

## Longitudinal Findings From the Normative Aging Study: III. Personality, Individual Health Trajectories, and Mortality

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Individual differences in physical and psychological health trajectories were examined in 1,515 Normative Aging Study men. Mean age at baseline was 47.15 years (range = 28–80), and average follow up was 18.55 years (range = 8–25). Both linear and nonlinear growth curves were estimated with random-effects models and then clustered to identify patterns of change. Men whose physical health trajectories were characterized by high, increasing symptoms were higher in hostility and anxiety, were overweight, and smoked. Those whose trajectories were characterized by low symptoms were emotionally stable, educated, nonsmokers, and thin. Men with high, stable psychological trajectories had high hostility; those with low, stable trajectories had high emotional stability; those with moderate anxiety levels had nonlinear trajectories with peaks in psychological symptoms at different life stages. Personality had life-long effects on health trajectories, but these effects varied across traits and health outcomes.

A truism in adult development is that individual differences increase with age (Maddox, 1987; Nelson & Dannefer, 1992). For example, some individuals show little age-related decrement in physical health until very late in life, whereas others begin to show marked impairment in middle age. In other words, there are individual differences in developmental trajectories across the life span. Attempts to test “hard” developmental stages—ones that are thought to be universal, sequential, and irreversible—have found that it is necessary to examine both individual and contextual differences in adult change (Featherman & Lerner, 1985; Ford & Lerner, 1992; Levenson & Crumpler, 1996). In other words, one model does not adequately characterize the life course of all individuals. There are individual differences in how transitions

such as career entry, parenthood, retirement, and divorce are traversed (Abeles, Steel, & Wise, 1980; Belsky & Rovine, 1990; Bossé, Levenson, Spiro, Aldwin, & Mroczek, 1992; Chiriboga & Catron, 1991) as well as contextual differences in the effects of occupational mobility on health (Mare, 1990; Pavalko, Elder, & Clipp, 1993).

In personality research, questions of whether or not personality changes have gradually given way to determining who changes, which aspects change, and how much a given aspect changes at any given developmental phase (Aldwin & Levenson, 1994; Jones & Meredith, 1996; Lachman, 1989; Mroczek & Spiro, 1999; Roberts & DelVecchio, 2000). Similarly, in health research, there is general acknowledgment both that personality affects health (Friedman & Booth-Kewley, 1987) and that there are individual differences in health trajectories across the life span (Nowlin, 1985). However, very few studies have examined the link between personality and health trajectories across the life span.

### Personality and Health

There is a widespread acceptance of the idea that personality has effects on health and longevity. A variety of studies have shown consistent, albeit modest, effects of personality, especially hostility and neuroticism, on the incidence and prevalence of illnesses such as cardiovascular disease, cardiovascular reactivity, predilection toward poor health behaviors, adaptation to illness, and compliance with medical regimes, all of which can eventually affect mortality. (For reviews see Friedman & Booth-Kewley, 1987; Friedman, Hawley, & Tucker, 1994; Scheier & Bridges, 1995; Spiro, Aldwin, Ward, & Mroczek, 1995; Van Heck, 1997; Wiebe & Smith, 1997.)

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This study was supported by National Institute of Aging Grant AG13006 and by the Department of Veterans Affairs Cooperative Studies Program (CSP/ERIC).

The Normative Aging Study is a research program of the Massachusetts Veterans Epidemiology Research and Information Center (MAVERIC). Portions of this study were presented at the Annual Meeting of the American Psychological Association in 1999. We thank Leanne Friedman for her help with data analyses and Keith Widaman and Elizabeth Clipp for their helpful comments on earlier drafts.

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Many, if not most, of the studies that examine the relationships between personality and health are cross-sectional, and those that are longitudinal are often prospective, considering whether personality at some point in time predicts subsequent morbidity or mortality, rather than truly longitudinal, examining repeated measures on relevant constructs. In other words, they examine whether a personality trait assessed at the onset of a study is associated with higher relative risks for a particular disease or mortality. What exactly goes on between these two points in time remains a mystery. For example, one can speculate that the relationship between hostility and cholesterol levels (Niaura et al., 2000) will, over time, explain at least some of the association between hostility and heart disease, but very few studies have examined the relationship between personality and health trajectories.

### Health Trajectories in Adulthood

Most studies of psychosocial factors and health in older adults simply control for the effects of age or examine only linear relations between age and health. However, a nonlinear relationship between age and mortality has long been recognized (i.e., survival curves), and other health measures may also exhibit nonlinear trends with age. Recognizing the complex, nonlinear relationship between age and health may yield a more complete understanding of the nature and etiology of individual differences in health across the life span.

There are relatively few studies that examine nonlinear trajectories in adulthood, as well as the factors modifying those trajectories. Those that do tend to use relatively small samples and identify trajectories on the basis of visual inspection (Barnett, Marshall, & Singer, 1992; Belsky & Rovine, 1990). For example, Clipp, Pavalko, and Elder (1992) coded open-ended data on physical and mental health to identify trajectories over a 40-year period. They used eight assessments for the 634 men in the Terman Study who had complete data over this time period. Using visual inspection,

they grouped the trajectories into six categories (see Figure 1). Five categories described the physical health trajectories: constant good health, constant poor health, decline at end of life, linear decline, and decline and recovery. For psychological symptoms, however, they added a sixth category, variable problems. However, given that this study was limited by its reliance on open-ended data, it is possible that a variable category might also be found for physical health if other measures were used.

For mental health, the trajectory groups differed only in marital status, with single men more likely to have constant poor mental health. However, physical health trajectories varied as a function of cause of death. For example, individuals who died from massive heart attacks may have had little or no period of disability, whereas emphysema or cancer victims tended to have a definite decline in health. Clipp et al. (1992) also found age and cohort effects on physical health trajectories but no effect of marital status. Men with more education were more likely to be characterized by constant good health. The older cohorts were slightly more likely to exhibit linear declines, whereas the younger cohorts were more likely to report declines followed by improvements, which were seen as reflecting improvements in medical treatment, or perhaps recovery from acute conditions.

Maddox and Clark (1992) used growth curve models to examine aggregate changes in functional impairment by using data from the Longitudinal Retirement History Study, which followed employed men and employed, unmarried women. From an initial panel of over 11,000, they selected 2,728 people who fit three criteria: no missing data on any of the six assessments, aged 58–68 years in 1968, and consistently poor or “nonpoor” over the decade studied. Functional ability was assessed with a 4-point scale that assesses the presence or absence of impairment in three different domains. Maddox and Clark fit polynomial growth curves to the sample as a whole and found many quadratic, cubic, and higher order terms, which further interacted with sex, economic status, and education.

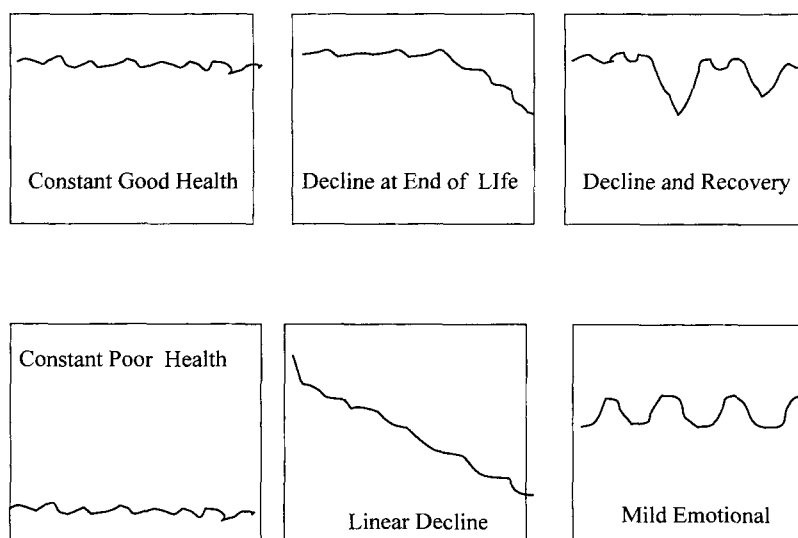


Figure 1. Proposed patterns of health trajectories. Adapted from “Trajectories of Health: In Concept and Empirical Pattern,” by E. C. Clipp, E. K. Pavalko, & G. H. Elder, Jr., 1992, *Behavior, Health, and Aging*, 2, p. 166. Copyright 1992 by Erlbaum. Adapted with permission.

Women, the poor, and those with little education reported higher levels of disability over time; however, the sex differences were eliminated once education and economic status were controlled. Interestingly, few accelerated curves were seen; rather, asymptotic curves were more the norm. Although this could be seen as supporting Manton and Stallard's (1990) hypothesis of extended impairment, it might also be due to a ceiling effect, given the limited range of their measure of functional ability.

A handful of studies have examined individual physical health trajectories over shorter periods of time. Rodin and McAvay (1992) examined self-reported health at eight occasions over 2 years in 264 older men and women. However, they simply grouped individuals into those whose self-rated health was stable and those who experienced a decline. Those who had new illnesses, increased their number of doctor visits, increased in depression, and decreased in self-efficacy were more likely to demonstrate declines in self-rated health.

Verbrugge and Balaban (1989) examined health and activity trajectories over a 2-year period by using health diary data in 117 older men and women. Although they found considerable variation in trajectories, their scoring indicated only whether a shift had occurred, and they plotted linear slopes to characterize those trends. Fitting linear slopes to these highly variable data appears to have resulted in many zero slopes, as the positive and negative changes canceled each other out. Nonetheless, nonmarried people tended to have poor health trajectories and lower activity levels. A subsequent study by Verbrugge, Reoma, and Gruber-Baldini (1994) followed 165 persons with chronic morbidity for 1 year post hospitalization. Using visual inspection, they found substantial fluctuation in individual health trajectories and concluded that "changes in function for the chronically-ill persons are seldom linear" (p. 106).

An interesting study by Tennen and Affleck (1996) used daily diaries to assess pain. They found that aggregate (or nomothetic) analyses often yielded relatively few correlates of pain but that ipsative analyses often yielded interesting associations between personality processes and fluctuations in pain. This and other work has spurred a reexamination of the relative strengths of nomothetic versus ipsative or idiothetic analyses, which focus on how an individual changes over time (Lamiell, 1981). Some researchers are beginning to focus on methods bridging the two approaches, such as growth curve and random-effects models (e.g., Affleck, Zautra, Tennen, & Armeli, 1999; Collins & Horn, 1991; Gottman, 1995; Willett, 1988).

These methods examine individual trajectories of change over multiple occasions and allow one to examine predictors of those changes (cf. Aldwin, Spiro, Levenson & Bossé, 1989; Almeida & Kessler, 1998; Ge, Conger, & Elder, 1996; Mroczek & Spiro, 1999; Spiro, Aldwin, Levenson, & Bossé, 1990; Steiger, Gauvin, Jabalpurwala, Séguin, & Stotland, 1999).

In the first of a series of papers from the Normative Aging Study (NAS), Aldwin et al. (1989) examined the relationship between age and health by using the Cornell Medical Index (CMI; Brodman, Erdman, & Wolff, 1956). Analyses based on a nomothetic perspective found only a weak relationship between age and physical symptoms. However, Aldwin et al. also conducted individual analyses, computing growth curves or trajectories for each individual, fitting as many as eight observations per person over 22 years. Analysis of individual growth curves revealed marked

changes with age in physical health symptoms. On average, the men reported an increase of one new physical symptom every 3 years. Mental health, however, appeared to be relatively stable. Nonetheless, there were substantial individual differences in trajectories in adulthood, and a subsequent study began to address these differences.

The second study examined whether neuroticism predicted the extent of changes in these linear health trajectories (Spiro et al., 1990). Although neuroticism was moderately correlated with baseline physical symptoms, it only weakly predicted changes over time. However, both of these studies were limited by examining only a small set of predictors and covariates of change and by their restriction to linear trajectories.

Another approach would be to examine patterns in individual assessments of change. For example, Block (1971) identified different patterns of ipsative change in personality. Magnusson (1998) has argued that identifying patterns of change in individuals allows one to take a more holistic approach and to examine combinations of variables within individuals, whereas Bergman (1998) suggested that cluster analyses may be particularly useful in identifying basic patterns of change.

### Present Study

This study has two purposes. First, we wished to extend the examination of individual differences in health trajectories by identifying different types of patterns of change with age, in particular to examine whether the nonlinear patterns suggested by Clipp et al. (1992) and Maddox and Clark (1992) could be seen by using a more parametric measure of health. Using random-effects models, we fitted polynomial growth curves, including both quadratic and cubic terms. We then applied cluster analysis to the growth curve estimates for each individual to identify different patterns of change. This approach was inspired by three studies that used clustering techniques on individual growth curves: an early study of cognitive change by McCall, Appelbaum, and Hogarty (1973); one on sales performance by Hofmann, Jacobs, and Baratta (1993); and one on changes in self-esteem in adolescence by Hirsch, DuBois, and Brownell (1993). However, all three of these studies used relatively small samples, and none focused on health. Nonetheless, they illustrate the usefulness of this technique (see also Smith & Baltes, 1997).

Second, we were also interested in whether different health trajectories reflected individual differences in personality, as well as in sociodemographics and health behaviors. Based on prior research, we expected that men in the trajectory clusters associated with poorer longevity would report higher levels of hostility and anxiety, whereas those in trajectories associated with better longevity would be more emotionally stable. We were particularly interested in whether personality affected general symptom levels (e.g., higher or lower) or was associated with different types of curves. For example, would the effects of hostility be seen primarily in midlife, when heart disease starts to become prevalent, or could it be traced throughout the life span?

## Method

### Sample

The NAS was founded in 1961 to examine aging processes in normal, healthy men. More than 6,000 men were screened between 1961 and 1968 for good health (i.e., absence of chronic disease, blood pressure no greater than 140/90, and evidence of "geographic stability," defined as having extensive kinship ties in the area and stated intentions to remain in Boston). The sample was generally representative of the Boston population in the late 1950s, and most of the men were of European extraction (Bossé, Ekerdt, & Silbert, 1984). The original NAS population was 2,280 men with an age range of 21 to 80 years at entry into the study, with an average age of 42. About 90% of the sample was between the ages of 30 and 60. Ignoring the 180 men who were lost to follow-up (most occurred between the first and second exams), the panel consisted of 2,100 men.

Because we considered nonlinear trajectories, we included only men who had four or more observations after enrollment.<sup>1</sup> (We excluded the initial observation at enrollment because it was used for screening purposes.) This differentially omitted some of the older individuals, and thus our study focuses more on change among the middle-aged and the young-old adults. The resulting sample included 8,804 observations on 1,515 men, with a mean age at baseline (second exam) of 47.15 years ( $SD = 8.50$ , range = 28–80).

### Procedure

At study intake (1961–1970), the men received a physical examination and completed the CMI, a self-report inventory of symptoms and illnesses (Brodman et al., 1956) and the Social Screening Questionnaire (Rose & Bell, 1971), which assesses demographics. Personality was assessed subsequently (1965–1967) on only half of the men, those who completed the 16 Personality Factor Questionnaire (16PF; Cattell, Eber, & Tatsuoka, 1970) in small groups.

Every 3 to 5 years after enrollment, the men completed the CMI at home, with older men (52 years or more) completing questionnaires more frequently (at 3- rather than 5-year intervals). Thus, the health behaviors, demographics, and personality variables used to differentiate among the symptom trajectory clusters were measured before the health assessments, which began between 1968 and 1975. The men were followed for an average of 18.55 years (range = 8–25).

### Measures

**Self-reported health.** The CMI (Brodman et al., 1956) was used to assess self-reported physical and psychological symptoms. It consists of 195 dichotomous items assessing illnesses, symptoms, family health history, and health behaviors. Physical symptoms (144 items) are divided into 12 scales assessing different organ systems (e.g., sensory, respiratory, and cardiovascular). The CMI also contains 51 items assessing mental health symptoms. The NAS uses a slightly modified scoring system (see Aldwin et al., 1989). Five items assessing health behaviors (e.g., smoking and drinking) and 7 items assessing family history of physical illness are omitted from the physical symptom score. Five items assessing family history of mental illness are omitted from the psychological symptom score. Scores are computed by summing the remaining items separately for physical and for mental health.

**Sociodemographic variables.** Marital status, educational attainment, and ethnicity were assessed at entry into the study. These variables were dichotomized into married/nonmarried, high school or less versus some college or more, and White/non-White. Most of the men (90.71%) were married, about half (54.92%) had at least some college, whereas nearly all (98.23%) were White.

**Health behaviors.** At the initial examination, the men were interviewed concerning their smoking behavior and classified as current smokers,

never smokers, and former smokers. About a third of the sample (34.58%) reported smoking at study intake; an additional third (33.49%) were former smokers.

**Personality.** Forms A and B of the 16PF were collected on half of the NAS sample (1,111) men from 1965 to 1967; thus, only 784 men in the current sample had personality data. Each form of the 16PF consists of 187 trichotomously scored items, grouped into 16 scales; raw scale scores from Forms A and B were summed. Of particular interest are those scales most likely to be associated with health outcomes (see Friedman & Booth-Kewley, 1987; Spiro et al., 1995), especially those defining Cattell's Anxiety factor, such as Scales C (Emotional Stability or Ego Strength), L (Suspiciousness or Hostility), O (Apprehensiveness), and Q4 (Tension). There were no differences in education, occupation, or alcohol or tobacco consumption between the NAS men who took the 16PF and those who did not. However, men who did not take the 16PF were on average 1 year older and were slightly heavier, with a body mass index (BMI) of 26.0 versus 25.5 (Spiro et al., 1995).

**Mortality.** Regular mailings to NAS participants are used to maintain vital status information; death certificates are obtained for all decedents. In the sample for this study, 400 men (24.0%) were deceased as of October 1998. For 371 of these men, we have obtained death certificates and coded them for cause of death by using ICD-9-CM (International Classification of Diseases, Version 9, Clinical Modification). Of these deaths, 149 (40.2%) were due to cancer, 143 (38.5%) were due to cardiovascular disease, and the rest were due to a variety of causes (e.g., respiratory disease, accidents, and suicides) that we coded as "other."

### Analyses

To identify patterns of change, we first used a random-effects model (Laird & Ware, 1982; Singer, 1998) to estimate individual growth curves for each outcome (CMI physical and psychological symptoms). A random-effects model estimates an overall growth curve for the sample and estimates individual growth curves as deviations from the aggregate curve. Next, we clustered the individual growth curves (i.e., the random-effects estimates) to identify patterns of trajectories. We computed and plotted the mean random-effects estimates for each cluster to characterize its trajectory. Finally, we categorized the men according to their cluster membership and used this membership as the independent variable to compare clusters on sociodemographics, health behaviors, personality, and mortality. Depending on the distribution of the variables, we used multivariate analyses of variance or chi-squares to determine whether there were differences among the trajectory groups. All analyses were conducted by using SAS (1996).

Figure 2 illustrates this process for two of the clusters. The top panels present the raw data; the bottom panels show the random-effects curves. Simply put, our analytical process smooths out the individual trajectories

<sup>1</sup> This is a restriction unique to the present situation because mixed models allow inclusion of cases with only one observation. However, because we were clustering the individual random-effects estimates, we needed each case to have estimates of the intercept, linear, quadratic, and the cubic terms. This could result in some additional bias to the sample, so we compared the 171 men who had only three visits with the 1,515 men in the sample. There were no differences in age, education, marital status, ethnicity, mental and physical health symptoms at baseline, nor in personality traits. The main difference was that the men with only three visits were more likely to be smokers (47.24% vs. 34.58%),  $\chi^2(1, N = 1,686) = 10.23, p < .001$ , and they were more than twice as likely to have died (45.61% vs. 17.49%),  $\chi^2(1, N = 1,686) = 74.99, p < .001$ . They were three times as likely as the men with four or more visits to have died of cardiovascular disease (21.64% vs. 7.00%) and twice as likely to die of cancer (18.71% vs. 7.72%),  $\chi^2(1, N = 1,686) = 76.14, p < .001$ .

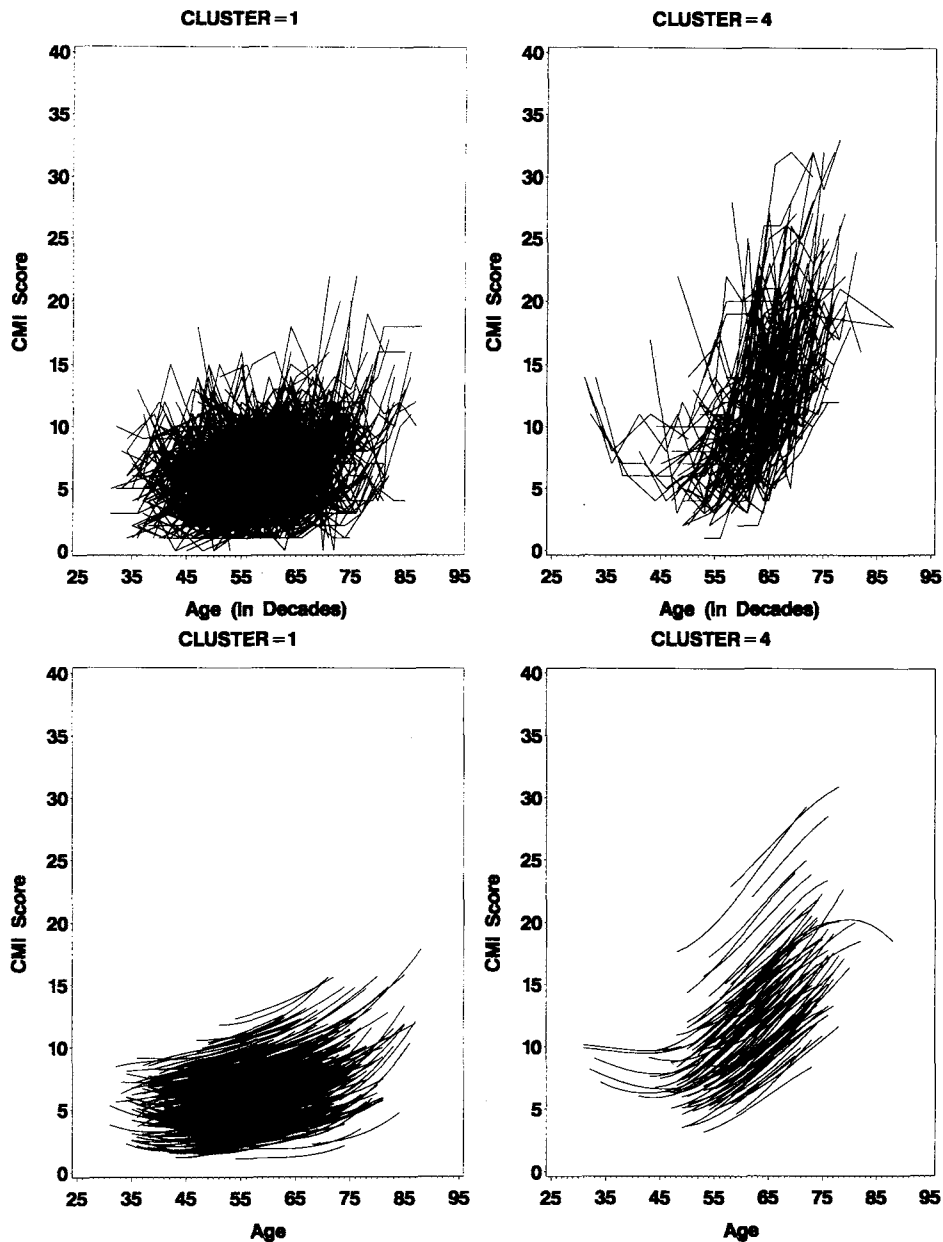


Figure 2. Raw data and predicted curves for two clusters. CMI = Cornell Medical Index.

and then groups them with others who have fairly similar trajectories in order to identify discrete patterns of change in symptoms across the life span. More detail about the analyses is provided below.

*Individual growth curves.* Random-effects models are ideally suited to long-term studies such as the NAS, because they can accommodate unbalanced data. That is, individuals can have varying start-up times and varying intervals between measurements, and missing data do not lead to omission of all data for an individual (Laird, 1988; Willett, 1988).

We fit polynomial growth curves by using maximum-likelihood estimation (PROC Mixed; SAS, 1996) for each outcome for the sample as a whole and allowed individuals to vary around the overall curve. Thus, each person is characterized by an individual growth curve (random effects) estimated as deviations from the overall curve (fixed effects). We used decades rather than years to increase the interpretability of the parameter

estimates. To decrease multicollinearity, we centered each term (Rogosa & Willett, 1985; Willett, 1988). Multicollinearity is especially problematic in polynomial equations, given that the squared and cubed terms are powers (or multiples) of age. The question of whether to center age at the overall sample mean or at the mean for each person is complex, but on the basis of arguments provided by Kreft, de Leeuw, and Aiken (1995; cf. also Mehta & West, 2000), we opted for an overall centering. Thus, we centered age at the midpoint of follow-up (age 55). We then divided age by 10 to convert age from years to decades; thus, slopes indicate change per decade.

For each outcome, we compared four progressively more complex models: intercept only, linear, quadratic, and cubic polynomials on age. The fixed-effects and random-effects specifications included the same terms (i.e., if we fit a cubic fixed effect, then the random effects also included the cubic term). Because of the irregular and varying intervals

between CMI, we allowed the covariances among random effects to be freely estimated (i.e., we fit an unstructured covariance matrix) rather than forcing a more restricted model. Thus, for the cubic model, we fit the following equation:

$$Y_{it} = B_0 + B_1t_i + B_2t_i^2 + B_3t_i^3 + v_{0i} + v_{1i}t_i + v_{2i}t_i^2 + v_{3i}t_i^3 + e_{it},$$

where  $Y_{it}$  = the predicted number of symptoms for individual  $i$  at age  $t$ ; the  $B$ s refer to the fixed effects (e.g.,  $B_0$  = intercept,  $B_1$  = the linear,  $B_2$  = the quadratic, and  $B_3$  = the cubic age effect). Note that because of the centering of age, when  $t = 0$ ,  $Y_{it}$  is the predicted number of symptoms at age 55. The  $v$ s are individual deviations from the overall curve (e.g.,  $v_{0i}$  = the deviation of individual  $i$  from overall intercept  $B_0$ , and so on). The term  $e_{it}$  represents the deviation of the individual's observed from his predicted curves at age  $t$ . The variances of the  $v$ s are the random-effects estimates. We identified the best model for each outcome on the basis of several considerations: the significance of the higher order random-effects estimates coupled with several measures of fit (Akaike's information criterion [AIC], the log likelihood [LL], and the residual variance).

The estimated intercept and slope parameters for each man were then used as dependent variables in subsequent analyses, usually regression analyses, to identify predictors of the slopes. Because a primary purpose of this study was to delineate patterns of change, we instead used the intercepts and slopes as dependent variables in a cluster analysis.

*Cluster analysis of growth curve parameters.* Prior to clustering the growth curve estimates, we set aside a small proportion (5%) of cases with a low probability of cluster membership, based on a preliminary  $k$ th nearest neighbor clustering. Our rationale was based on the arguments of Bergman (1988; cf. also Edelbrock, 1979; Goodman, 1975), which are that it may be difficult to classify all of the people all of the time. Some of these cases may be outliers because of errors in their data, or they may be members of relatively rare clusters that are underrepresented in a given sample. Omitting potential outliers often improves the accuracy of clustering algorithms (Edelbrock, 1979; Milligan & Cooper, 1987). Hofmann et al. (1993) omitted from their cluster analysis cases in which growth curve estimates were  $\pm 3$  SDs from the sample mean. Regardless of the rationale or the method, these residual cases can be treated as a separate cluster and compared to those that result from the cluster analysis (McCall et al., 1973).

The individual growth curve estimates obtained for each individual from the random-effects model were cluster analyzed to identify patterns of growth or trajectories. Our goal was to identify statistically (rather than visually) distinct clusters of growth curves, in contrast to the studies cited earlier. Separate analyses were conducted for the physical and the psychological symptom growth curves. We clustered the random-effects estimates of the intercept, linear, quadratic, and cubic coefficients for each person, clustering persons rather than variables.

Because a primary purpose of this study was to determine whether Clipp et al.'s (1992) visual identification of six clusters could be replicated by using statistical techniques, we initially extracted six clusters. In addition, we used two empirical criteria, the pseudo  $F$  and pseudo  $r^2$  (Milligan & Cooper, 1987) to determine the number of clusters. The pseudo  $F$  indicates the separation among all clusters at a given step in the analysis; the pseudo  $r^2$  indicates separation among the two most recently joined clusters. The number of clusters is determined by examining plots of the criteria against number of clusters, similar to examining a scree plot in factor analysis. For the pseudo  $F$ , one seeks a local maximum; for the pseudo  $r^2$ , a cluster where the value is low for a given step, but higher at the next step.

Correlation coefficients (as opposed to distance measures) of standardized data were used as input to the cluster program because they yield clusters that recover the pattern information (shape and scatter) while ignoring differences in elevation among clusters (Edelbrock, 1979; Overall, Gibson, & Novy, 1993; Scheibler & Schneider, 1985). We used a two-step approach to cluster the individual random-effects growth curve

estimates (Huberty, DiStefano, & Kamphaus, 1997; Smith & Baltes, 1997). First, Ward's hierarchical agglomerative method (PROC Cluster; SAS, 1996) was used to identify preliminary clusters; it was then followed by an iterative  $k$ -means clustering algorithm (PROC Fastclus; SAS, 1996). Ward's method is widely recognized as one of the best at recovering clusters within data (e.g., Edelbrock, 1979; Milligan & Cooper, 1985, 1987). Beginning with as many clusters as there are observations, Ward's method combines clusters to minimize the within-cluster sums of squares.

One limitation of Ward's method is that once observations are assigned to a cluster, they remain in that cluster, even though the appropriateness of that cluster for some members may have changed because of the addition of new members. Thus, we followed Ward's clustering with a second procedure, iterative  $k$ -means. The clusters identified by Ward's method were used as the starting seed, and observations were moved iteratively to the cluster with the nearest centroid, the centroids were recalculated, and additional moves were made until convergence was obtained. This is analogous to rotating the matrix in factor analysis. In factor analysis, many of the items load initially on the first few factors; rotation redistributes the items across factors. Similarly, in the initial clustering, many men began, and remained, in the initial clusters; the  $k$ -means clustering allowed men to move to more appropriate clusters.

## Results

The findings are presented separately for the two outcomes, physical and psychological symptoms. The results of the random-effects model estimation are described first. Then, we present the results of the cluster analysis of the random-effects estimates, followed by comparisons of the clusters on demographics, health behaviors, personality traits, and mortality.

### *Physical Symptom Trajectories*

For physical symptoms, we fit 8,804 observations on 1,515 men. On the basis of several criteria, we identified the cubic model as providing the best fit to the data. The residual variance decreased from the quadratic to the cubic model (from 10.497 to 10.107). Further, AIC and LL were best for the cubic model (AIC = -25,528.8; -2LL = 5,1035.50). Finally, all of the random-effects variance estimates for the cubic model were significant, although not all of the corresponding fixed-effects estimates were significantly different from zero.

The fixed-effects portion of the model is presented in the upper panel of Table 1. For the sample as a whole, only the linear effect of age was statistically significant ( $B_1 = 1.909$ ,  $p = .002$ ). Neither the quadratic (age<sup>2</sup>) nor the cubic (age<sup>3</sup>) effect was significant. The intercept was 10.096. Thus, at age 55, the NAS men reported about 10 symptoms, and they gained about 2 symptoms per decade.

However, the random-effects portion of the model (shown in the middle panel of Table 1) showed that there was significant variation among men in their average level (intercepts), as well as in the linear, quadratic, and cubic effects of age. The significance of these variance estimates (see the lower panel of Table 1) also suggested that some men deviated significantly from the overall curve in markedly nonlinear ways. That is, there were some men whose individual growth trajectories were quadratic or cubic and differed significantly from the average trajectory.

Table 1  
Results of Random Effects Models for Physical and Psychological Symptoms

Effect	Physical symptoms						Psychological symptoms					
	Estimate	SE	<i>t</i>	<i>df</i>	<i>Z</i>	<i>p</i>	Estimate	SE	<i>t</i>	<i>df</i>	<i>Z</i>	<i>p</i>
Fixed-effects estimates												
Intercept	10.096	3.381	2.99	1514		.0021	1.618	1.231	1.31	1514		.1891
Age	1.909	0.792	2.41	7286		.0159	-0.157	0.302	-0.52	7325		.6028
Age <sup>2</sup>	0.126	0.210	0.60	7286		.5494	-0.004	0.092	-0.05	7325		.9585
Age <sup>3</sup>	-0.036	0.150	-0.24	7286		.8107	0.039	0.072	0.52	7325		.5996
Random-effects variances												
Intercept	35.422	1.437			24.64	.001	7.492	0.089			-0.43	0.097
Age	5.543	0.580			9.56	.001	2.092	0.196			10.68	0.001
Age <sup>2</sup>	1.259	0.290			4.34	.001	0.432	0.106			4.08	0.001
Age <sup>3</sup>	0.064	0.027			2.33	.002	0.474	0.015			3.10	0.001
Residual	10.106	0.204			49.50	.001	2.712	0.055			49.17	0.001
Random-effects covariances												
Int, Age	4.125	0.644			6.41	.0001	0.089	0.162			0.54	.3864
Int, Age <sup>2</sup>	-1.793	0.304			-5.88	.0001	-0.426	0.083			-9.10	.0001
Age, Age <sup>2</sup>	-0.002	0.174			-0.01	.9915	-0.069	0.053			-1.30	.1936
Int, Age <sup>3</sup>	6.095	0.198			0.48	.6298	0.097	0.052			1.87	.0651
Age, Age <sup>3</sup>	-1.006	0.209			-4.81	.0001	-0.475	0.072			-6.57	.0001
Age <sup>2</sup> , Age <sup>3</sup>	-0.064	0.027			2.13	.0226	0.002	0.011			0.14	.8865

Note. Int = Intercept.

Figure 3 presents the predicted fixed-effects curve for physical symptoms, from ages 35 to 75.<sup>2</sup> Note that the overall trajectory is a weighted composite of the individual curves that extend over only a portion of the age range considered (cf. Schnurr, Spiro, Aldwin, & Stukel, 1998). The men initially ranged in age from 28 to 80 and were followed for an average of 18.55 years. Most men were in midlife at the start of the study; thus, graphing the change in symptoms from ages 35 to 75 captured the majority of the age range represented by the sample, without unduly weighting the relatively few men at the extremes of the age range.

In the fixed-effects model, only the linear slope estimate was significant. The predicted, composite curve shows that, on average, symptoms increased steadily from about 7 symptoms at age 35 to 14 symptoms at age 75. Examination of the random (or individual) effects, however, revealed that some men's growth curves were markedly nonlinear, requiring quadratic and cubic polynomials to describe the trajectories. This strongly suggested that there were different patterns in trajectories, and thus we used cluster analysis to identify the patterns.

On the basis of Ward's clustering of the random-effects estimates, the pseudo *F* and the pseudo *r*<sup>2</sup> suggested that there were six clusters, as hypothesized. In addition, we also retained as a "cluster" the outliers. Using the six cluster centroids obtained by Ward's clustering as seeds, an iterative *k*-means clustering reassigned 40% of the observations to another cluster, most from the initial Cluster 1 to Cluster 3 (*n* = 110), from initial Cluster 2 to Cluster 1 (*n* = 127), or from initial Cluster 3 to Cluster 2 (*n* = 144).

Figure 4 shows the mean physical symptom trajectories of these seven clusters. The general tendency was for physical symptoms to increase with age; however, there were marked differences among clusters in both the average level (number of symptoms) and in the shape of the trajectories, most of which were nonlinear.

Three of the trajectory clusters (1, 2, and 3) reflected the existence of relatively few (approximately five) symptoms in the mid-30s and are represented by the lines with symbols. Cluster 1 (low, slow increase) showed little increase in symptoms until age 63; Cluster 2 (low, moderate increase) increased at a slightly more rapid rate, especially in their 50s. Cluster 3 (low, rapid increase) was characterized by an asymptotic curve, with a relatively rapid increase in symptoms in the 40s, which then leveled off around age 55.

Two clusters (5 and 6; hatched lines) reflected moderate symptom levels (about 10) in the 30s but showed markedly different trajectories. Cluster 5 (moderate, rapid increase) showed a nearly linear and rather rapid increase in symptoms with age, leveling off only in the late 60s. In contrast, Cluster 6 (moderate, slow increase) showed only a very slow rate of increase in the subsequent decades.

Two clusters (0 and 4) reflected high symptom levels (about 15) in the 30s and are represented by broken lines. Cluster 0, the outliers, had the highest number of symptoms (nearly 15) at age 35 and increased, on average, linearly. At every age, the men in this cluster reported the highest number of symptoms of any group. In

<sup>2</sup> Note that we regressed symptoms against age in decades, centering at 5.5. Thus, the actual estimates were computed from -2.0 to 2.0; this was translated back into age for ease of interpretation. We also compared the fixed and random estimates between the present sample (four or more visits) and the larger sample that included men with only three visits. In the larger sample, the intercept was slightly higher, 10.161 versus 10.096, but the other estimates were slightly lower; but again, usually different only in the second decimal place. However, the cubic estimate would have decreased by a third (.024 vs. .036), supporting our decision to omit these men.

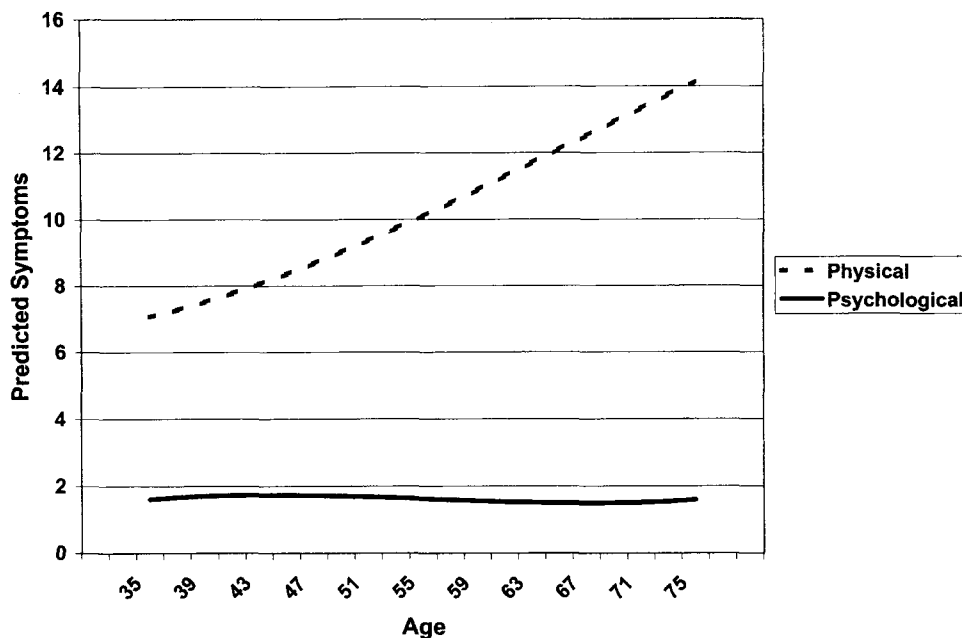


Figure 3. Population average trajectories.

contrast, Cluster 4 (high, U-shaped) showed an unexpected pattern: They started out with a high number of symptoms, decreased in their 30s and 40s, but then increased rapidly starting at about age 55.

We then examined the correlates and consequences of cluster membership. There were no significant differences in marital status among the clusters (results not shown), probably because nearly all of the NAS men were married at the beginning of the study. Table 2 presents the significant differences among the clusters in health behaviors and sociodemographic variables. There was considerable overlap in age ranges, with most groups ranging in age from about 30 to 75. However, there were differences in the average ages between the groups. Cluster 6 (moderate, slow increase) was the youngest, with an average age of 42 at baseline, whereas Cluster 4 (high, U-shaped) was the oldest, averaging 52 years of age at baseline. It is interesting that both of these clusters had relatively restricted age ranges compared with those in the other clusters (with the exception of Cluster 5). The respondents in Cluster 4 ranged in age from 31 to 65, whereas those in Cluster 6 ranged in age from 28 to 58. In contrast, Cluster 0 ranged in age from 29 to 80, and the others ranged from about age 30 to 75.

As Table 2 also indicates, Cluster 1 (low, slow increase) was the best educated, with over 60% having at least some college; the outliers were the least educated, with less than half (44.74%) attending college. This latter group also had the highest percentage of minorities (7%, whereas the other clusters had less than 3%).

The clusters also differed in their health behaviors. The respondents in Cluster 1 (low, slow increase) had the lowest BMI (25.37) and were least likely to be current smokers (26.73%). In contrast, the two groups that had the most rapid increase in symptoms with age (Clusters 0 and 5) had the highest BMIs at baseline (26.48 and 26.49, respectively), and nearly half were current smokers (40.5% and 46%).

Personality at baseline also significantly differentiated the trajectories (Table 3)<sup>3</sup>. The two clusters (1 and 2) with low levels and relatively slow increases in symptoms with age also reported the highest level of emotional stability and the lowest levels of apprehension, tension, and suspiciousness. (*Mens sana, corpora sana.*) Although Cluster 3 also started out with low symptoms, they increased more rapidly, and indeed, their personalities differed from the men in the other two low clusters. They were significantly higher on apprehension than the other two and significantly lower than Cluster 1 on emotional stability.

In contrast, the groups with moderate and high symptom levels were very low on emotional stability and were high on suspiciousness, apprehension, and tension. The outliers were the lowest on emotional stability and the highest on suspiciousness of any of the groups. Personality did not appear to differentiate between the two moderate (Clusters 4 and 6) and the two high groups (Clusters 0 and 5), who all tended to have similar personality structures, but it did differentiate them from the two "optimal aging" groups.

The clusters also differed in both their mortality status and their causes of death (Table 4). Given the strong impact of age on mortality, it is not surprising that the youngest group (Cluster 6) had the lowest rates of mortality (6.61%), and the oldest group (Cluster 4) had the highest rates (33.98%). However, age was not the only factor. Cluster 2 (low, moderate increase) also had very low mortality rate (15.95%). Cluster 0, despite their high levels of symptoms, had an average mortality rate (26.32%).

Causes of death also revealed some interesting patterns (the 26 men whose cause of death was not known were omitted from this analysis). Cluster 4 was the group with the U-shaped curve, and

<sup>3</sup> Tables showing the complete results from all of the 16PF scales are available from Carolyn M. Aldwin.



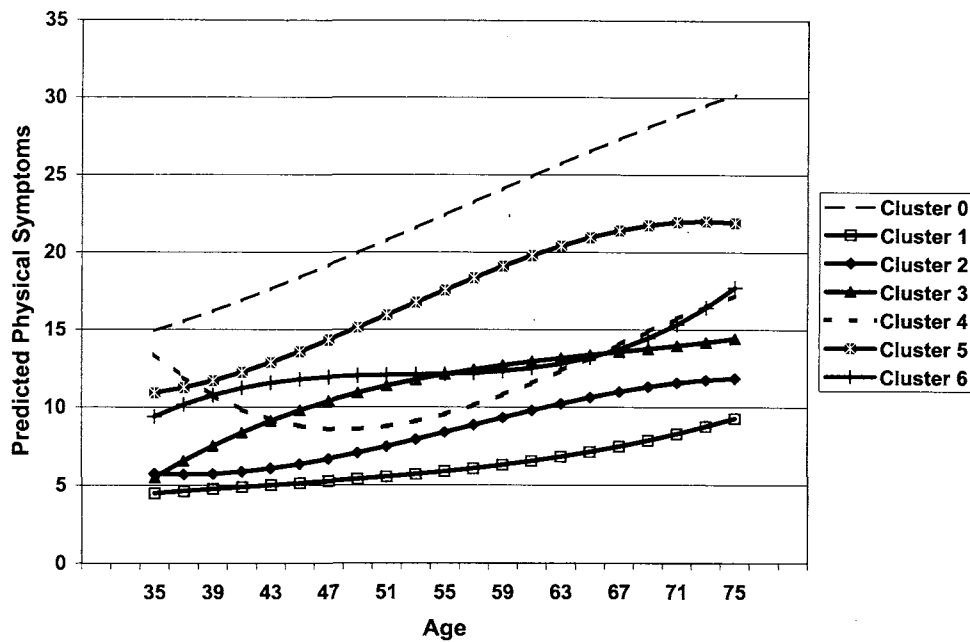


Figure 4. Physical symptom trajectory clusters.

the men in this cluster had the highest rates of coronary heart disease (15.31%) and “other” (e.g., pulmonary disease, accidents, and suicides; 8.16%). Perhaps these men recognized their health problems in early life and moderated their health behaviors in midlife, thus decreasing their number of symptoms, but then the underlying disease processes accelerated in later life. Men in Cluster 5 (high, rapid increase) were most likely to die of cancer (13.67%).

*Psychological Symptom Trajectories*

For psychological symptoms, we fit 8,843 observations on 1,515 men. The cubic model was selected as providing the best fit to the

data. The residual variance decreased from the quadratic to the cubic model (from 2.921 to 2.721). The AIC and LL were best for the cubic model (AIC = -19,766; -2LL = 39,510). Finally, all of the random-effects variance estimates were statistically significant for the cubic model.

The fixed effects (in the upper right-hand portion of Table 1) show that for the sample as a whole, the predicted number of psychological symptoms did not change with age: Neither the linear, quadratic, nor cubic effects were significant. However, the random effects (shown in the middle portion of Table 1) demonstrate that there was significant variation among men in the effects of age. The linear, quadratic, and cubic effects were all highly

Table 2  
Significant Differences Among Physical Symptom Trajectories in Demographics and Health Behavior Habits

Variable	Cluster							F or $\chi^2$ (6, 1508)	Duncan's post hoc range test ( $p < .05$ )
	0 (n = 76)	1 (n = 439)	2 (n = 178)	3 (n = 129)	4 (n = 339)	5 (n = 253)	6 (n = 101)		
Continuous variables									
Age									
M	46.69	48.44	44.80	49.87	52.24	45.84	42.42	28.27***	6 < 0-5; 4 > 0, 2-6;
Range	29-80	31-74	29-71	30-76	31-65	31-65	28-58		3 > 1 2 5; 1 > 2 5
BMI	26.48	25.37	25.72	26.13	25.85	26.49	25.82	4.59***	1 < 0 3 5; 0 5 > 1-2
Categorical variables									
Higher education	44.74	61.66 <sup>a</sup>	54.18	48.40	60.19	50.00	51.67	18.48**	
White	93.24	97.77	98.43	99.53	98.99	99.28	98.29	14.53*	
Current smoker	40.5 <sup>a</sup>	26.73 <sup>a</sup>	36.13	34.91	29.29	46.04 <sup>a</sup>	46.15	30.04***	

Note. The ns may vary slightly because of missing data. Clusters: 0 = outliers; 1 = low, slow increase; 2 = low, moderate increase; 3 = low, rapid increase; 4 = high, U-shaped; 5 = moderate, rapid increase; 6 = moderate, slow increase; BMI = body mass index.

<sup>a</sup> Cell contribution to  $\chi^2 > 3.84$ .

\*  $p < .05$ . \*\*  $p < .01$ . \*\*\*  $p < .001$ .

Table 3  
Significant Differences in Personality Among Physical Symptom Trajectories

Subscale	Cluster						F(6, 777)	Duncan's post hoc range test	
	0 (n = 42)	1 (n = 241)	2 (n = 103)	3 (n = 61)	4 (n = 165)	5 (n = 130)			6 (n = 51)
Emotional Stability (Scale C)	30.50	35.06	34.98	33.09	32.26	32.26	31.31	6.70***	1 > 0 3 6; 2 > 0 4-6; 0 < 1-3
Suspiciousness (Scale L)	17.45	14.55	15.20	15.79	16.43	17.21	17.21	5.10***	1 2 < 0 5 6
Apprehension (Scale O)	22.53	18.31	18.73	21.17	20.15	22.03	22.57	6.75***	1 2 < 0 3 5 6
Tension (Scale Q4)	23.80	19.70	20.01	21.82	23.25	23.92	24.25	6.17***	1 2 < 0, 4-6

Note. Clusters: 0 = outliers; 1 = low, slow increase; 2 = low, moderate increase; 3 = low, rapid increase; 4 = high, U-shaped; 5 = moderate, rapid increase; 6 = moderate, slow increase.  
\*\*\*  $p < .001$ .

significant, indicating that there was considerable individual variance in change in mental health with age. However, the variance for intercepts was not significant, suggesting a lack of individual differences in the average level of psychological symptoms at age 55.

Figure 3 presents the overall predicted curve for psychological symptoms on the basis of the fixed-effects portion of the model. On average, the NAS men showed very little change in mental health with age.

Because the random-effects estimates were significant, we looked for different patterns of trajectories by using cluster analysis. Contrary to expectations, the pseudo  $F$  and the pseudo  $r^2$  suggested that there were four rather than six clusters, plus the "cluster" of outliers. The iterative  $k$ -means clustering reassigned membership for 10% of the men from the Ward's clusters, most from initial Cluster 1 to Clusters 2 ( $n = 60$ ) or 3 ( $n = 48$ ).

Figure 5 presents the mean psychological symptom trajectories for the final clusters. Cluster 1 (low, stable) was represented by the solid line. This group had a low, constant number (<1) of psychological symptoms with age. In contrast, the outliers (Cluster 0; broken line) showed very high symptoms (about 10), which de-

creased a bit in midlife but then increased in later life. The three most interesting clusters (2-4) all started out with moderate symptom levels (approximately 3 to 5 symptoms), but showed markedly different trajectories, and are represented by lines with symbols. Cluster 2 exhibited a shallow U-shaped curve, with relatively high symptoms in the 30s, a decrease in midlife, and then increasing again in late life, somewhat similar to the J curve reported elsewhere (Aldwin et al., 1989; Kessler, Foster, Webster, & House, 1992). Cluster 3 exhibited a curve that showed a steady increase until about age 65, then started to level off, and may reflect problems with retirement (Bossé et al., 1992). Cluster 4 showed a peak in the 40s, perhaps reflecting a group of men who had a midlife crisis.

As with the physical symptom clusters, we examined differences in health behaviors and sociodemographic variables among these clusters. There were no significant differences in age, ethnicity, marital status, or BMI among the mental health trajectory clusters (results not shown). However, there were differences in education and smoking status (see Table 5). The outlier cluster was the least educated, with only 35% completing at least some college. On the whole, there were also modest differences in smoking status among the clusters. Clusters 2 and 4 had the highest rates of

Table 4  
Differences in Mortality Status Among Physical Health Trajectories (by Percentage)

Health outcomes	Cluster						$\chi^2(18)$	
	0	1	2	3	4	5		6
Mortality status								
<i>n</i>	76	461	395	219	103	140	121	
Deceased	26.32	21.26	15.95 <sup>a</sup>	25.57	33.98 <sup>a</sup>	26.43	6.61 <sup>a</sup>	38.29***
Causes of death								
<i>n</i>	76	451	390	215	98	139	120	
CVD	6.58	6.43	6.15	8.84	15.31 <sup>a</sup>	6.47	4.17	50.64***
Cancer	11.84	7.76	5.64	10.70	7.14	13.67 <sup>a</sup>	1.67 <sup>a</sup>	
Other	7.89	5.32	3.08	4.65	8.16 <sup>a</sup>	5.76	0.0 <sup>a</sup>	
Survivor	73.68	80.49	85.13	75.81	69.39	74.10	94.17	

Note. Clusters: 0 = outliers; 1 = low, slow increase; 2 = low, moderate increase; 3 = low, rapid increase; 4 = high, U-shaped; 5 = moderate, rapid increase; 6 = moderate, slow increase; CVD = cardiovascular disease.  
<sup>a</sup> Cell contribution to  $\chi^2 > 3.84$ .  
\*\*\*  $p < .001$ .

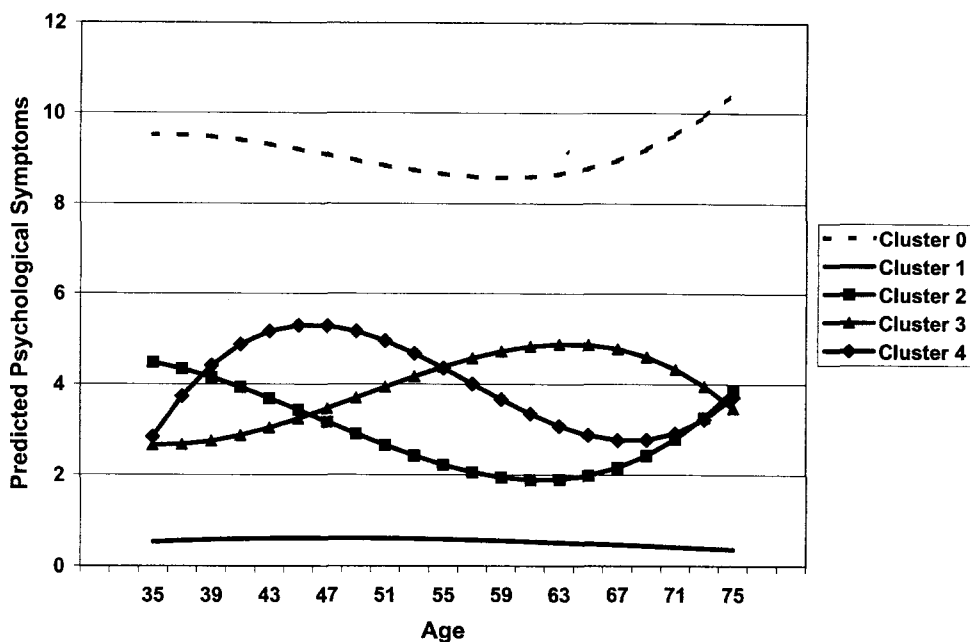


Figure 5. Psychological symptom trajectory clusters.

current smoking (44%), although it was not significantly higher than in the other clusters.

Perhaps the most important aspect of Table 5, however, is that it indicates the sample size of the groups. Note that Cluster 1, the low, stable group, contained the overwhelming majority of the NAS men ( $n = 1,113$ ). The other groups were relatively rare, ranging in size from 77 to 133. This may reflect in part the psychometric characteristics of the CMI mental health measure, as well as the selection criteria for entry into the NAS (see discussion below).

Table 6 presents data on personality differences for the half of the sample who completed the 16PF. Note that there were far more differences in personality among the psychological symptom clusters than there were among the physical symptom clusters. Cluster 1 (low, stable) was significantly higher on emotional stability and control than were the other groups, and they were lowest on sensitivity, suspi-

cioussness, and apprehension (all are measures of Cattell's anxiety factor and indicate that this cluster was characterized by low anxiety). The outliers had the highest scores on suspiciousness and apprehension and the lowest scores on emotional stability.

In general, the three groups that had reported moderate symptom levels were intermediate in scores between the low stables and the outliers, but there were some interesting differences between the moderate groups. Cluster 4 (midlife crisis) was significantly higher on apprehension; tension, and unconventionality than the other moderate groups, whereas Cluster 2 (U-shaped) was higher on the controlled subscale.

We also examined differences in mortality status and cause of death between the clusters. However, there were no significant differences, partially because of the extreme unbalance in the cell sizes (data not shown).

Table 5  
Significant Differences Among Psychological Trajectories in Demographic and Biomedical Variables

Variable	Cluster					$\chi^2(4)$
	0 ( $n = 77$ )	1 ( $n = 1113$ )	2 ( $n = 133$ )	3 ( $n = 114$ )	4 ( $n = 77$ )	
Higher education	35.06 <sup>a</sup>	56.89	56.39	53.51	46.75	16.28**
Current smoker	31.51	32.69	43.75	37.50	44.74	10.67*

Note. The  $n$ s may vary slightly because of missing data. Clusters: 0 = outliers; 1 = low stable; 2 = U-shaped; 3 = retirement peak; 4 = midlife peak.

<sup>a</sup> Cell contribution to  $\chi^2 > 3.84$ .

\*  $p < .05$ . \*\*  $p \leq .01$ .

**Table 6**  
*Significant Differences in Personality Among Psychological Symptom Trajectories*

Scale	Cluster					F(4, 788)	Duncan's post hoc range test
	0 (n = 40)	1 (n = 592)	2 (n = 69)	3 (n = 52)	4 (n = 40)		
Emotionally Stable (Scale C)	27.75	34.98	31.46	30.10	30.45	22.41***	1 > 0 2-4; 0 < 1 2 4
Sensitivity (Scale I)	19.25	17.30	17.94	17.96	19.65	3.17**	0 4 > 1
Suspiciousness (Scale L)	19.95	14.77	17.70	17.81	17.63	19.02***	0 > 2-4 > 1
Apprehension (Scale O)	27.80	18.33	22.20	23.23	25.80	34.48***	0 4 > 2 3 > 1
Unconventionality (Scale Q1)	19.45	20.84	20.12	20.85	18.20	4.28**	4 > 1 3
Controlled (Scale Q3)	22.50	25.23	23.78	22.42	21.88	11.13***	1 > 0 3 4; 2 > 4
Tension (Scale Q4)	28.13	19.59	25.59	25.48	28.58	30.85***	4 > 1-3; 0 > 1 2

Note. Clusters: 0 = outliers; 1 = low stable; 2 = U-shaped; 3 = retirement peak; 4 = midlife peak.  
\*\* *p* < .01. \*\*\* *p* < .001.

*Overlap Among the Physical and Psychological Symptom Clusters*

We explored the overlap between membership in the physical and psychological symptom clusters to examine the extent to which men with good physical health had good mental health as well. For each of the seven physical symptom clusters, Table 7 shows the distribution of men across the five psychological symptom clusters (i.e., each row percentage sums to 100). The largest overlap was between two of the low physical symptom clusters (1 and 2) and the low stable psychological cluster (1). Over 85% of the men in the two groups with a pattern of low physical health symptoms also had a stable pattern of few psychological health symptoms. In contrast, less than one fourth of the men in the physical symptom outlier cluster were also in the psychological symptom outlier cluster, suggesting that volatility does not necessarily covary across the two types of symptoms.

Discussion

At the aggregate level, the health trajectories seemed deceptively simple. Physical symptoms appeared to increase linearly

**Table 7**  
*Overlap Among the Physical and Psychological Symptom Clusters (by Percentage of Physical Symptom Cluster)*

Physical cluster	Psychological cluster				
	0 (n = 77)	1 (n = 1113)	2 (n = 133)	3 (n = 115)	4 (n = 77)
0 (n = 76)	22.4	32.9	13.2	21.1	10.5
1 (n = 461)	1.7	86.6	5.9	2.4	3.7
2 (n = 395)	2.5	84.8	5.8	5.1	1.8
3 (n = 219)	4.1	71.7	7.3	8.7	8.2
4 (n = 103)	4.9	62.1	16.5	13.6	2.9
5 (n = 140)	11.4	50.0	9.3	21.4	7.9
6 (n = 121)	9.9	52.1	32.3	4.1	11.6

Note. Physical clusters: 0 = outliers; 1 = low, slow increase; 2 = low, moderate increase; 3 = low, rapid increase; 4 = high, U-shaped; 5 = moderate, rapid increase; 6 = moderate, slow increase. Psychological clusters: 0 = outliers; 1 = low stable; 2 = U-shaped; 3 = retirement peak; 4 = midlife peak.

with age, and there was little change in mental health. At the individual level, however, there was considerable variability in both physical and psychological symptom trajectories, and these trajectories were often nonlinear, as indicated by the random-effects model. Using cluster analysis, we found that there were different and coherent patterns of change in symptoms across the life span. After removing 5% of the sample as potential outliers, we identified six clusters of physical symptom trajectories and four clusters that characterized psychological symptom trajectories.

*Physical Symptoms*

For physical symptoms, the modal pattern (Cluster 1) among the NAS men was characterized by a low number of symptoms that started increasing only in later life and resembled Clipp et al.'s (1992) constant good health group.<sup>4</sup> In many ways, this group could be seen as an optimal aging group. Several of the other clusters demonstrated markedly nonlinear patterns that roughly corresponded to some of Clipp et al.'s other groups (see Figure 1). Although we did not have a group that had constant poor health (probably because of the selection criteria used at NAS entry), two clusters (0 and 5) started out with relatively high levels of symptoms and increased linearly. Cluster 4 showed an accelerated increase in symptoms in late life and had the highest mortality. We did not have a "decline and recovery" group, but we did have a group that recovered and then declined (Cluster 4).

Further, different patterns of sociodemographics, health behaviors, and personality variables characterized the physical symptom trajectory clusters. Surprisingly, the clusters were more likely to differ on personality traits than on the biomedical or sociodemographic variables we considered, indicating the importance of personality for health across the life span. Cluster 1, although not the youngest group, had the lowest level of symptoms, the best health behaviors, the highest level of emotional stability, and the lowest level of hostility. However, they did not have the lowest level of mortality, perhaps reflecting their relatively wide age range at baseline (29–80). We found earlier that emotional stabil-

<sup>4</sup> Note that Clipp et al. (1992) were modeling health and declines in health were shown as decreases, whereas we are modeling symptoms and a worsening of health is shown as an increase.

ity was also a protective factor against the development of hypertension (Spiro et al., 1995), and the role of emotional stability in optimal aging needs to be considered further.

Personality had effects on health across the life span. Some of the effects were on general symptom levels; that is, personality characteristics appeared to best discriminate the clusters that started out with low symptom levels from those that started out with high levels, with the moderate groups in the middle. However, personality was also related to the shape of the trajectories. For example, among the three clusters that started out with low symptoms, Cluster 3, which had the most rapid increase, also reported higher anxiety levels. Thus, aspects of personality appeared to have both protective and vulnerability effects throughout the life span for physical health.

Curiously, though, the cluster with the highest symptom levels (Cluster 0) did not have the highest mortality rates; perhaps they had a variety of nonlethal symptoms (see Verbrugge, 1989). The highest mortality rate was found among those who reported a high initial level of symptoms, which then decreased in midlife and rose rapidly in later life (Cluster 4). It should also be noted that Cluster 4 was the oldest group, although there was considerable overlap in age among clusters. Not surprisingly, the youngest group (Cluster 6) had the lowest levels of mortality, despite moderate levels of symptoms that increased slowly with age.

Similar to Clipp et al. (1992), different trajectories also had different causes of death. The men in Cluster 4, the U-shaped trajectory, were most likely to die of cardiovascular disease. In contrast, the mean trajectory of Cluster 5 was characterized by rapid, nearly linear increases, and the men in this cluster were most likely to die of cancer.

### *Psychological Symptoms*

Compared to Clipp et al. (1992), we found that fewer (rather than more) trajectories characterized psychological symptoms than physical symptoms. In part, this may be because most of the NAS men reported few, if any, psychological symptoms. Further, the CMI assesses psychological symptoms in a more trait- than state-like manner, because it uses dichotomous items that ask about usual or typical behaviors, and thus may be less likely to be endorsed and less sensitive to change. Thus, a restriction of range in mental health observed in the NAS may have limited our ability to detect other patterns.

Nonetheless, some interesting patterns of change in mental health emerged. The overwhelming majority of men were in the low, stable cluster, which may reflect the psychometric characteristics of the CMI. However, the outliers reported consistently high symptoms, which did increase slightly with age. Again, the low, stable cluster was characterized by emotional stability, control, and low levels of suspiciousness and anxiety, whereas the outliers had very high levels of hostility and anxiety.

Of greatest interest were the three clusters (4, 5, and 6) that had moderate levels of symptoms in the 30s. These clusters all had nonlinear trajectories and were clearly distinguished by the timing of their symptom peaks: one in midlife, one around retirement, and one in late life. Although personality characteristics did not distinguish among the moderate groups, they were moderate in hostility and anxiety, compared with the low and high groups. Moderate levels of hostility and anxiety may predispose individuals to

mental health problems at some point over the life span, but other, perhaps environmental, factors may affect the timing of the problems.

There was considerable overlap in membership between the two modal physical and psychological trajectory clusters (>80%), both characterized by low, stable symptom trajectories. Another very interesting finding was associated with the two residual clusters (designated as Cluster 0 for both the physical and psychological symptom trajectories). The men in these clusters were outliers; that is, they did not appear to fit well with the others. For both physical and psychological symptoms, men in the residual clusters were more likely to have smoked and to be high on two markers of anxiety, Suspiciousness (Scale L) and Apprehension (Scale O). However, the overlap between the two clusters was about 25%, suggesting that only some of the men in this sample were outliers on both physical and psychological symptom trajectories.

### *Limitations and Generalizability*

Despite the fact that the personality, sociodemographic, and biomedical variables were assessed before the health symptoms, and because the sample was screened for good health at the beginning of the study, causal directionality remains unclear. As with many longitudinal studies, the starting point was arbitrary. Prior health status, physical or mental, may have contributed to personality characteristics at baseline. For example, good health earlier in life may well have contributed to greater emotional stability and lower anxiety.

Nonetheless, some of the patterns confirm long-established findings. Individuals in the cluster with the lowest socioeconomic status tended to have the highest mortality, whereas the healthiest cluster tended to have the best health behaviors. Further, in terms of personality, the clusters with the lowest mortality rates tended to be more emotionally stable and less hostile, suggesting a protective role for personality with respect to at least some health problems (see Spiro et al., 1995).

This study has several limitations. Because the NAS is relatively homogenous with respect to ethnicity and marital status, we may have underestimated the contribution of these variables to health and mortality. For example, perhaps marital status did not contribute to differences in physical symptom trajectories because most of the NAS men were married, although Clipp et al. (1992) also found this. The present sample included no women, and thus the results cannot be generalized to that half of the population. We did, however, replicate some of the trajectory patterns found in a prior study that did include women (Clipp et al., 1992), and it is noteworthy that the gender differences that Maddox and Clark (1992) reported disappeared when education and income were taken into account.

We included only individuals with four or more visits, which also biased the sample in some unexpected ways. As indicated in Footnote 1, there were no differences between the men with three versus four or more visits at baseline in age, education, marital status, ethnicity, physical and psychological symptoms, and BMI. Soberingly, the only differences were that the men with fewer visits were more likely to be smokers and were more likely to have died, primarily of heart disease and cancer. Omitting these men, however, did not alter the fixed effects, but the cubic random effect would have been underestimated.

There was a marked imbalance in the distribution of individuals across clusters. Although we could have used clustering techniques that would have grouped individuals into clusters of relatively equal size, we decided that this would force the distribution to be arbitrary, and we preferred to determine the "natural history" of change. Thus, the majority of the sample fell into low, stable clusters for both physical and psychological symptoms. This may have reflected the original NAS sampling design. Because the purpose of the NAS was to differentiate between normal and disease-related aging processes, we began with healthy men, regardless of their age. Thus, the size of these low, stable clusters may be an artifact of the initial NAS selection criteria.

This imbalance across clusters may have affected the findings in other ways. It is not surprising that most of the significant differences among the clusters in personality characteristics were found between the larger clusters and the rest of the clusters. In part, this may have also been due to measurement characteristics. As mentioned earlier, a limitation of the CMI is that the psychological symptoms in particular showed marked floor effects, and measures having better distributions might have yielded more differentiated clusters.

### Significance and Future Studies

Despite these limitations, there were a number of noteworthy findings. The first is the extent of variability in the random effects or individual curves, compared to the relative stability or simplicity of the fixed effects or aggregate curves. In aggregate, physical symptoms increased linearly with age, and mental health did not appear to change at all. However, the individual curves showed marked, nonlinear patterns: Some people had exponential increases in symptoms with age, others increased early in adulthood and then plateaued, and some enjoyed remarkably good health until very late in life. Although most NAS men reported few psychological symptoms, clearly some had difficulty negotiating specific periods of life, including midlife, retirement, and old age. This underscores the importance of an idiographic approach to understanding age-related changes in health. We believe that the statistical approach we used identified meaningful and replicable patterns, which nonetheless require replication.

Second, this is one of the first studies to trace the impact of personality on health across the life span, as opposed to using personality simply to predict health outcomes. Although emotional stability and hostility had clear life-long effects, the effects of anxiety were more variable. There are some indications that the anxiety scales created a vulnerability that may manifest only in relationship to environmental and perhaps genetic factors. Although personality is generally seen as a risk factor, this study underscores the importance of emotional stability as a protective factor, not only for hypertension (Spiro et al., 1995) but also for health in general throughout the life course. Identifying the mechanisms through which emotional stability protects health is a crucial next step.

Finally, the findings do reflect the promise of the NAS: to characterize individuals who age optimally. Founded at a time when the distinction between disease and aging processes was first recognized, the explicit goal was to identify why individuals maintained good health despite old age and to understand the factors that lead to this outcome (Rose & Bell, 1971). Although

good health behavior habits are clearly important, the approach taken here suggests that optimal aging may be the result of a complex combination of personality traits, health behavior habits, socioeconomic status, and undoubtedly other environmental and genetic factors as well. Identifying how these myriad components interact over the life course to produce optimal aging can enhance future intervention strategies.

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Received November 17, 1998

Revision received October 18, 2000

Accepted January 17, 2001 ■

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