

PART I

Models of Health/Risk Behavior and Behavior Change

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CHAPTER 1

Healthy Life-style Across the Life-span: The Heck with the Surgeon General!

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Introduction

Health times are changing. Eggs are again a healthy food. Avoiding cholesterol-laden eggs won't solve elevated-cholesterol problems for most people. Salt intake, however, can lead to high blood pressure, and thereby perhaps threaten cardiovascular health. Except, maybe eggs are not so healthy, possibly because of their high levels of saturated fat. And the threat from salt intake seems only true for certain people who are sodium sensitive. Butter is full of saturated fat, so you should switch to margarine. Wait. Margarine, containing hydrogenated oils, is loaded with trans fatty acids, which makes it a poor alternative to butter. Try the new and expensive kind of cholesterol-lowering margarine.

Where does all of this conflicting health advice come from? Some of this changing advice results from new scientific discoveries. New studies constantly address a piece of the puzzle of the development of chronic illness. Since cardiovascular disease is by far the greatest killer in the Western world, it and its risk factors (serum cholesterol, blood pressure, diet, stress) receive lots of research attention, usually fragmentary. Another part of this contradictory advice results from clinicians and reporters who overstate their findings. Individual studies are rarely multi-faceted, long-term, and definitive. So as each finding emerges, it receives more attention than justified; then later, another, different piece of the picture is revealed.

But part of the confusion results from scientists who misunderstand their findings. It is this scientific mis-step that is the subject of this chapter.

In 1989 I wrote a book entitled *The Self-Healing Personality*. I wrote:

“Since eggs are high in cholesterol, some scientists have urged people to make drastic changes in their diets – avoid all eggs. However, cholesterol does not go

directly from our stomachs into our blood. The human body processes the cholesterol in food and makes its own cholesterol. The level of cholesterol in our blood is affected by hereditary factors, by the amount of fat (especially saturated fat) in the diet, by exercise, and by stress. It is also affected by other, as yet unknown, factors. Avoiding eggs will by itself have little or no effect on blood cholesterol in most people.

Many products on the supermarket shelves are now advertised with the ridiculous slogan, 'No cholesterol!' Believe it or not, I recently purchased a bunch of bananas that had a 'No cholesterol' sticker attached to them. This labelling indicates a grave public misconception of the best ways to promote health.

For a whole host of reasons, it is healthy to eat lots of fruit and vegetables. Bananas do fall into this category, but no scientist really knows all the exact details of why fruits and vegetables are good to eat. Certainly a lot more than cholesterol content is involved . . .

How many people are now feeling guilty when they eat a steak? The guilt is likely a greater problem than the steak. It is true that there is substantial evidence that high animal fat intake is unhealthy. At a restaurant near my home, I observed a fat man devour a huge fatty chunk of prime rib. He concluded the meal with a large piece of chocolate cake a la mode. If he does this often (as he evidently did), his arteries may pay the consequences. But people who occasionally enjoy eating a trimmed piece of broiled steak as part of a varied diet are giving themselves an excellent source of protein and minerals" (Friedman, 1991/2000: 130).

Now, more than a decade later, both the popular and scientific literatures are filled with articles questioning the "ban" on eggs and steak. They claim there is "new research" (e.g., "Eat your heart out: Forget what you know about eggs, margarine and salt", *Time* magazine, 1999). So how could I presciently write those words so long ago? All I had to do was read the scientific literature and think about its full context. There was never any convincing study even remotely indicating that eliminating high-cholesterol eggs from breakfast would improve the health of the population. Similarly, eating an occasional steak (full of essential proteins and minerals) was never shown to be worse for one's arteries than many other common foods, including drinking milk. But scientists misunderstood their own findings.

As we shall see, our health promotion efforts and our public health systems are too often built around a pathology model, derived from traditional conceptions of "treating" disease. These approaches often ignore the social context of people's lives, and the psychosocial influences that push and pull them in healthy or unhealthy directions across time. In the scientific arena, this orientation often means that each result from a particular scientific study is seen as an important and direct causal step on the road to disease. Anything that seems to be associated with an increase in a risk factor is a threat! Thus we encounter a litany of health advice – do's and don't's sometimes relevant to the proximal causes of ill health but ignorant of the long-term causal patterns.

Furthermore, such advice appears in isolation, disease by disease. All together, in the popular arena, this faddish approach produces people who have had it up to their noses with conflicting medical advice. They have had their

fill of half-baked baloney casseroles. So they junk all the advice and return to eating junk food. They say, "The heck with the Surgeon General!"

The truth be told, this exclamatory subtitle is not original. Rather, it was stolen from a huge billboard on the highway between San Diego and Riverside. The huge letters proclaim, "The heck with the Surgeon General." This is followed by the phrase "Inhale a big juicy star." It is an advertisement for Carl's Junior star hamburgers. Forget about warnings, and inhale loads of fatty hamburgers! Millions do. The burgers are accompanied by fries and shakes.

Backlash

A study in the *Journal of the American Dietetic Association* documented this backlash against promulgated nutritional advice (Patterson et al., 2001). This research used a random digit telephone survey of residents of Washington state, weighted to be representative of the population. More than two-thirds of the respondents asserted that the government should not tell people what to eat, and many complained about low-fat diets. More importantly, people evidencing high "nutrition backlash" ate more fat and fewer servings of fruits and vegetables.

The causal direction of these associations with nutrition backlash is not established. Patterson et al. (2001) concluded that it is likely that people who are annoyed with constant government and media harping on low-fat diets are more likely to disregard the advice altogether, and eat a fat-laden and low-fruit diet. The government advice backfires. This is also the prediction of psychological reactance models, which forecast that threats to one's personal freedom produce negative reactions that increase one's resistance to persuasion. This reactance against health advice may be especially true among people concerned with control issues (Rhodewalt and Davison, 1983). It is also the case that people may generally see themselves as less susceptible to such influence when the persuading entity is an irrelevant "outgroup" such as the government (Terry et al., 1999).

On the other hand, social psychological theory and research on cognitive consistency predicts that people who know they are eating high-fat, low-fruit diets will be more likely to evidence this "nutrition backlash" when asked about their diet. That is, if one is eating French fries, pork chops, and ice cream on a regular basis, then one is unlikely to assert that the government is doing a fine job in warning people about the health risks of such diets. Such thoughts and behaviors would be inconsistent, dissonant, and unperceptive. In this case, it is not annoyed people who ignore health advice, but rather misbehaving people who become annoyed with the advice (Abelson et al., 1968).

It is likely, however, that both sorts of causal directions account for the association between poor dietary habits and dissatisfaction with government preaching and scientific reversals. Some people will not attend to health messages, will not believe them if they hear them, and will not change their behaviors even if they hear and believe the message. Various cognitive, emotional, and informational processes are at work. On the other hand, other people will

form unhealthy habits and behave in unhealthy ways for a variety of interpersonal and situational reasons, and they then will form negative attitudes about health promotion as a function of these behaviors (Rodin et al., 1990).

The Skinny on Fat

Human beings have evolved to enjoy eating fat. In fact, people cannot live without fat in their diets. There are many different types of fats. There are fats from dairy products and fats from meats, there are artificial fats from food processors, and there are fats from produce ranging from soy and nuts to olives and avocados.

There are fat people who do not eat much fat, and there are skinny people who eat a lot of fat. Many people gain weight as they age, but many do not. Although it is known that some people who eat a lot of saturated fat will raise their cholesterol levels, a subsequent long-term causal link to all-cause premature mortality from this single behavior has not been directly documented as a major risk to the population.

Medical advisors who recommend addressing high serum (blood) cholesterol in people at high risk for cardiovascular disease through dietary changes in fat intake are piecing together different sorts of findings. But it has always been controversial whether simple diet-based attempts (such as avoiding eggs) at serum cholesterol reductions are needed for healthy young or middle-aged adults, especially given the often minimal or unexpected effects on serum cholesterol and health of moderate dietary changes (Kaplan et al., 1992; Taubes, 2001; Taylor et al., 1987). Further, any beneficial effects preventing deaths from cardiovascular disease might be offset by increased risk from other diseases.

Fat and carbohydrate metabolism in the body is complicated, and it is not clear that a high carbohydrate diet is especially healthy as a replacement. Add in considerations of physical activity, stress, alcohol, and culture, and the complexity multiplies dramatically (Epel et al., 2001). Note that during the years since the government and some health advisors have begun preaching fat intake reduction, the incidence of obesity among Americans has increased dramatically.

Of course such issues do not negate the documented associations between certain habits and disease. For example, there is a vast amount of evidence associating fruit intake with good health, and increasing one's fruit consumption of delicious fresh fruit might yield better health as a lagniappe (extra gift) for the lucky.

Other Health Promotions

Strangely reminiscent of the fat controversies, there is currently a governmental effort to increase the amount of exercise individuals do, as part of "Healthy People 2010" (<http://www.aoa.gov/factsheets/LONGEVITY.html>).

There is good correlational evidence that people with good cardiovascular fitness are at lower short-term risk of morbidity and premature mortality (US Department of Health and Human Services, 1996). But what will happen if we attempt massive public persuasion campaigns? Will we increase the numbers of anorexics? Will we increase the use of diet pills or weird diets? More bulimia? Will we have people injuring themselves running, or dropping dead from heart attacks? There are sure to be unintended consequences. A similar campaign was launched when John Kennedy was president, and now, 40 years later, many segments of the population are more obese and less fit than ever.

Many other health campaigns, similarly based on short-term and fragmentary evidence, are now underway. People are advised to use liberal doses of sunscreen when out in the sun. They may hear that an alcoholic drink a day is a good idea. They are advised to seek friends, go to church, stay married, meditate, lift weights, take vacations, get more sleep, eat breakfast, express their feelings, be cheerful, get more hugs, massage their children, floss their teeth, use disinfectant soaps, take supplements and herbs, and make other substantial (and often expensive) changes in their lives so that they will live longer. In all of these cases, there is mixed evidence, sometimes suggesting that the recommended interventional practices can be harmful, economically wasteful, or have unanticipated consequences over the long term. The clearest exception here is cigarette smoking, for which there is excellent evidence that avoiding or stopping smoking will improve health and longevity.

Scientific Inferences about Health

Much of the difficulty with health promotions derives from that abiding bugaboo of epidemiology, namely the conundrum that correlation does not mean causation. We observe associations among peoples, behaviors, customs, places, and health, but we do not usually know whether a corresponding intervention will have long-term salutary effects. For example, although it has been recognized for more than half a century that people better integrated into the community have better health, the implications for intervention are still unclear (Burg and Seeman, 1994; House et al., 1988; Stout et al., 1964).

Even with cigarette smoking, causal relations to health were controversial for decades, as we could not randomly assign half of the teenage population to be smokers, and then follow them for 50 years. What sort of evidence was finally mustered? First, there is a much higher incidence of disease and premature death among those engaging in the behavior. Second, there is clear temporal priority (e.g., smoking precedes lung cancer). Third, there is a dose to response relationship (heavier smokers have greater risks). Fourth, the relationship is consistent with other existing physiological knowledge (cigarette smoke has substances that damage living cells). Fifth, the association is consistent in different populations (men, women, in different ethnic groups, and in countries around the world). Sixth, there are animal analogs. Seventh, intervention seems to have an effect (people who stop smoking often have

better subsequent health than those who continue smoking). Together, these sorts of evidence almost completely rule out competing explanations for the observed relationship between smoking and cancer and premature mortality, and so make us very confident in our casual inference. Even here, however, it may be that there are complex relations among genetics, personality, smoking, and disease (Eysenck, 1985).

In an attempt to address the complexity, indeed messiness, of the naturally occurring interactions of individuals and varying environments, the medical community has increasingly turned to the randomized clinical trial. This has led to some odd, artificial, and perhaps dangerous studies. For example, the drugs tamoxifen and raloxifene are being studied (and used) for the prevention of breast cancer in healthy women who are at risk of breast cancer, despite sometimes significant side effects and risks (National Cancer Institute, 2001). Will we go down similar paths for personality and social psychology and health? That is, will we pursue similar litanies of healthy psychosocial characteristics? Will we then pursue drug or genetic interventions on personality and social relations?

How could we possibly pursue randomized clinical trials of personality, stress, social relations, and community? Should we make certain children more cheerful and optimistic, make certain adults more sociable and extroverted (preventive Prozac?), and test effects of divorce, recession, and community disharmony through randomized clinical trials? I hope not.

In many ways and for many reasons, the best means of ascertaining healthy lifestyles and understanding health-promoting life pathways is through long-term longitudinal study. By amalgamating the lessons of careful and comprehensive longitudinal research, a sensible and scientific approach to psychosocial health promotion can be constructed. Such longitudinal research often yields unexpected implications. The remainder of this paper reports illustrative findings from one such comprehensive effort, the eight-decade Terman Life Cycle Study.

The Terman Cohort

The Terman Gifted Children Study (later renamed the Terman Life Cycle Study) began in 1921–22 when most of the 1,528 participants were in elementary school. Continued until the present, it is the longest study of a single cohort ever conducted, and the only such major study with rich data collected regularly throughout the life-span (from childhood to late adulthood and death). My colleagues and I (especially Kathleen Clark, Michael Criqui, Leslie Martin, Joseph Schwartz, Carol Tomlinson-Keasey, and Joan Tucker) have made major efforts to follow up on and improve the data set. Data have been collected and refined on the subjects' social relations, education, personality, habits, careers, families, mental health, life stress, physical activities, and physical health; most importantly, we have collected death certificates and coded date and cause of death (Friedman et al., 1995c). Until our project began, the study

aimed primarily to describe the life course of gifted individuals (Terman and Oden, 1947). That is, the study was originally focused on addressing such issues as whether bright children were introverted eggheads (it turned out that they were not). Few predictive studies using the data had been undertaken, with little or no study of health as a function of individual differences. Because of the richness of the psychosocial data across many decades, and because of hard health outcomes (especially longevity and cause of death), these data provide an excellent opportunity to tease apart interacting factors relevant to health.

The Sample

Terman's aim was to secure a reasonably random sample of bright California children, and so most public schools in the San Francisco and Los Angeles areas in the 1920s were searched for bright children, nominated by their teachers and tested by Terman. The sample was later characterized as a productive, intelligent segment of twentieth-century middle-class American men and women. The average birth date was 1910. Most were pre-adolescent when first studied; those still living are now in their 90s. Most important is the fact that the data are collected prospectively, without any knowledge of the eventual health outcome, thus avoiding several common sources of bias in the data collection phase of such studies.

The sample is relatively homogeneous on dimensions of intelligence and social class. A resulting advantage is that these people had the ability to understand medical advice, had a place to exercise, had routine health care, and so on; the sample thus allows a clearer focus on the effects of psychosocial variables. The results are not directly generalizable to other groups, in other times, in other circumstances, but there is little reason to suspect that most relationships analyzed will be strongly influenced by the characteristics of this sample. For example, there is no reason to suspect that the relationship between personality traits and longevity is different for bright people than it is for people of average intelligence. (The sample is actually much more representative of the population than the various prospective studies that have followed samples of physicians or nurses.) The homogeneous nature of the sample might restrict the range on the predictor variables; however, our work shows that this is not the case for most variables of interest; there is generally a more than adequate range of individual differences and environmental stressors. Nevertheless, caution is obviously needed in generalizing from any single sample, especially when social or cultural variables are likely to affect a particular relation or finding. For example, socioeconomic status is very relevant to health in the US population as a whole, but is not so important in this restricted sample.

Overall, the data are remarkably complete. A low attrition rate of only 6 percent applies to most longevity analyses. Those lost from the sample did not differ in any known ways on relevant variables.

Neglect of Precursors and Complex Causal Pathways, Including Self-selection into Environments

I have noted that our health promotion efforts and public health systems are too often built around a pathology model, derived from traditional conceptions of “treating” disease. These approaches, which ignore the social context of people’s lives, often arise from the unrealistic causal models implicitly assumed. For example, they may say, “Here we have a person with high serum cholesterol and so we need to reduce cholesterol intake”; “Here we have a person who is overweight and so we need to teach weight loss skills”; “Here we have a person with high stress and we need to teach relaxation skills.” These approaches assume that the program begins at time zero – that you exist with certain risks at a certain point in time. But, in fact each person is on a certain trajectory that comes from previous characteristics and experiences, which are often quite different and unique. All overweight adults have not come from the same place, nor for the same reasons. So the causal *intervention* models are often wrong, or at least very imprecise or limited (Friedman, 1990; Suls and Rittenhouse, 1990).

Importantly, there is *self-selection* or pull into risk conditions. That is, people seek out healthier or unhealthier situations as a function of personality and pre-existing stress. I call the forces that pull some individuals towards healthy or unhealthy situations *tropisms* (Friedman, 2000). Just as phototropic plants move towards a source of light, some individuals grow towards more fulfilling and health-promoting spaces while other individuals remain subject to darker, health-threatening environments. A person’s personality and temperament (psychophysiological reactivity resulting from genes, early hormonal exposures, and early experiences) is not independent of the environment (Snyder and Cantor, 1998). For example, neuroticism (a tendency towards anxiety and depression) and aspects of temperament tend to predict to negative life events, thus making it misleading to think of personality, located within the individual, as randomly encountering various stressful or unstressful events (Bolger and Zuckerman, 1995; Magnus et al., 1993; McCartney et al., 1990; Scarr and McCartney, 1984; Van Heck, 1997; Wills et al., 2000).

Such more complex paths to health risks clearly emerge from analyses of the participants in the Terman Life-Cycle study. Let us first consider a significant factor of adulthood stability and stress, and then consider certain relevant aspects of personality.

Marriage and Divorce

Numerous epidemiological studies have found that married individuals, especially married men, have a significantly lower mortality risk than single and divorced individuals. It is usually assumed that this association reveals a protective effect of marriage. Perhaps a spouse serves as a buffer against stress.

Perhaps a spouse helps insure co-operation with medical regimens like taking pills on time. Perhaps a spouse is quick to call for emergency help when needed. (In fact, there is also evidence to support each of these associations – Friedman, 2002.) But studies of causal mechanisms have been difficult without access to a lifelong study. The resulting advice is “get married” or “stay married” to be healthy, an inference not justified by such data.

Using the Terman archives, supplemented by death certificates we collected and coded, the association between marital history at mid-life and mortality (as of 1991) was studied in the sample of participants (Tucker et al., 1996) ($N = 1,077$). As of 1950 (when they were about 40 years old), the vast majority of the participants were alive, mature, and had married if they were ever going to marry. We classified them as to whether they were currently and steadily married ($N = 829$), married but not in the first marriage (inconsistently married) ($N = 142$), never married ($N = 102$), or currently separated, widowed or divorced ($N = 70$). Very few had been widowed by this point.

Results confirmed that consistently married people (especially men) live longer than those who are single due to marital breakup. But intriguingly, the results suggested that this is not necessarily due to the protective effects of marriage itself. Controlling for gender and self-reported health, we found (in survival analyses) that the inconsistently married people were at higher risk for premature mortality than the steadily married, and that the currently split people were at even higher risk. Inconsistently married men had a relative hazard of mortality of almost 1.4 (40 percent greater risk), and separated or divorced men had a relative hazard of 2.2. That is, men who were currently married, but had previously experienced a divorce, were at significantly higher mortality risk compared with consistently married individuals. Since both groups were currently married (in 1950), the marriage itself could not be the relevant protective factor. Furthermore, controlling for number of years married had minimal effect on the association between marital history and mortality risk.

Since divorce is recognized as one of the greatest social stressors, perhaps the stress of the divorce harms health or sets in motion other harmful behaviors. If this is true, this divorce effect may dissipate over time, as those who erred (or had bad luck) the first time around, settle into stable remarriages. In fact, this is the case, as men who experienced marital dissolution and remarried were at higher risk prior to age 70, and then their relative mortality risk declines (Tucker et al., 1999). To the extent that the stress of divorce increases mortality risk, strong advice to “get married” (for social support) ironically may increase rather than decrease one’s risk, since one cannot face the stress of divorce if one has not married.

What about tropisms, the pull into certain social environments? Interestingly, part of the relationship between marital history and mortality risk in the Terman participants may be explained by childhood psychosocial variables, which were associated with both future marital history and mortality risk (Tucker et al., 1996). Some people evidently are poor bets both for stable marriage and a long life.

In sum, it is possible that the stress of divorce and its concomitants, coupled with selection into stable or unstable married roles, are more important health mechanisms than the sustenance provided by marriage itself. An incorrect causal inference might be drawn from simple observation of the association between marriage and health. And an invalid, simple preventive intervention (“Get married to promote health”) may be designed.

Precursors

What are these lifelong pathways that the adults with a consistent and stable marriage are traveling? In other words, where have they come from, both psychologically and socially? Individuals who were divorced or remarried reported (retrospectively) that their childhoods were significantly more stressful than those who got and stayed married. (They scored highly on such items as “marked friction among family members during childhood.”) Is there any more objective, prospective evidence for this?

Because there has never before been a lifelong prospective study of family stress predictors of mortality risk, the Terman cohort provides a unique opportunity to examine longer-term pathways. Family stress (particularly parental divorce) is known to predict unhealthy behaviors such as smoking and drug use in adolescence as well as poor psychological adjustment (Amato and Keith, 1991; Block et al., 1988a; 1988b; Chassin et al., 1984; Hawkins et al., 1992). Could such detrimental effects of parental divorce reach across the lifespan and affect (or at least predict) one’s own marital relations and eventual mortality risk?

Divorce of one’s parents during childhood can certainly affect one’s future mental health. There is good longitudinal evidence that children of divorce, especially boys, are at greater risk for observable behavior and adjustment problems (Amato and Keith, 1991; Block et al., 1988a; Hetherington, 1991; Jellinek and Slovik, 1981; Shaw et al., 1993; Zill et al., 1993). Most of the conceptual analyses concern a lack of social dependability or ego control – impulsivity and nonconformity, although neuroticism and low emotional stability are also often implicated.

We examined the Terman children ($N = 1285$) whose parents either did or did not divorce before the child reached age 21, who were of school age in 1922, and who lived at least until 1930 (Schwartz et al., 1995), using hazard regression analyses (survival analyses) to predict longevity, controlling for gender. Children of divorced parents faced one-third greater mortality risk than people whose parents remained married at least until they reached age 21. In light of the overwhelming evidence from other studies indicating damaging *psychological* impacts of parental divorce, this finding does provoke serious consideration. Death of a parent had very little effect, consistent with other research indicating that parental strife and divorce is a greater influence on subsequent psychopathology than is parental death (Tennant, 1988).

Importantly, the Terman study participants who experienced a marital breakup were more likely to have seen the divorce of their own parents.

Given that parental divorce is associated with one's own future divorce risk, and given that one's divorce is predictive of increased mortality risk, it is the case that one's unstable adult relations "explains" some of the detrimental effect of parental divorce. However, even after controlling for one's (adult) divorce, parental divorce during childhood remains a significant predictor of premature mortality, suggesting that it may have additional adverse consequences in adulthood.

Is childhood personality also relevant to these pathways? Indeed, part of the association between marital status and mortality risk seems to be due to a selection into steady marriages. Terman participants who were impulsive children, grew up to be both less likely to be consistently married and more likely to die younger (Tucker et al., 1996; 1999).

Thus, there do seem to be precursor selection effects at work. Childhood impulsivity and parental divorce predicted marital instability, and these are also predictive of earlier mortality. These variables explain some of the mortality differential between consistently and inconsistently married participants.

Personality

Perhaps we should therefore turn to personality as a key determinant of health. Here too we find that long-term patterns are most important.

Sociability

As a more general aspect of the well-documented associations between marriage and health, a large amount of evidence establishes that people with various personal and community ties, usually termed *social support*, are generally healthier (Cohen, 1991). It thus seems sensible that more sociable people would be healthier, and that development of sociability in children and adolescents should be encouraged. This conclusion again neglects precursors and complex causal pathways, including self-selection into environments. It turns out that there is little evidence that sociability itself predicts health and longevity. This is confirmed by the Terman data.

In 1922, the participant's teacher and parents (usually the mother, or both parents together) rated the subject (on 13-point scales) on trait dimensions chosen to measure intellectual, volitional, moral, emotional, aesthetic, physical, and social functioning. The scales used are remarkably modern in their appearance. Several other rated variables from the 1922 Terman assessment were also chosen for their similarity to some of the 25 trait ratings. Based on correlational and factor analyses, we defined Sociability as: fondness for large groups, popularity, leadership, preference for playing with several other people, and preference for social activities such as parties. We later showed that Sociability was strongly related to Extraversion but also significantly correlated with Agreeableness, as measured by the NEO Personality Inventory (Martin and Friedman, 2000).

In terms of life-span mortality risk, the Terman children who were rated by their parents and teachers as popular, fond of large groups and social activities, and so on did not live longer than their unsociable peers (Friedman et al., 1993). There was simply no evidence that sociable children were healthier or lived longer across many decades. In fact, sociable children were somewhat more likely to grow up to smoke and drink (Tucker et al., 1995).

To confirm this finding, we also examined Terman's own grouping of the men in the sample into "scientists and engineers" versus "businessmen and lawyers." Terman found marked personality differences, with the former group much more unsociable and less interested in social relations at school and in young adulthood. When we analyzed mortality risk, however, we found the scientist and engineer group at slightly *less* risk of premature mortality (Friedman et al., 1994). Examination of the pathways and tropisms suggests that these studious men often wound up well adjusted, working in positions well integrated into society.

Conscientiousness and Neuroticism

Conscientiousness – a tendency to be prudent, planful, persistent, dependable – is not highly related to the personality measures typically used in health research (Friedman et al., 1995a; Marshall et al., 1994). It turns out, however, to be relevant to understanding pathways to health.

Teachers and parents rated the Terman children on items that formed a scale of "Conscientiousness-Social Dependability" (comprised of prudence-forethought, freedom from vanity-egotism, conscientiousness, and truthfulness). This childhood measure was a good predictor of mortality risk across the life-span (Friedman et al., 1993; Tucker et al., 1995). Survival analyses suggest that the protective effect of conscientiousness is not primarily due to accident avoidance, although injury deaths do tend to be higher among the unconscientious. Conscientiousness seems to have more far-reaching and general effects. Childhood unconscientiousness predicts a host of unhealthy mechanisms and tropisms, including adult smoking, adult alcohol consumption, and less social and work stability and accomplishment. Subsequent studies by others confirm the health importance of conscientiousness. For example, a study of conscientiousness and renal deterioration in patients with diabetes found that time to renal failure was much longer in those with high conscientiousness (Brickman et al., 1996).

Interestingly, Conscientiousness, which exhibited the strongest predictive power in childhood was also the best predictor of mortality risk when personality was assessed in adulthood (Friedman et al., 1995c; Martin and Friedman, 2000). Yet childhood conscientiousness was reliably, but not strongly, related to adult conscientiousness ($r = 0.13$). This set of findings points again to the need to look at the larger context. In childhood, conscientiousness as measured by parental ratings is a key personality predictor of longevity, and in adulthood, conscientiousness as measured by self-report items is a key predictor of longevity. Yet this is more of an orientation to life than a "risk

factor" like serum cholesterol. (Note that both of these conscientiousness measures are highly associated with both rational judgments about what it means to measure conscientiousness and with NEO PI-R measurement of Conscientiousness – Martin and Friedman, 2000).

What about neuroticism, since there is all sorts of evidence that many diseases are associated with higher levels of hostility, anxiety, and depression (Friedman, 1991/2000; Friedman, 2002; Friedman and Booth-Kewley, 1987)? Having more emotional stability as a child was somewhat protective in the Terman sample, but adult neuroticism did not turn out to be a simple risk factor for earlier mortality. (And permanency of moods in childhood was not strongly related to adulthood neuroticism.) It may be the case that there are two or more types of health-relevant neuroticism. For example, an unhealthy neurotic may smoke, drink, take pills, oversleep, overeat, and seek self-destructive pleasure, all in an attempt to reduce anxiety, improve depressed mood, or cope with feelings of anger. A healthy neurotic, on the other hand, might direct worry and anxiety toward avoiding germs, seeking lots of medical care, wearing seat belts, saving money, buying insurance, and so on. Furthermore, some people thrive on challenge and competition, and so there are "healthy Type A's" (Friedman et al., 1985). The construct of neuroticism may be too broad to distinguish such subtypes, without knowing more about the situation and life pathway. For example, among the Terman children, those neurotics who grew up in stable families were not more or less prone to premature mortality; but those neurotics who faced parental divorce were at increased risk (Martin et al., submitted).

A further example comes from religiosity, sometimes offered as the royal road to health. The Terman women who viewed themselves as more religious in adulthood (approximately age 40) had a somewhat lower risk for premature mortality over the next several decades than those who were less religiously inclined. These women had healthier behaviors, more definite purposes and goals, more positive feelings about their futures, and reported being somewhat happier than their less religiously inclined peers. But such women were so inclined in childhood, often grew up in more positive families, joined more organizations, smoked less, drank less, and so on. In this particular circumstance of twentieth-century middle-class women, religiosity appeared to be part of a generally healthy lifestyle, but not a direct cause of it (Clark et al., 1999).

Even gender effects can be complex. As is typical, females do significantly outlive males in the Terman sample. However, in both men and women, individuals who were more male-typical in their occupational preferences tended to show higher mortality rates than individuals who were more female-typical. These associations were not due to a specific cause of death (Lippa et al., 2000).

Generality

As noted, the Terman Life-cycle sample is not directly representative of the current US population. First, as in any longitudinal cohort study, the Terman study participants were born in a certain era and grew up in specific social

times. Second, Terman sampled only bright Californians, and few ethnic minorities were present in the classrooms Terman sampled, often because they were not allowed to be present. It is therefore important to ask what limits on generality may result. Any variable that impacts both our predictors and our outcomes could alter the relations. For example, the sample members have good education, access to medical care, and ability to understand the American medical system and medical prescription; therefore in no case should the findings be directly generalized to people who face significant deficiencies in any of these areas. In addition, *effect size* estimates from this research should not be directly generalized to the US population as a whole. Nevertheless, there is a wide range of usual personality, life challenges, and social relations within the sample, and so it is well suited to explore such issues. It is especially valuable for pointing out some of the complexities that occur as certain life-paths unfold across time. Although bright children growing up in California in the 1920s faced some unique challenges and so the results should not be carelessly generalized to other groups of people in other historical contexts, it is also the case that the findings fit in an understandable way with what is already known about the correlates of better or worse mental and physical health.

Co-morbidity

One would undoubtedly find it odd to be administered a treadmill test for cardiovascular fitness as a screening test for cancer. Activity and fitness are believed to be ways to prevent cardiovascular disease, by lowering blood pressure, raising levels of high density lipoprotein, decreasing reactivity, reducing stress, improving fat metabolism, and a host of other postulated (and often documented) mechanisms to keep arteries clean and supple. Yet a large prospective study of middle-aged men, not atypically, found that physically fit men (as assessed by maximal oxygen uptake at baseline, and also by exercise test duration) were much less likely to die prematurely not only from cardiovascular disease, but also from all-causes and non-cardiovascular causes (Laukkanen et al., 2001).

This study was not published in an oncology journal. Analogously, a study of activity effects (or stress effects) on immune system response to tumor growth would not be published in a cardiology journal. Yet, recently, many in the biomedical research community have come to be surprised by what are termed "co-morbidity effects." This usually means that people at high risk of or having a high incidence of one disease are also at high risk of or have a high incidence of other, seemingly unrelated, diseases. People with so-called mental diseases are more likely to have so-called physical diseases (and vice versa). People with diabetes are more likely to have cardiovascular disease, and so on. In the psychological sphere, it is not only the case that hostile people are at higher risk of cardiovascular diseases and depressed people are at higher risk for cancer, but that hostile people are at higher risk of cancer and depressed people are at higher risk for cardiovascular diseases (Friedman, 1998; 2002).

Such findings are only surprising if you are a cardiologist who never studies cancer, an oncologist who never studies heart disease, a cancer-prevention psychologist who never studies diabetes, or a diabetes prevention-and-control psychologist who never studies cancer. They are not surprising to health psychologists studying resilience and self-healing, nor are they surprising to many developmental health psychologists. For example, Jessor's work on adolescents clearly demonstrates that those who like and value school, participate in family and church activities, have good kids as friends, and value health are more likely to engage in a host of healthy behaviors like healthy diet, exercise, and seat-belt use (Jessor et al., 1998). Although these conclusions, which I term *co-salubrious* effects, seem eminently sensible when pointed out in this manner, many health promotion conceptions are not socially or developmentally or contextually sensitive.

Conclusion

What conclusions can be drawn? First, we need to examine individual life patterns. Rather than taking a piecemeal approach, rather than educating people about endless lists of things not to do, it may prove more efficient and effective to launch people onto healthy life paths, and intervene intensively only for those few people at special high risk. Although the proof is not yet in, it may be that the more likely people are to be doing a few important things earlier in life, the more likely it is that other healthy styles and behaviors will follow later in life.

Second, we need greater focus on the social context – the person in the situation, and situation selection. This means studying the match between people and their environments, and why people wind up in certain unhealthy environments. In many ways, a self-healing personality is one in which there is a healing emotional style involving a match between the individual and the environment, which maintains a physiological and psychosocial homeostasis, and through which good mental health promotes good physical health (Friedman, 1991/2000).

Third, we need to consider cultural changes, both in the medical culture and in the broader societal culture. In terms of medical culture, we also need to break down the walls between different health institutes and narrow approaches to disease. We need to include *overall* health (not going system by system or disease by disease), as well as overall quality of life, as outcomes in our research.

In the broader societal culture, we need to recognize the complexities of socialization. As one example, there is a lot of smoking and a lot of lung cancer in Kentucky, but little smoking and little lung cancer in Utah. Should we spend a lot of time and money on designing anti-smoking newspaper ads in Kentucky, or might we focus more on comparing the tobacco farm southern culture to the LDS (Mormon) culture of Utah?

How is culture changed? It is not just more education. Rather, structural changes are often more efficient and effective. Yet no one objects to spending

billions and billions on treating cancer, and millions and millions on research on cancer, but how about subsidizing the many public health structures that affect behavior? But it is not only a role for government. The cruise industry is booming as people spend thousands of dollars to sit and eat 24 hours a day, but they do not have time or money to stay in shape, swim with their children, cook dinners, or go to church or their yogi. These are lifelong community values.

In sum, when psychosocial aspects of health are considered at a deep and time-sensitive perspective, we already know a lot about how to promote health, and it does not mainly involve campaigns against eggs, more warning labels on margarine, or even more exercise campaigns. Although we do need to keep researching healthy behavior and nutrition, physiology, immunology, infections, safety engineering, and so on, that is not where many of the greatest payoffs likely will come. Rather, the Terman data and many other sources of information suggest that stable people, well-integrated socially and with their community, living in a healthy culture – a healthy lifestyle across the life-span – will mostly have long, productive lives. But the context for each individual cannot be ignored. The bottom line is that psychosocial and behavioral factors look different in their relation to health when they are considered across the context of the life-span, than they do when considered at one point in time.

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