find some intervention (whether behavioral, social, pharmaceutical, immunological or genetic) to treat or prevent disease, hopefully in a straightforward and cost-effective way.

One commonly-studied model, the health behavioral model, proposes that personality leads to disease through patterns of unhealthy behaviors such as poor diet, smoking, and lack of physical activity. Mediational analyses of behavior help point to likely causal pathways, by showing whether the association between personality and disease decreases in whole or part when the unhealthy behavior is controlled (usually statistically). Such findings are evidence (but not proof) of the causal model. For example, it might also be the case (and often is the case) that other factors are correlated with the trait, the behavior, and the disease. Even if such a link is established, treatments for the personality imbalance (e.g. treatments for anxiety) will not necessarily prevent the disease (e.g. diabetes) unless the mediators (e.g. overeating, glycemia regulation) are also affected. Even if the mediator is affected, the disease risk or mortality risk many not diminish if another unhealthy behavior (such as drinking) takes its place.

Genetic endowments and early experiences can affect both later personality and later health and longevity. This is the underlying biological model, or third variable model, of personality

and health, in which changing the health would not usually affect personality, and shaping the personality would not usually alter the health risks. Although attention is usually on genetics, *in utero* and early post-natal effects are an interesting example, such as when stress during pregnancy affects later reaction patterns and health of the offspring, an effect well demonstrated in rhesus monkeys and likely in humans (Coe & Lubach, 2005; Lobel, Dunkel-Schetter, & Scrimshaw, 1992; see also Caspi et al., 1997). However, other than by locating a simple genetic defect or potent early stressor, the underlying biological model is notoriously difficult to evaluate in humans and it is often simply ignored. This is slowly changing, with increasing focus on genes, environmental challenge, and health (Caspi et al., 2003).

Commonly overlooked as well are explanations involving disease-caused personality changes. Many diseases can subtly affect personality years before the disease shows itself in clinical form. So it may appear that personality is causing disease (or is a risk factor) when the reverse is true. Many times, these are brain diseases, since changes in the brain can obviously affect one's typical response patterns. The primary problem can originate in the brain (e.g. small hemorrhagic stroke), or there could be toxins (e.g., lead, manganese), infections (e.g. syphilis), or behaviors (e.g. alcoholism) that affect the brain and personality both directly and indirectly. Or, prescription pharmaceuticals for various medical conditions can affect personality in documented and often undocumented ways. Even social reactions to disease diagnosis (such as prejudice against HIV) can precipitate alterations in personality, in turn altering one's life-path. And, multiple such pathways of disease-affected personality can be operating simultaneously.

A less studied but very interesting model of a causal link between personality and disease involves individual differences in gravitating towards or choosing unhealthy situations. Given modern understanding of personality, the usual model of a person randomly encountering various stressful events is untenable. The fact is that the individual helps create and select situations and events. For example, neuroticism tends to predict to negative life events. (See the Example of Neuroticism below.) This phenomenon also illustrates some of the limits of non-primate animal studies of personality and disease. That is, although mice and hamsters allowed to develop social bonds may have faster wound healing and better disease recovery (DeVries et al., 2007), rodents' personalities do not generally lead them into or out of extended social networks or the rat races of Los Angeles and New York.

Finally, of course, there is the psychophysiological model, with stress and poor coping directly and indirectly affecting metabolism, immunity, and the cardiovascular system. Many health psychologists (especially those not expert in PNI), implicitly assume that this sort of causal model has been (or soon will be) established as the causal mediator between personality and disease. This model as well can benefit greatly from being placed it into the broader context of understanding individual differences in disease across time.

The Importance Of Individual Differences In Disease and Mortality Risk Across Time

The Example of Neuroticism

As noted, a limit of the traditional studies of personality and health has been too little attention to multivariate assessment of predictors, controls, and outcomes; to some extent, this is redressed by taking a more epidemiological approach. However, many purely epidemiological approaches are limited by ignoring situation selection, by discounting the psychology of actual day-to-day reactions and behaviors, and by erroneously controlling away demographic factors that are rich and complex, such as socioeconomic status. Combining strengths, we need a *life-span epidemiological personality* approach.

One example concerns neuroticism, generally thought to involve anxiety, depression, hostility, and vulnerability. In 1987, a meta-analysis revealed that not only hostility, but also depression was reliably associated with cardiovascular disease (Booth-Kewley & Friedman, 1987). This finding ran counter to the prevailing wisdom about the importance of (narrowly construed) Type A behavior, and it was viewed skeptically at the time (Mathews, 1988). However, the general association between depression and risk of heart disease has since been confirmed in a many studies (Barefoot & Schroll, 1996; Barefoot et al., 2000; Ford et al., 1998; Januzzi et al., 2000; Rugulies, 2002; Suls & Bunde, 2005). When an appropriately broad frame is employed, depression also predicts other diseases.

Using a familiar approach that sees a risk factor (depression) as a direct causal agent, clinicians therefore began treating depression in a effort to prevent the further development or exacerbation of heart disease. Surprising to some, treating depression in recent heart attack patients does not necessarily reduce the risk of death or second heart attack (ENRICHD, 2003). The confusion that resulted from such a research result (and an over-reliance on pure epidemiology) is reminiscent of the confusion that surrounded failed Type A intervention studies decades earlier. How and why is depression predicting heart disease and why only in some people? Viewing depression as a simple “medical risk factor” outside the interconnected web of associations is short-sighted. The same might prove true of its correlates, such as inflammation.

Situational selection is also clearly germane. Neurotic people, including depressives, are more likely to encounter negative events and to interpret them in a more negative manner (Bolger & Zuckerman, 1995; Magnus et al., 1993; Mroczek & Almeida, 2004; Taylor, Repetti & Seeman, 1997). To detect these evocative effects requires a life-span design.

Immune processes are also relevant, but in more than one way. Although depression is often associated with immune dysfunction similar to high stress effects, immune enhancement has also been observed with depression. Further, there is some evidence of reverse causation; that is, pro-inflammatory cytokines may be a partial cause of depression (see Kemeny, 2007 for a review). As Kemeny (2007) put it, “a relationship between a psychological factor and a change in the immune system may be due to the simple impact of the mind on the immune system, the effects of the immune system on the mind, both, or neither” (p. 111).

Life-span and epidemiological work likewise suggests neuroticism can be health protective as well as harmful. One explanation for these conflicting results is that there are at least two quite different life paths characterized by neuroticism (Friedman, 2000). In one case, adults who are pessimistic, resentful, and anxious will fail to adhere to treatment regimens, engage in an unhealthy lifestyle, have disrupted homeostasis, lose sleep, and have a lack of social support, all of which increase the risk of poor health. The other pathway leads to better health as a consequence of neurotic vigilance and treatment adherence. Indeed, recent research supports the idea that factors associated with neuroticism, such as self-reports of psychological distress and mental strain, can sometimes predict *lower* mortality risk (Gardner & Oswald, 2004; Korten et al., 1999; Weiss & Costa, 2005).

Finally, there is evidence of common genetic vulnerability to depression and coronary artery disease (McCaffery et al., 2006). Here again the classic risk factor intervention (“treat depression”) will not succeed, because to the extent that both depression and heart disease derive in part from a genetically based vulnerability in the serotonin system, interventions to affect depression will not necessarily have expected effects on heart disease risk.

The Example Of The Human Termites

Health research often seems to be addressing the question, "*Why* do people become sick?" when it is actually studying "*Who* becomes sick?" There is astounding variability in susceptibility to various illnesses and in the speed and likelihood of recovery. From an evolutionary standpoint, it is not surprising that there is so much variation, given the vast number of potential threats to health and reproductive success. Yet health researchers tend to be disease-focused investigators, concerned with curing a particular scourge rather than searching for basic scientific principles of human health.

A significant problem for researchers of personality and disease is that one cannot randomly assign a person to personality or to stress. Because we are left with correlational designs (at least for human studies), we need rich data collected across many years to be able to untangle the various plausible causal linkages explained earlier in this paper. (Likewise, animal studies ideally would strive to capture elements of these long-term processes.) Highly informative studies would start with initially healthy people, follow them for many years, have repeated assessments of individual differences, and continue until the participants have died.

For more than 17 years, my colleagues and I have studied personality and longevity in the Terman cohort, derived from the Terman Gifted Children Study that began in 1921. The 1,528 Terman participants (who later called themselves "Termites") were, for the most part, first studied as elementary school children, and have been followed ever since. It is the longest study of a single cohort ever conducted so intensively, with rich data collected regularly throughout the life-span. Importantly, we have collected death certificates and have professionally coded date and cause of death (Friedman et al., 1995). We have also developed an extensive set of variables and indexes, derived from the Terman items, interviews, and questionnaires, but validated in new ways by our team.

The Terman sample was later characterized as a productive, intelligent segment of 20th century middle-class American men and women (mostly White) (Sears, 1984; Subotnik, Karp, & Morgan, 1989). Although it is non-representative of the general population, the sample is actually more representative of the population than the various well-known prospective studies of samples of physicians or nurses. In addition, examinations of the impact of IQ selection on relations among traits correlated with IQ show little distortion. Most importantly, the results are continually being validated by others in other samples.

What have we found? Beginning with childhood, we first tested whether variables representing major dimensions of personality could predict longevity across the life-span (Friedman et al., 1993). We examined all items collected by Terman in 1922 that seemed relevant to personality. In 1922, one of each participant's parents (usually the mother, or both parents together) and each participant's teacher were asked to rate the participant on 25 trait dimensions (using a 13-point scale) chosen to measure intellectual, volitional, moral, emotional, aesthetic, physical, and social functioning. The scales used are remarkably modern in their appearance, and we created reliable and valid personality measures. The most interesting and surprising results concerned conscientiousness, and those will be described here.

We constructed a childhood measure of Conscientiousness–Social Dependability with the four items of: prudence, conscientiousness, freedom from vanity/egotism, and truthfulness. This corresponds roughly to the five factor model (McCrae & John, 1992) dimension of Conscientiousness, and we have documented the correspondence between conscientiousness measured with this scale and the contemporary NEO PI-R conscientiousness (although the measure is not identical to the NEO-PI measure) (Martin & Friedman, 2000). Survival analyses revealed that childhood conscientiousness was clearly related to survival in middle to old age.

For example, a person at the 75th percentile on conscientiousness had only 77% of the risk of a person at the 25th percentile of dying in any given year (Friedman et al., 1993).

We then derived (from the archival Terman data) personality measures for adulthood that we likewise validated by showing them to be consistent with the five factor contemporary conceptions (Martin & Friedman, 2000; Martin, Friedman & Schwartz, 2007). Adult conscientiousness was "measured" when the participants were in their 30′s and 40′s. As of the year 2000, 70% of the men and 51% of the women in this sample had deaths verified by us. We again found conscientiousness (now measured in adulthood) to be significantly related to mortality risk. Those low on adult conscientiousness died sooner.

Some partial evidence about the casual links comes from comparing the relative benefits of higher sustained levels of conscientiousness over the life span versus higher levels only in adulthood. If high conscientiousness in adulthood is most important, then adult health behaviors are likely very relevant, whereas if conscientiousness throughout life is most predictive, then underlying biological variables also become plausible. It turned out that conscientiousness, measured independently in childhood and adulthood, predicts mortality risk across the full lifespan, and the link from childhood remains robust when adult conscientiousness and certain behavioral variables are controlled (Martin, Friedman & Schwartz, 2007). The lowest mortality risk is for those with high conscientiousness at both time points.

The Friedman et al. findings on conscientiousness and longevity have now been confirmed in follow-up studies by others. One five-year study examined the relation of personality to mortality in 883 older Catholic clergy members, and employed the NEO Five Factor Inventory. Those scoring very high on conscientiousness were about half as likely to die as those with a very low score (Wilson et al., 2004). Another study examined participants in a Medicare demonstration study with over 1000 participants, aged 65–100 (Weiss & Costa, 2005). The participants in this prospective study were older, sicker, and more representative of the elderly population. Over the five years of follow-up, persons high in conscientiousness were significantly less likely to die.

Evidence of the generality of the importance of conscientiousness comes from a study using the Midlife Development in the United States Survey, a nationally representative sample of 3,032 non-institutionalized civilian adults (Goodwin & Friedman, 2006). This study examined the association between the five-factor traits of personality and common mental and physical disorders. The results showed that Conscientiousness (protectively) was reliably associated with reduced risk of illness: those with diabetes, hypertension, sciatica, urinary problems, stroke, hernia, TB, joint problems, and a variety of mental illnesses and substance abuse problems had significantly lower levels of conscientiousness, compared to those without each disorder. There are also narrower studies such as one that focused on conscientiousness and renal deterioration in patients with diabetes. It found that time to renal failure was longer in those with high conscientiousness (Brickman et al., 1996). Another prospective study of chronic renal insufficiency found that patients with low conscientiousness had a substantially increased mortality rate over the four-year term of the study (Christensen et al., 2002).

Why is conscientiousness so relevant to health and longevity? It seems that many of the various models of personality and health and longevity are relevant. For example, a meta-analysis of 194 studies examined conscientiousness-related traits and leading behavioral contributors to mortality -- tobacco use, diet and activity patterns, excessive alcohol use, violence, risky sexual behavior, risky driving, suicide, and drug use (Bogg & Roberts, 2004). It found conscientiousness-related traits were negatively related to all the risky health-related behaviors and positively related to the beneficial health-related behaviors. In other words, the behavioral

model has good support. However, in the Terman data, although conscientious Terman children were less likely to grow up to be heavy drinkers and smokers, and less likely to suffer death from injury, health behaviors alone did not explain away the effect. As noted, conscientiousness seems to have a more far-reaching and general involvement.

In this case, later disease (and higher mortality risk) obviously could not be causing the conscientiousness personality, and so various disease-caused personality change models can be ruled out. The evidence thus points to the likelihood of some sorts of third variables affecting both personality and longevity (in a causal sense), in addition to the effects of healthy and risky behaviors. Evidence from other research suggests that this might be linked to serotonergic functioning (Carver & Miller, in press). However, it is also the case that more controlled, secure, and hard-working persons at age 18 are headed to better careers within a decade, which in turn can further enhance their health and their subsequent conscientiousness (Hogan & Ones, 1997; Judge & Ilies, 2002; Roberts, Caspi, & Moffitt, 2003). And conscientious college women experience lower rates of divorce (Roberts & Bogg, 2004), in line with models about self-selection into stressful environments. In our Terman studies, study of the association between marital history at midlife (in 1950) and longevity showed that consistently married individuals lived longer than those who had experienced marital breakup, but this was not only due to the protective effects of marriage itself (Tucker et al., 1996). Rather, participants who were currently married, but had previously experienced a divorce, were at significantly higher mortality risk compared with consistently married individuals; consistently married individuals had been more conscientious children than were inconsistently married individuals.

Our other studies with the Terman participants likewise show that personality can reliably predict health and longevity in this cohort (and be confirmed in other cohorts), but causal linkages are usually not simply summarized by a single model. For example, cheerfulness and optimism are often associated with well-being, health, and longevity, but not in simple ways, especially if methodological care is taken and all the relevant causal links are considered (Pressman & Cohen, 2005). In fact, in the Terman data, children who were rated by their parents and teachers as more cheerful/optimistic, and as having a sense of humor, died *earlier* in adulthood than those who were less cheerful, growing up to drink more alcohol, smoke more cigarettes, and engage in more risky hobbies and activities, although other (unknown) factors are likely more relevant (Friedman, 2007; Martin et al., 2002; Schulz et al., 2000).

Conclusion

In the three or so decades since it was discovered that the immune system could be classically conditioned, scientific research in health psychology has blossomed as biological processes were revealed that linked to both psychological constructs (like personality, coping, and perceived stress) and health outcomes. Suddenly the old quest for disease-promoting psychological patterns gained credibility, as physiological mediating mechanisms potentially could be uncovered. But taking a step back from the focus on individual differences and PNI reveals that the topic has multiple dimensions. Various pathways likely co-exist, often simultaneously and with feedback loops, and it is unwise to attend to only one path.

The dominant biomedical model of disease exerts constant pressure to focus on something gone wrong inside the individual as the cause of disease, whether it is a genetic abnormality, an infection, a toxin, allostatic load, or injury (trauma). The treatment then involves eliminating or repairing the problem. When first applied in psychosomatic medicine, the goal was thus to resolve the inner conflict, or stop the worrying, or cure the hurry-sickness. However, modern understanding of personality leads to attention to a dynamic unfolding across time, as biological and temperamental predispositions encounter, select, and create varying environments that in

turn further shape the individual. This sophisticated consideration of the individual personality forces a broader and deeper analysis, closer to the true complexity of well-being.

Using data derived from the 8-decade Terman study, and collecting new information about the participants' longevity and cause of death, we have discovered that numerous, simultaneous linkages likely do exist among personality, behaviors, environments, and health and longevity. For example, conscientiousness is a key predictor of longevity across the full lifespan, and biological correlates are waiting to be fully documented, but simple interventions or "cures" for unconscientiousness are unlikely to be found or be successful. This importance of individual reaction *patterns* and changes over time is echoed in a review of studies of chronic stress and the hypothalamic-pituitary-adrenocortical (HPA) axis, which concluded that "models positing an orderly and uniform HPA response to chronic stress are no longer appropriate. A new wave of theories needs to be developed to incorporate the moderating influences of timing, nature of stress, controllability, and individual psychiatric response" (Miller, Chen & Zhou, 2007, p. 38).

Research on personality and immunity has not proved to be the conceptual panacea initially hoped for by some psychologists and immunologists (Ouellette & DiPlacido, 2001). Well-controlled studies of personality, stress, and illness fail to find simple and comprehensive psycho-immunological mediating mechanisms (Kemeny, 2007), although there are intriguing commonalities between human and animal personality research findings (e.g. Capitanio, Mendoza, & Baroncelli, 1999; Cacioppo & Berntson, 2007; Sapolsky, 2005). We do now know, however, much more about the complications and complexities. Most especially, progress is being made on developing more sophisticated models and methods for addressing the links between personality and health.

There is astounding variability in susceptibility to various illnesses and in the speed and likelihood of recovery. In an initially healthy population, traditional risk factors like blood pressure, diet, activity, amount of work, occupational stress, weight (BMI), and so on are important to health but mostly do a weak job (except at the extremes in risk levels) of predicting who will develop coronary disease, who will develop colon cancer, who will die from such disease, and when and why they will succumb while their neighbors may recover. Absent a devastating onslaught such as from HIV infection, heavy tobacco use, starvation, or certain powerful genetic diseases, variability seems diabolically multifaceted, but perhaps eventually understandable.

A half century ago, two pediatricians followed a number of families for about a year, doing throat cultures for streptococcal bacteria every few weeks. In a surprising result, they found that most of the time, the strep infections did not produce any symptoms of illness. The strep bacteria by themselves did not cause illness. But when the families were stressed, the strep illness was more likely to develop (Meyer & Haggerty, 1962). (On the other hand, no one developed the strep illness without exposure to the bacteria.) Yet the biomedical approach remained dominant, even as such studies helped lay the conceptual foundation for a biopsychosocial approach. Today, even research in health psychology that considers the full biopsychosocial model has found the phenomenon resistant to simple explication. For example, in one of the few comprehensive series of human studies with carefully (experimentally) controlled exposure to the infectious agent (virus), Cohen and colleagues have demonstrated that factors such as self-reported chronic stress and lack of a positive emotional style predict increased risk of subsequent upper respiratory infection (Cohen et al., 1998, 2006); however, it has been difficult to identify either behavioral or immunological mediators of such effects (see also Overmier & Murison, 2000, on ulcers). I suggest that this complexity may be due to the fact of multiple causal links—behavioral links, stress links, biological and temperamental underlying third variables, situational selection, and reverse causal links-- simultaneously

operating, and with different stages across time. In other words, current research in PNI addresses part of this challenge by elucidating one important mediating mechanism between the individual and the health outcome. Such research may prove even more valuable, however, if it takes into account the broader set of forces involved in personality and disease.

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