

# Personality Stability in Late Adulthood: A Behavioral Genetic Analysis

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**ABSTRACT** A sample of 833 twins from the Minnesota Twin Study of Adult Development and Aging completed the Multidimensional Personality Questionnaire (MPQ; Tellegen, 1982) twice, averaging 59.4 ( $sd = 9.7$ ) years of age at first and 64.4 ( $sd = 10.2$ ) years of age at second testing (average retest interval 5.0 years,  $sd = 2.36$ , range 1.0–10.4 years). Both means and standard deviations of scale scores were extremely stable from first to second testing. In addition, sample participants tended to retain their rank order on the scales (average  $r = .76$  across scales). Bivariate biometric analyses showed that the genetic influences on most of the scale scores were almost perfectly correlated across the two waves (range .95 to 1.00). The nonshared environmental influences were also highly correlated across the two waves (range .53 to .73). Models specifying identical variance components at the two time points and fixing the genetic correlation to 1.00 provided improved fit. The results suggest that the high stability of personality in later adulthood has a strong genetic foundation, supplemented by stability of environmental effects.

A substantial body of evidence has accumulated for the stability of personality in adulthood. Such evidence is especially strong for the consistency of an individual's relative position in a group (Roberts & DelVecchio, 2000). Consistency of this type, known as differential

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stability (Caspi & Roberts, 2001), is generally measured by a correlation coefficient. Such coefficients have been estimated for a variety of self-report personality questionnaires, ages, and retest intervals (e.g. Lubinski, Schmidt, & Benbow, 1996; McCrae & Costa, 1990; McGue, Bacon, & Lykken, 1993), but also for personality ratings by clinicians, spouses, and acquaintances (e.g. Conley, 1985; Costa & McCrae, 1988). In addition, there is strong evidence for the constancy of absolute levels of personality measures over time (Caspi & Roberts, 2001; Costa & McCrae, 1988). Roberts and DelVecchio (2000) concluded that the evidence to date suggests that relative stability of personality over a period of several years increases with age, with correlations going from .31 in childhood to .64 around age 30, and leveling off at .74 for the ages 50 to 70.

Stability of personality also decreases as the time interval between observations increases, yet impressive levels of stability have been observed over lengthy time spans in adulthood. Though 30-day retest reliabilities for well-constructed scales can range from .80 to .90 (reflecting the measurement capacity of the instrument; Tellegen, 1982), retest stabilities across spans on the order of 25 years can remain in the range of .60 to .70 (Costa & McCrae, 1992b; Helson & Klohnen, 1998), with higher stabilities (on the order of .65 to .80) reported for shorter time intervals of 6 to 12 years (Costa & McCrae, 1988, 1992b; Viken, Rose, & Koskenvuo, 1994). For adults under age 30, comparable stabilities for 6- to 10-year time intervals are only slightly lower, ranging from .48 to .60 (McGue et al., 1993; Viken et al., 1994). The particular aspect of personality measured appears to have little effect on the extent of stability, nor do sex or method of measurement (Roberts & DelVecchio, 2000).

That genes contribute to personality throughout the lifespan is also very well established. Loehlin and Nichols's (1976) study of almost 800 pairs of adolescent twins and dozens of personality traits has stood the test of time, having been replicated many times using a variety of personality measurements and sample populations across a broad range of ages. Bouchard and Loehlin (2001) summarized the findings of 25 years of investigations as showing that 40–60% of individual differences in personality can be attributed to genetic variation. McCrae et al. (2000) have used this consistency of results across samples and many age ranges, construed in light of temperament as a constitutional predisposition to personality qualities, to argue that personality traits, even at the facet level, are genetically controlled

dispositions for which the environment plays no fundamental role and merely sets the stage for expression. They maintain, therefore, that both continuity and change in personality have their sources in genetic influences, with genetic influences on basic temperament contributing stability and genetic influences on developmental processes contributing to change.

The concept of genetic influences on developmental processes is familiar when considering children and adolescents, for whom it is not difficult to conceive of and even demonstrate psychological processes that parallel the clear physical manifestations of development. In considering adults, however, the concept is more foreign. Though adults, too, experience physical changes over time, we tend to think of the physical manifestations of aging as resulting from gradual and passive decline in function rather than from the kinds of well-regulated and dynamic processes that govern change in youth. This may greatly oversimplify the actual situation, as there is growing evidence, from fruit flies as well as mammals, that many of the organism's biological responses (manifested by genetic expression) to aging, at least, are very analogous to the sorts of developmental processes that operate in youth (Helfand & Rogina, 2003). That responses to physical aging may be under systematic genetic developmental control in such nonhuman organisms hints at a similar role for psychological mechanisms such as personality characteristics in humans. For example, there is evidence for systematic personality change with age (Srivastava, John, Gosling, & Potter, 2003), with the general trait of Big Five Extraversion showing some decrease and Conscientiousness increasing. These patterns at the level of general traits may result from much more specific changes, such as the observation of increased Harm Avoidance with age (Carmichael, 1993). It is possible that this increase could be a genetically controlled adaptive psychological response to increased physical fragility. Thus, McCrae et al.'s (2000) conception of genetic sources of change in adulthood is plausible, though it requires testing.

Roberts and Caspi (2003) have addressed the question of genetic and environmental influences on continuity and change in personality somewhat differently. Noting that personality stability is more common than change in personality traits, they point out that, almost by definition, trait theories make no explanation for personality change. On the other hand, developmental theories focus primarily on change, and they tend to present change as change in responses to

circumstances rather than as changes in characteristics intrinsic to the individual such as personality. To address the gap, they describe the ways in which genetic and environmental influences, psychological function, and habitual person-environment transactions can contribute to stability. Very generally stated, these same processes can also contribute to change in the form of genetic influences on developmental processes, responses to environmental circumstances that reflect genetic as well as environmental influences, observational learning, learning generalizations, and learning from others' descriptions of ourselves. They thus see both personality stability and change as the results of an ongoing series of transactions between genetic and environmental influences, which both have elements of stability and change. This conception also should be tested.

In behavior genetic studies, environmental influences are generally categorized as either shared or nonshared. Shared environmental influences are those that contribute to similarity among reared-together relatives. Frequently given examples of such influences refer to experiences of children, such as growing up with a common set of parents who provide a certain level of socioeconomic status, values such as religion, and common experiences such as travel, presence of alcoholism, or provision of a college education. But the concept of shared environmental influences can be extended to adults as well. It is easy to conceive of these kinds of experiences as having lifelong influences, and there is evidence for the existence of such effects for some of them. The process of measuring the magnitude of shared environmental influences does nothing to identify the specific influences operating, so conceptual plausibility of this sort is all that is necessary to justify their quantification in this manner. Environmental influences can be shared by siblings in adulthood as well. For example, siblings may continue to associate with each other frequently in adulthood, sharing holidays and care for elderly parents, among many other things. Thus, in adults, shared environmental influences include both lifelong effects of shared childhood experiences and the effects of currently shared experiences. The concept of shared environmental influence has had little effect on the conception of personality emergence, stability, and change, however, because most studies have shown little evidence of it for personality (Bouchard & Loehlin, 2001).

Nonshared environmental influences are those that contribute to differences between siblings, and the same sort of discussion applies.

In childhood, experiences such as having different schoolteachers, participating in different sports or other leisure activities, and differential parental treatment would qualify, and these experiences could have effects that persist into adulthood. In addition, in adulthood, siblings typically have many different experiences involving spouses, jobs, and financial security (again among many other things). Nonshared environmental influences are rarely measured directly. Rather, they are measured as residual variance, which means that the term in which they are included always includes random error of measurement and often includes systematic method bias as well. Thus we would tend to expect relatively little relationship between nonshared environmental influences over time, due, in part, to measurement error and, in part, to changes in nonshared environmental circumstances over time. This means that one way to evaluate some of the implications of Roberts and Caspi's (2003) and McCrae et al.'s (2000) conceptions of stability and change in personality is to use a longitudinal twin study.

When it is carried out in longitudinal form, the twin study method that serves as the cornerstone of behavioral genetic analysis can offer important contributions to our understanding of the nature of both the observed stability of and change in personality over time. In particular, such studies can address the extent to which genetic and environmental influences change with time by estimating the amounts of variance attributable to genetic and environmental influences at varying time points. They can also address the extent to which genetic and environmental influences contribute to stability and change by estimating the extent to which the same genetic and environmental influences remain in place over time and new ones come into play. Loehlin (1992) reviewed longitudinal behavior genetic studies of variation in personality from childhood into adulthood in order to summarize the evidence for various kinds of and changes in genetic influence. Though most of the studies he reviewed did not address genetic and environmental sources of personality stability and change directly, he concluded that although there are significant sources of genetic influence on personality change in childhood, after that, genetic influences contribute primarily to personality stability rather than to change. This conclusion may have been driven by the relative lack of change in adulthood to evaluate.

Genetic and environmental sources of stability of or change in personality in adulthood have been addressed specifically in just a

few longitudinal studies. These studies have primarily focused on the traits of Extraversion and Neuroticism as defined by Eysenck (Eysenck & Eysenck, 1985). Viken et al. (1994) found that the same genes contributed to personality variance over time, based on a sample of nearly 15,000 male and female Finnish twins ranging in age from 18 to 53 at baseline and tested on average 6 years later. They also observed that heritabilities decreased with age, primarily resulting from increasing variance associated with nonshared environmental influences at the two time points, and that the correlations between nonshared environmental influences at the two time points increased with age from about .30 at age 18 to about .55 at age 53. Pedersen and Reynolds (1998) also conducted a longitudinal study, involving 2,209 participants of the Swedish Adoption/Twin Study of Aging who averaged 60 years of age at baseline and reported over a 9-year period. Like Viken, they concluded that the genetic influences on Extraversion, Neuroticism, and the Five-Factor Model trait of Openness were stable. Despite the stability of both mean levels of the personality traits and genetically influenced variance, they noted both generally increasing nonshared environmental variance and increasing change with time. Neither study showed evidence of new genetic contributions after age 30. Very similar results were obtained for Positive and Negative Emotionality and for Constraint from the Multidimensional Personality Questionnaire (MPQ; Tellegen, 1982) in a Minnesota sample of 127 younger twin pairs averaging 20 years of age at baseline and 30 at retest (McGue et al., 1993). In general, the data suggested higher levels of genetic influence in early adulthood, declining to about 40% over the age 20–30 period, with little evidence of new genetic contributions during the period observed. Similar results were also obtained by Angleitner and Spinath (2002) using Five-Factor Model NEO-PI-R (Costa & McCrae, 1992a) self- and peer reports in a German sample spanning a large adult age range.

The studies discussed above have focused either on the very broad personality constructs of Extraversion and Neuroticism or on early adulthood. They have also focused primarily on the extent to which genetic factors contribute to personality stability and/or change. They have established evidence for a strong genetic foundation influencing stability in these broad traits across the adult lifespan, but we are not aware of any study that has investigated the extent to which these findings apply to more specific personality traits in late

adulthood, nor are we aware of any study that has investigated specifically the question of environmental as well as genetic influences on stability over time. It is important to examine the stability of more specific personality traits as well as the broad traits such as Extraversion and Neuroticism because these broad traits are manifested in various specific ways, and stability at the level of the broad traits could mask change at the specific level. For example, both sociability and adventurousness are often considered to be aspects of Extraversion. A person, however, could change from being adventurous but not particularly sociable to being sociable but not particularly adventurous without changing in overall level of Extraversion. It is important to estimate the extent of environmental as well as genetic influences on stability of personality because they can help us to distinguish between McCrae et al.'s (2000) conception of both stability of and change in personality as primarily genetic in origin and Roberts and Caspi's (2003) perspective of a larger role for the environment in personality stability.

Thus, the purpose of the current study was to investigate the extent and sources of stability of and change in personality as measured by the primary trait scales of the MPQ in late adulthood. We made use of the MPQ because it was designed to measure 11 specific personality traits with no item overlap among its primary scales (Tellegen, 1982). It therefore measures specific aspects of personality with high fidelity. Nevertheless, the primary scales of the MPQ cover a broad range of traits, showing good bandwidth. Thus, the MPQ combines fidelity and bandwidth in the measurement of personality, making it especially well suited to a comprehensive investigation of the genetic and environmental sources of personality stability.

The MPQ is a self-report personality inventory that was constructed by an exploratory factor-analytic process with the goal of developing relatively pure indexes of trait dispositions within normal personality. The result was 11 relatively independent primary trait scales. In factor analyses, the primary scales load on three higher-order factors, Positive Emotionality, Negative Emotionality, and Constraint, which relate to neurobiological mechanisms and map onto constructs of emotion and temperament. That is, Positive and Negative Emotionality incorporate temperamental dispositions toward those two emotions and are linked to brain motive systems underlying the appetitive-approach and defensive-withdrawal behaviors (Derryberry & Reed, 1994; Lang, 1995; Sutton & Davidson,

1997). Constraint incorporates traits related to behavioral restraint and reversed impulsivity, which has been characterized in terms of low brain arousal and reduced conditionability (Zuckerman, 1991). The primary MPQ scales have high internal consistency and 30-day retest reliabilities, and they cover a range of relatively psychometrically pure traits encompassing temperament, behavioral regulation, and interpersonal and imaginative styles. The MPQ's combination of high reliability, construct independence, broad coverage of the personality domain, and neurobiological referents make it a particularly good choice for investigation of the extent and sources of personality stability. Table 1 briefly characterizes the MPQ scales.

Positive Emotionality reflects the tendency to be actively and pleurably engaged with one's social and work environments; it is similar to the Extraversion dimension of the Eysenck Personality Questionnaire (EPQ; Eysenck, 1960) and the Five-Factor Model (FFM; McCrae & Costa, 1997) and to the Ascendance factor of the California Psychological Inventory (CPI; Gough & Bradley, 1996). The primary scales loading on positive emotionality reflect agency and communion; they are Wellbeing, Social Potency, Achievement, and Social Closeness. Negative Emotionality is characterized by perceptions of the world as threatening, problematic, and distressing; it is similar to Neuroticism as defined by many personality measures. Its primary scales reflect neurosis, estrangement, and confrontation; they are Stress Reactivity, Alienation, and Aggression. Constraint is marked by the tendency to be restrained (impulsiveness reversed), conventional, and careful (sensation-seeking reversed). It is related to the Conscientiousness factor in the FFM (Church, 1994). The primary MPQ scales loading on Constraint are Control, Harm Avoidance, and Traditionalism. The 11th primary MPQ trait scale, Absorption, reflects emotional responsiveness to sensory input and loads on both Positive and Negative Emotionality.

Specifically, in this study, we addressed three issues in a longitudinal, population-based Minnesota sample of primarily older-adult twins. First, we assessed the extent of personality stability and change, given measurements at two time points. Second, we estimated the genetic and environmental influences on the personality scale scores at both time points. Third, we investigated the extent to which the same genetic and environmental influences were involved

**Table 1**  
**Description, Retest and Internal Consistency Reliability of the 11 Multidimensional Personality Questionnaire Primary Scales**

Primary scale	Higher-order factor representation	Self-descriptions of high scorers	30-day retest correlation	Coefficient alpha
Well-being	Positive emotion	Happy, cheerful, active	.90	.89
Social potency	Positive emotion	Forceful, decisive, persuasive	.82	.88
Achievement	Positive emotion	Works hard, persists, likes long hours	.88	.84
Social closeness	Positive emotion	Sociable, likes people, warm and affectionate	.92	.85
Stress reactivity	Negative emotion	Nervous, easily upset, troubled by guilt	.89	.89
Alienation	Negative emotion	Victim of bad luck, feels mistreated	.87	.83
Aggression	Negative emotion	Physically aggressive, vindictive	.82	.79
Control	Constraint	Reflective, cautious, careful	.82	.85
Harm avoidance	Constraint	Does not enjoy excitement of adventure and danger	.88	.84
Traditionalism	Constraint	Endorses high moral standards, supports religious values	.90	.84
Absorption	Mixed	Emotionally responsive to engaging sights and sounds	.91	.88

*Note.* Retest correlations are based on a sample of 75 male and female college students. From *Brief Manual for the Differential Personality Questionnaire* by A. Tellegen, 1982, p. 6. Reprinted by permission.

at the two time points. We expected considerable stability of personality, as well as a substantial genetic contribution to stability across time, as anticipated in the conceptions of the sources of personality stability and change of both McCrae et al. (2000) and Roberts and Caspi (2003). To the extent that we also observed substantial environmental contributions to stability across time, our results would tend to provide greater support for Roberts and Caspi's conception.

## METHOD

### *Sample*

The study sample was drawn from participants in the Minnesota Twin Study of Adult Development and Aging, a population-based registry including 901 intact monozygotic and same-sex dizygotic twin pairs born in the state of Minnesota. Most were born between 1904 and 1934, but 29% were born between 1936 and 1955, and one pair was born in 1897. This study has been described previously (Finkel & McGue, 1994). Briefly, participants were identified from Minnesota State Health Department birth records and recruited using state marriage and motor vehicle records; the initial participation rate was approximately 60%, likely due to the 4–5-hour length of the in-home assessment, which included a series of cognitive, personality, and lifestyle measures. Zygosity was determined using a five-item questionnaire that has been shown to provide over 95% accuracy (Lykken, Bouchard, McGue, & Tellegen, 1990). Twins were assigned as Twin 1 and Twin 2 by order of birth.

A total of 1,123 participants (including 378 members of complete male twin pairs, 556 members of complete female twin pairs, and 189 twins whose co-twins did not provide data) completed the MPQ in the early 1990's. In addition, 833 of these individuals (118 members of complete male MZ twin pairs, 106 members of complete male DZ twin pairs, 266 members of complete female MZ twin pairs, 168 members of complete female DZ twin pairs, and 90 male twins and 85 female twins whose co-twins did not provide data) completed the MPQ again, on average 5.0 years later ( $SD$  2.4, range 1.0–10.4). Participants averaged 59.4 years of age ( $SD$  9.7, range 27.0–92.0) at first and 64.4 ( $SD$  10.2, range 32.2–98.7) at second administration. Though the full age range was rather large, the majority of participants were between ages 50 and 70 at first testing; less than 18% of participants were under age 50, and less than 10% were over age 70.

*Analysis of Mean-Level Personality Stability*

To assess mean-level stability of our personality measures, we fit a hierarchical linear model (Bryk & Raudenbush, 1992), with overall mean scale score and retest interval as fixed effects and twin pair and initial score as random effects, to each set of MPQ scale scores. The use of interval of testing was necessary because the testing interval was reasonably evenly distributed over the range from 1.5 to 9 years. We made use of the hierarchical linear modeling technique rather than analysis of variance so that the estimates of statistical precision resulting from our models would be correctly adjusted both for the variable retest interval and for the correlations between scale scores within twin pairs.

*Age and Sex Adjustment*

Twins share a common age and, in our sample, a common sex. Thus age and sex effects act to increase twin similarity (McGue & Bouchard, 1984). To correct for these effects in the biometric analysis of the twin data, we adjusted the personality scores at the two time points by subtracting the sex-appropriate mean from each individual's score and regressing out the linear component of age at each time point. In doing so, we did not norm the residuals to unit variance as we wished to be able to identify factors that contribute to variability in whatever personality change we observed.

*Biometric Modeling*

Our quantitative genetic model is based on the assumption that the observed phenotypic variance ( $V_p$ ) is a linear additive function of genetic (A) and shared (C) and nonshared (E) environmental variance, respectively. Symbolically,

$$V_p = A + C + E.$$

Under this model, the nonshared environmental variance represents residual variance not explained by either of the other two sources. The nonshared environmental component also includes variance attributable to measurement error. Genetic variance can be additive in the sense that if multiple genes influence the trait, they do so independently of each other. It can also be nonadditive, reflecting dominance and other polygenic effects. We note that there is evidence for nonadditive effects on personality, but little evidence for shared environmental effects (Bouchard & Loehlin, 2001; Finkel & McGue, 1997; Tellegen et al., 1988). For additive genetic effects, the expected covariance between any two members of a twin pair as a function of the variance components given above can be

specified as,

$$\text{COV}_{(MZ)} = A + C$$

$$\text{COV}_{(DZ)} = .5A + C.$$

Heritability estimates of this form are based on several assumptions. The first is that twins (both MZ and DZ) are representative of the population as a whole for the trait in question. For personality traits, in particular as measured by the MPQ, this appears to be the case (Johnson, Krueger, Bouchard, & McGue, 2002). Second, we assume that MZ twins share trait-relevant environmental influences to the same degree as DZ twins. Numerous attempts have been made to uncover circumstances in which this assumption does not hold, with generally negative results (e.g., Borkenau, Riemann, Angleitner, & Spinath, 2002; Loehlin & Nichols, 1976). Another assumption is that there is no assortative mating (meaning that the parents of the twins were not similar) for the traits in question. Because there is some marital resemblance (with coefficients ranging from .10 to .20) for most personality-related traits (Price & Vandenberg, 1980), including those measured by the MPQ (Lykken & Tellegen, 1993), this assumption generally has the effect of understating heritability estimates. Such relatively small values, however, are unlikely to reduce estimates of genetic influence substantially.

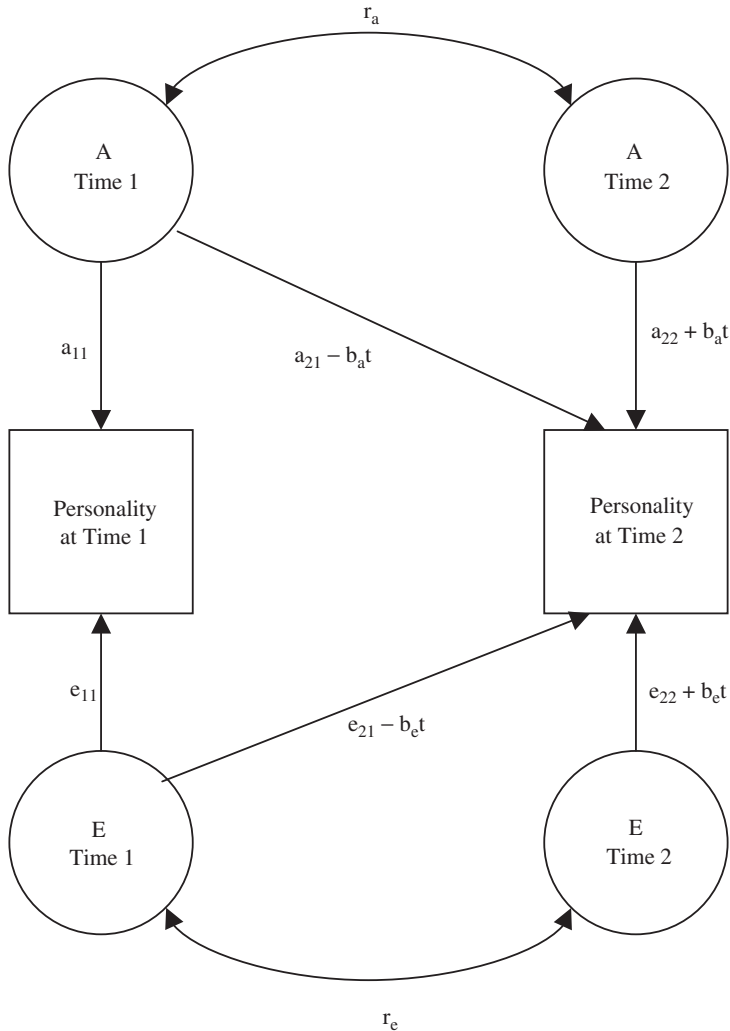
In models of this form, we also assume that there are no gene-environment interactions or correlations. It is possible to develop models assessing the effects of such correlations and interactions (Purcell, 2002), but it is necessary to specify and measure the environmental moderators. When left unmodeled, as we have done here, the presence of gene-environment interactions or correlations do not render the resulting estimates of genetic and environmental influences meaningless. Rather, they state them at overall population levels. Though this could mask differences in underlying subgroups within the data, our goals in this study involved clarification of such overall population level effects. This is an important step in the long-term process of developing conceptualizations of the operation of gene-environment correlations or interactions in a manner coherent and specific enough that they can be tested.

The final assumption is that the portion of variance attributable to genetic influence is additive. Violations of this assumption could mask shared environmental effects in reared-together twin studies, but without reared-apart twins, we have no way of addressing the extent to which the assumption holds in the current study. We note that there have been reports of significant nonadditive effects on personality, particularly as measured by the MPQ (though the particular effects do not generally

replicate), but little evidence for shared environmental effects (Tellegen et al., 1988; Bouchard & Loehlin, 2001).

We began our biometric analyses by using Mx (Neale, 1997) to fit bivariate Cholesky models (ACE models) estimating additive genetic (termed the "A" parameter), and shared or common (termed the "C" parameter) and nonshared (termed the "E" parameter) environmental contributions to variance in and covariance between the age- and sex-adjusted MPQ personality scale scores at the two time points (referred to as Time 1 and Time 2). Because we had a substantial number of broken twin pairs in our sample, we made use of the raw data option in Mx so that the unmatched twins could contribute information about the variances at the two time points. The path coefficients in the Cholesky models we fit were partial regression estimates, with the genetic and environmental path estimates to Time 1 stated directly. The effects of genetic and environmental influences on Time 2 personality, however, were decomposed into those specific to Time 2 and those in common with Time 1. We explicitly made accommodation for the length of the retest interval by allowing it to moderate the decomposition of the genetic and environmental influences on Time 2 personality (a copy of the Mx script we used to do so can be obtained from the first author upon request).

We derived two pieces of information from the Cholesky decomposition. Figure 1 depicts the information we computed. First, we estimated the correlation between the genetic effects on personality at Time 1 and the genetic effects on personality at Time 2. This is known as the *genetic correlation*. The genetic correlation is defined as the square root of the proportion of the total heritability of the Time 2 scale score that can be explained by genetic effects at Time 1. Thus, when the genetic correlation is 1.00, all of the genetic influences are common to the two times. This also means that there is no room for genetic influence on whatever change has taken place over the interval. When the genetic correlation is 0.00, completely different sets of genetic influences act to affect individual differences at the two ages, and change could be genetic or environmental in origin to any degree. In other words, the genetic correlation reflects the extent to which the genetic effects overlap at the two times, regardless of their relative contribution to phenotypic variance. A similar interpretation can be given to correlations of environmental influences, and we made the analogous calculation to estimate the nonshared environmental correlation between scale scores at Times 1 and 2. Thus, if stability in personality were primarily genetic in origin, we would expect high genetic correlations and very low environmental correlations. If stability in personality reflected transactions between genes and environments, we would expect substantial correlations at both the genetic and environmental levels. The shared environmental influences on the personality scale



**Figure 1**

Heuristic for the information that can be derived from a Cholesky decomposition of personality scale scores at Time 1 and Time 2.

A refers to genetic influence and E to nonshared environmental influence.  $r_a$  and  $r_e$  refer to genetic and nonshared environmental correlations.  $t$  refers to the retest interval and  $b_a$  and  $b_e$  to its regression coefficients.

scores were generally so small that the shared environmental correlation between scale scores was not meaningful. We computed the genetic and environmental correlations for the mean retest interval of 5 years for the

sample. Where the length of the retest interval had a significant effect (as measured at  $p = .02$  or less; to recognize that, we applied 14 separate tests), we also computed the range over which these correlations would fall for the retest interval range of 1.0–10.4 years.

## RESULTS

### *Attrition Analysis*

There were small differences between those who completed the MPQ at Time 1 but who did not complete it again at Time 2 and those who completed it at both times. Uncorrected for the effects of multiple testing, the significant differences included the following: Those who completed the MPQ only at Time 1 were slightly younger ( $M = 57.7$  vs.  $M = 59.4$  for those completing it at both times,  $p = .01$ , standardized mean difference  $-.17$ ) and had marginally lower Social Closeness scale scores ( $M = 15.1$  vs.  $M = 15.9$  for those completing it at both times,  $p = .02$ , standardized mean difference  $-.17$ ), higher Alienation scale scores ( $M = 2.3$  vs.  $M = 1.9$  for those completing it at both times,  $p = .02$ , standardized mean difference  $.16$ ), higher Aggression scale scores ( $M = 3.2$  vs.  $M = 2.7$  for those completing it at both times,  $p = .00$ , standardized mean difference  $.20$ ), and lower Harm Avoidance scale scores ( $M = 21.3$  vs.  $M = 22.6$  for those completing it at both times,  $p = .00$ , standardized mean difference  $-.27$ ). All of these differences would generally be considered small (Cohen, 1988).

### *Analysis of Mean Level Change*

Table 2 gives the results of the hierarchical linear model analyses of Time 1 and Time 2 scale scores, conducted to assess the significance of changes in mean level while correcting for the correlated nature of the twin observations. We show the means at the two time points,  $t$ -statistics for the estimated parameters for time, and the associated probabilities. To put the mean differences in perspective, we also show their effect sizes (standardized mean differences). There was a clearly significant mean difference only for Achievement. There were small (Cohen, 1988) mean effect size differences of  $-.15$  for Aggression and  $.23$  for Traditionalism, though the mean difference was not significant for these scales after adjustment for the varying retest interval and correlated twin observations. Because our

**Table 2**  
**Significance Levels and Standardized Effect Sizes for the Mean Difference in Multidimensional Personality Questionnaire Scale Scores Over Time**

Scale	Mean Time 1	Mean Time 2	T-value of difference	Degrees of freedom	Probability	Standardized effect size
<b>Primary</b>						
Well-being	19.85	20.05	.92	1900	.36	.05
Social potency	8.21	7.69	-2.61	1900	.01	-.09
Achievement	12.07	11.76	-3.31	1900	.001	-.07
Social closeness	15.70	15.89	.83	1894	.27	.04
Stress reactivity	8.95	8.88	.65	1885	.52	-.01
Alienation	1.97	1.94	.88	1883	.38	-.01
Aggression	2.83	2.45	.55	1880	.58	-.15
Control	16.84	16.75	.89	1877	.37	-.02
Harm avoidance	22.25	22.64	2.18	1874	.03	.08
Traditionalism	20.24	21.20	2.31	1867	.02	.23
Absorption	14.30	13.96	.74	1847	.46	-.05
<b>Higher order</b>						
Positive emotion	152.35	152.00	-1.17	1847	.24	-.03
Negative emotion	123.15	122.46	-1.93	1845	.05	-.06
Constraint	171.87	173.07	.08	1847	.94	.11

*Note.* Mean differences were tested using a hierarchical linear model measuring fixed effects of retest interval. Effect size is (mean Time 2 less mean Time 1)/pooled standard deviation. It is stated for the average retest interval of 5 years. We made no adjustment to the probability levels for the multiple tests.

sample included some people spanning a large age range, we also explicitly checked for effects in our data of age on extent of change prior to beginning our biometric analyses. We did this by regressing change in raw scale scores on age and retest interval for each scale. There were no significant effects.

### *Biometric Analyses*

We show the twin intraclass correlations for the primary and higher-order MPQ scales at Time 1 and Time 2 in Table 3. Because the MZ

**Table 3**  
**Monozygotic (MZ) and Dizygotic (DZ) Intraclass Correlations at**  
**Time 1 and Time 2 for the Multidimensional Personality**  
**Questionnaire Scales**

Scale	Time 1		Time 2	
	MZ (pairs = 254)	DZ (pairs = 213)	MZ (pairs = 175)	DZ (pairs = 130)
<b>Primary</b>				
Well-being	.27	.06	.33	.08
Social potency	.54	.23	.52	.26
Achievement	.46	.15	.39	.22
Social closeness	.45	.21	.46	.23
Stress reactivity	.49	.16	.46	.25
Alienation	.38	.16	.39	.19
Aggression	.29	.12	.48	.20
Control	.40	.11	.31	.07
Harm avoidance	.34	.22	.31	.20
Traditionalism	.57	.34	.55	.18
Absorption	.40	.29	.44	.12
<b>Higher order</b>				
Positive emotion	.43	.11	.40	.17
Negative emotion	.42	.24	.50	.11
Constraint	.53	.32	.54	.25

*Note.* Standard errors are less than .07 for MZ's and .09 for DZ's.

correlations generally exceeded twice the DZ correlations, genetic influence is suggested for all personality scales, with little, if any, shared environmental influence. The few exceptions to this, showing DZ correlations more than half the MZ correlations, tended to be the scales loading on the Constraint superfactor. Assortative mating could possibly explain the relatively high DZ correlations, and we note that the Constraint superfactor has, in fact, shown more assortative mating than the other superfactors (Lykken & Tellegen, 1993).

Table 4 summarizes the retest correlations within person and across MZ and DZ twin pairs. The cross-twin correlations express the degree of relationship between one twin's score at Time 1 and the co-twin's score at time 2 and therefore reflect the degree of association between the twins over time. Within-person correlations

**Table 4**

Retest Correlations Between Time 1 and Time 2 Testing Within Person and Across Monozygotic and Dizygotic Twin Pairs for the Multidimensional Personality Questionnaire Scales

Scale	Age-sex		Age-sex corrected for unreliability	Cross-twin	
	Raw within person	corrected within person		Monozygotic ( <i>n</i> = 350)	Dizygotic ( <i>n</i> = 260)
<b>Primary</b>					
Well-being	.74	.73	.81	.28	.07
Social potency	.86	.85	1.00	.56	.23
Achievement	.79	.79	.90	.39	.21
Social closeness	.80	.79	.86	.50	.13
Stress reactivity	.82	.81	.91	.48	.25
Alienation	.70	.70	.80	.24	.05
Aggression	.72	.69	.84	.44	.34
Control	.72	.72	.88	.32	.06
Harm avoidance	.72	.68	.77	.31	.18
Traditionalism	.80	.79	.88	.52	.30
Absorption	.80	.79	.87	.52	.17
<b>Higher order</b>					
Positive emotion	.83	.83	.93	.42	.16
Negative emotion	.81	.81	.91	.48	.18
Constraint	.83	.80	.90	.48	.26

*Note.* The cross-twin correlation is the correlation of each twin's score at Time 1 with that of the co-twin at Time 2. Standard errors are less than .02 for within-person, .05 for monozygotic twin, and .06 for dizygotic twin correlations. Age-sex correction for unreliability is based on 30-day retest correlations. 833 individuals completed the MPQ at both time points. Due to missing data for specific items, numbers of individuals from the full sample of 833 contributing to the within person correlations for each scale varied from 809 to 828.

ranged from .70 to .86, with an average of .76; after correction for 30-day retest unreliability, the lowest of these correlations was .77. As for the univariate twin correlations, the cross-twin correlations suggested significant common genetic influences on personality across time and little, if any, common shared environmental influence.

Table 5 presents the computed statistics for the between-time ACE Cholesky models we applied for each MPQ scale. We report the raw variance components scaled to a metric of 100 for Time 1 so that the reader can see both the extent to which they were increasing

**Table 5**  
**Computed Values From the Bivariate Cholesky Decomposition of the**  
**Multidimensional Personality Questionnaire Scale Across Time**

Scale	A		C		E		Totals		Mean time		Range over time	
	T1	T2	T1	T2	T1	T2	T1	T2	r <sub>a</sub>	r <sub>e</sub>	r <sub>a</sub>	r <sub>e</sub>
<b>Primary</b>												
Well-being	27	31	0	0	73	68	100	99	.95	.65	1.00-.87	N/A
Social potency	53	42	2	4	45	49	100	95	1.00	.70	N/A	N/A
Achievement	44	37	0	1	56	62	100	100	.96	.67	N/A	N/A
Social closeness	46	43	0	0	54	54	100	97	1.00	.63	N/A	N/A
Stress reactivity	41	30	4	10	55	60	100	100	1.00	.69	N/A	N/A
Alienation	14	23	25	25	61	56	100	103	.97	.67	1.00-.89	N/A
Aggression	29	29	0	0	71	55	100	84	1.00	.57	N/A	N/A
Control	39	30	0	1	61	72	100	103	1.00	.60	N/A	N/A
Harm avoidance	40	34	0	0	60	64	100	98	1.00	.53	N/A	N/A
Traditionalism	38	36	17	5	45	39	100	80	1.00	.58	N/A	N/A
Absorption	27	35	15	8	58	63	100	106	1.00	.68	N/A	N/A
<b>Higher order</b>												
Positive emotion	42	41	0	0	58	63	100	104	.97	.73	N/A	N/A
Negative emotion	33	45	9	2	58	51	100	98	1.00	.71	N/A	N/A
Constraint	42	44	7	7	51	47	100	98	.93	.64	N/A	.75-.49

*Note.* A refers to additive genetic variance for the scale, C to shared environmental variance, and E to non-shared environmental variance; T1 and T2 refer to Time 1 and Time 2; r<sub>a</sub> and r<sub>e</sub> refer to the genetic and non-shared environmental correlations between variance attributions at Time 1 and Time 2. Mean time genetic and environmental correlations are the estimates based on the mean time interval of 5 years. The range of these correlations over time refers to the estimates over the observed time interval of 1.0–10.0 years. The range is given only when the effect of retest interval was significant at  $p = .02$  or less. The variance components have been scaled to the Time 1 Total level to enable examination of both changes in raw variances and proportions of variance.

or decreasing over time and the proportions of variance that resulted. We computed the moderation effects of the retest interval for both genetic and nonshared environmental influences for all the scales, though we show the effects of these parameters only when they were significant. Each set of statistics measuring genetic and environmental variance components of scale scores was quite consistent across time for all of the scales, with the largest variations

being on the order of 10%. The magnitudes of the proportional influences on the personality scale scores were very comparable to those reported in other studies (Bouchard & Loehlin, 2001), except for the genetic influence on Alienation. That is, excluding Alienation and restating the raw variance components as proportions of the totals, the estimated genetic influences ranged from 27% to 53%, and estimated shared environmental influences were generally negligible, though Alienation, Traditionalism, and Absorption, in particular, showed some shared environmental influence. We believe that the unusually low estimate of genetic influence on Alienation resulted because the variance in that scale was notably smaller than those of the other scales with the exception of Aggression, resulting in depression of the correlations due to restriction of range. The freely estimated genetic correlations ( $r_a$ ) for the mean retest interval of 5.0 years ranged from .93 to 1.00. The estimated nonshared environmental correlations ( $r_e$ ) were also both significant and substantial; they ranged from .53 to .73. For the range of significant retest intervals included in the sample, the nonshared environmental correlations did not drop below .49.

The high genetic correlations and small amounts of mean level change in the data implied that there were no substantive genetic influences on change in personality over the time interval. It was possible, however, that the small mean level changes masked substantial changes at the individual level that could have either genetic or environmental influences. To evaluate this possibility, we computed the MZ and DZ twin correlations for change in raw scale scores over time. The largest correlation was .24, and it was the only one that was significant. Thus, consistent with the genetic correlations we computed, there was little or no evidence for genetic influence on change in personality over time for this sample of primarily older adults.

In our tables, we present the results for the ACE models, in spite of the fact that, for some of the models, including a parameter estimating nonadditive genetic influence instead of a parameter estimating shared environmental influence fit better. There is substantial evidence for the existence of such nonadditive genetic influences on MPQ scales (Finkel & McGue, 1997; Tellegen et al., 1988). Their estimation, however, is fraught with difficulty as they cannot be estimated simultaneously with both additive and shared environmental effects, and the power to estimate them is very weak (Martin, Eaves,

Kearsey, & Davies, 1978). Probably because of this, when they are estimated, their existence seems to vary considerably by sample, personality measurement scale, and over time (Bouchard & McGue, 2003). This was true in our data as well, as nonadditive genetic influences were estimated to be present at Time 1 but absent at Time 2, and vice versa, for several scales. Though we fit models that included nonadditive genetic variance, and they provided some improvement in fit for Wellbeing, Control, and Positive Emotionality, we decided to present the estimates from the ACE model because we believed that model to provide the most consistent estimates from the most general perspective regarding possible sources of genetic influence on personality. Though model fit could be improved by eliminating the C parameter, the relationships among model fit statistics for the types of models we applied remained the same whether including or excluding the C parameter, and these relationships were the real focus of our investigation.

## DISCUSSION

This study replicates prior findings of substantial stability of personality in adulthood (see Roberts & DelVecchio, 2000, for a recent review), and extends those results by focusing primarily on the level of stability of personality in late adulthood. In addition, the study extends previous research by measuring personality facets. Our personality measures, even at the primary scale level, were highly stable and showed genetic influences very similar to those from other personality studies (Bouchard & Loehlin, 2001). For all practical purposes, all of the genetic influences on personality at Time 1 were still present at Time 2 for each of the primary scales measured, as well as for the three higher-order factors, and there was no evidence that new genetic influences had been introduced. This could be viewed from the perspective of substantive genetic contributions to a range of stable latent personality traits, given that the genetic variance components were also substantive. At the same time, our results suggested that the nonshared environment also played a substantial role in the stability we observed, as the nonshared environmental correlations and variance components were also high for all of the scales. This, too, could be viewed from the perspective of a range of stable latent personality traits. In fact, our results indicated that the genetic and nonshared environmental influences on the latent stable

personality traits were roughly equal. This indicates that, though McCrae et al. (2000) appear to be correct that genetic influences contribute importantly to the stability of personality in late adulthood, the contribution of the environment is somewhat more important than their conceptualization would suggest. We think it likely that the nonshared environment makes the contributions to stability that we observed through transactions over time between genes and environment of the sort articulated by Roberts and Caspi (2003).

Increase or decrease in variance over time is one of the key issues in studies of aging. Increasing variance was addressed at some length in Pedersen and Reynolds's (1998) longitudinal personality study, and Viken et al. (1994) also noted increases in variance with age. We presented our findings in such a way that this issue could be evaluated directly here as well. Still, we found no systematic changes in variances over time. In fact, we found that we could improve model fit by constraining the variances at our two time points to be equal for all scales except Traditionalism. Pedersen and Reynolds interpreted the variance increases they observed in the context of terminal drop associated with impending death in the oldest members of their sample. Though our sample consisted primarily of older adults, 90% were under 70 years old at first testing and generally would not be considered elderly or likely to be facing terminal drop. Perhaps this contributed to the lack of increasing variability in our sample, though it would do nothing to explain the difference in this area between our results and those of Viken et al.

We presented raw variance components in our results, leading to the observation that genetic and nonshared environmental contributions to personality were about equal. The high degree of stability in the variances over time, however, makes median summary values expressing proportions of variance meaningful as a way of further exploring the contribution of nonshared environmental influences to the personality stability we observed. That is, corrected for measurement error, the median nonshared environmental correlation we observed was .65, suggesting that stable, nonshared environmental influences play a large role, accounting for over 40% of variance. That 40% of individual differences in personality can be attributed to stable, nonshared environmental influences is a striking finding. It is also a finding that is not unique to this study (Angleitner & Spinath, 2002; Pedersen & Reynolds, 1998; Viken et al., 1994), though

these other studies have drawn little attention to it. It suggests that a substantial portion of an individual's personality cannot be predicted by knowing his or her genetic background, nor by knowing the environment he or she shared with a co-twin. Rather, in keeping with Roberts and Caspi's (2003) conception, it can be predicted by his or her prior personality and the individual circumstances in which he or she lives, including the consequences of prior life events.

For example, the environmental circumstances of people's lives tend to be relatively stable in the 50 to 70 age range in which 73% of our sample fell at first testing (e.g., Schacter & Kuenzi, 2002). This is not to say that change does not take place in this age range: children may be leaving the home, elderly parents may be in need of care, job roles may change due to downsizing, early retirement, or promotion, etc. It is merely to say that, relative to other life periods such as early adulthood, people's lives tend to be subject to less change during later adulthood. This is true in general, but more importantly, it was certainly true for this sample in this geographic region at the particular point in history in which they were assessed, given the cultural-level environmental influences to which they were exposed. That is, in the mid-1990s, when this sample was assessed, Minnesota, where they resided, experienced no major catastrophic events such as wars, earthquakes, hurricanes, large terrorist attacks, major economic upheavals, or other disasters that might tend to cause major upheavals in the lives of many.

The environmental stability for particular individuals within this age range results from the highly individualized combination of circumstances people have chosen over time as a result of their genetically influenced natures and relatively stable circumstances that have emerged for a variety of primarily nonshared environmentally determined reasons. Thus, the environmental stability we have noted here contributes to a gene-environment correlation and/or interaction of the sort behavioral genetic studies have found very difficult to isolate and evaluate (Plomin, DeFries, McClearn, & Rutter, 1997), as there are many different ways in which it can occur. Particularly in this age range, for example, it is very common for people to live for many years in the same community (even in the same house), to be married to the same spouse or not married, to hold the same job, and to experience no changes in the people living in their home, to name just a few sources of possible environmental stability that would not generally be shared with their co-twins. Though change can of course

occur in this age range, for many, much of it is a period after children have left home and menopause for women has taken place and before retirement, major deteriorations in health and mobility, and spousal bereavement take place. With respect to marriage as one source of this kind of stability, Caspi and Herbener (1992, 1993) have presented evidence that spouses do not become more similar in personality over time, suggesting rather that the shared spousal environment plays a significant role in maintaining each spouse's particular personality. This probably takes place through the establishment of the stable individual habits and compromises in lifestyle unique to each married couple. We interpret these environmental sources of stability as contributing directly to the substantial nonshared environmental correlations as well as the raw nonshared variance components we observed.

Since Plomin and Daniels's (1987) review of behavior genetic findings, the nonshared environment has tended to be conceptualized as a combination of measurement error and experiences unique to each individual that are associated with personality change. The resulting searches (e.g., Krueger, Markon, & Bouchard, 2003; Reiss, Neiderhiser, Hetherington, & Plomin, 2000; Turkheimer & Waldron, 2000) for measured, nonshared environmental variables that can account for the substantial portions of the nonshared environmental influences on differences among individuals have revealed the elusive nature of these influences. The substantial component of stable personality that our results and those of others attribute to *stable* nonshared environmental influences suggests that the identification of such stable influences may be a far more promising research approach to the explanation of environmental influences on behavior. Individual experiences are always unique to the individual and thus highly idiosyncratic and difficult to measure. But individual experiences can be highly stable over time, and this stability should make them and their consequences more systematic and thus more readily measurable. This would mean collecting highly specific data on life circumstances in such areas as work, family and marital relationships, place of residence, community involvement, etc. and comparing the degree and sources of the absolute value of personality change in those who had experienced change in specific classes of circumstances to those who had not. Though our data included some measures of life circumstances such as marital status, type of residence, and occupation, many of the questions were not asked at both

time points, nor were they asked in a manner specific enough to evaluate to what extent change may have taken place. In addition, this sample would probably not be the best one in which to study the effects of changing life circumstances on personality change in this manner, as there was very little change in personality to explain.

This should not be taken to imply that we would expect the relative proportions of stable genetic and environmental influences to be constant over the lifespan or constant for people of a given age living in very different environmental circumstances. The very high level of stability of personality across time in our sample may have had an effect on the relative magnitude of the genetic and environmental influences on the latent personality phenotypes we modeled, as well as on the bivariate genetic and environmental influences we estimated. Specifically, for people of younger ages who might be expected to be more subject to developmental influences and to live in less stable circumstances, it seems possible that the relative proportions of genetic influences on these phenotypes would be larger and the stable nonshared environmental influences smaller.

The sample size used in this study is modest for a twin study, and confined to a regional cohort living in relatively stable economic and physical conditions when measured on a historical scale. Thus, care should be taken in drawing generalizations to other samples living in other circumstances. In addition, though the personality measure we used has good psychometric properties, it is only a single (self-report) measure and subject to the limitations of all such measures. This is true as well of the other longitudinal studies of personality (McGue, Bacon, & Lykken, 1993; Pedersen & Reynolds, 1998; Viken et al., 1994). Thus, the question of consistency of stability estimates across reporters and other methods of observation should be investigated as well. The question of genetic and environmental influences on personality stability and change over time should be investigated in other samples, using other personality measures at the facet level and other approaches such as adoption studies. Alternative approaches to evaluate the validity of the assumptions underlying the biometric models we and others have applied should also be pursued. Nonetheless, we believe that our results add substantially to the growing body of evidence for the conception of adult personality as an outgrowth of the transaction between a stable underlying genetic base and a set of environmental influences largely unique to the individual that reflect individual life events and their consequences

over time. Thus, we believe it fair to speculate that adult personality stability results from a combination of genetic and continuing environmental influences, while adult personality change results both from life events beyond the control of the individual and from new individual life choices. We intend to pursue this conception in future research.

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