Genetic Influences on Life Span and Its Relationship to Personality: A 16-Year Follow-Up Study of a Sample of Aging Twins

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Objective: The relationship between personality and life span is not well understood, and no study to date has examined genetic influences underlying this relationship. The present study aimed to explore the phenotypic and genetic relationship between personality and life span, as well as genetic influences on all-cause mortality. **Methods:** Prospective community-based study including 3752 twin individuals older than 50 years. Neuroticism, psychoticism, extraversion, and social desirability and pessimism/optimism were measured at baseline using the Revised Eysenck Personality Questionnaire and the Revised Life Orientation Test, respectively. Information on age at death was obtained 16 years after the initial assessment of personality. **Results:** Extraversion was inversely related to mortality with the risk of death decreasing 3% per unit increase of the extraversion score. Psychoticism and pessimism were positively related to mortality with a 36% and 39% increase in risk of death per unit increase in the respective personality score. Heritability of life span was 7%. Cross-twin cross-trait hazard ratios (HRs) were only significant for optimism/pessimism in monozygotic (MZ) twins with no significant differences in HRs between MZ and dizygotic twins in all traits; however, there was a trend for slightly higher HRs in MZ compared with dizygotic twins in psychoticism and optimism/pessimism. **Conclusions:** Extraversion, psychoticism, and optimism/pessimism are significant predictors of longevity; extraversion is associated with a reduction, and pessimism and psychoticism are associated with an increase in mortality risk. Genetic influences on longevity in Australian twins are very low (7%). Our data also suggest a small, albeit nonsignificant, genetic influence on the relationship of pessimism and psychoticism with life span. **Key words:** longevity, heritability, personality, genetic architecture, mortality, life span.

DZ = dizygotic; **MZ** = monozygotic; **SES** = socioeconomic status; **HR** = hazard ratio; **BMI** = body mass index.

INTRODUCTION

Personality can be defined as the characteristics of a person that account for consistent patterns of feeling, thinking, and behaving (1). The effect of personality on physical health and life span has been and continues to be an important topic in several areas of health care. Considering not only the increase in life expectancy but also the age-related decline in various cognitive and physical domains, the idea of predicting health behavior and mortality based on personality traits is of high interest to the public health system.

A growing number of studies have investigated the relationship between personality and longevity throughout the human life span (2–14). Although most studies reveal a significant personality-mortality association after controlling for other factors influencing mortality (e.g., socioeconomic status [SES], hypertension, smoking and drinking behaviors, physical activity, marital status, education), the findings are partly inconsistent, and only few studies are well replicated. Some studies have not used well-validated personality questionnaires to explore the relationship between personality and mortality, and the range of personality traits measured has varied widely. In addition, many have used relatively short (≤10 years) follow-up periods (2,3,6,7,9,11,13,15) or relied on specific samples of the population, for example, only men or clinical samples (16,17). Furthermore, health-relevant behaviors promoting health or putting it at risk (e.g., smoking, social relationships, or regular visits to the physician) may underlie the association between personality and longevity, but this has not been investigated to any great extent. It is possible that shared genes underlie health or health behavior and particular personality traits (6), but no study to date has explored the extent of genetic and environmental covariation of personality and longevity. Heritability for personality traits ranges from 40% to 50% (18–21). In addition, a life span heritability of 20% to 54% (22–26) has been reported, mainly in Scandinavian samples, with some indication of a higher heritability for men than for women (26) and an increase of heritability of life span after the age of 60 years (22).

The present study has three aims. First, to extend and replicate previous findings on the relationship between personality (extraversion, neuroticism, psychoticism, social desirability, and pessimism/optimism) and life-span utilizing a large population based sample of ageing twins. We use a well-validated and reliable personality questionnaire and a 16 year follow-up period, considerably longer than most previous studies. In addition, we include several important covariates (health behaviors) in order to explore whether they may function as mediators between personality and longevity. This is also the first study to include psychoticism and social desirability as predictors of mortality. Second, this paper is the first to explore heritability of longevity in an Australian sample and a comparatively recent birth cohort (born 1899-1944). Third, we aim to extend previous studies by exploring genetic and environmental influences on the (in our sample) significant relationships between several personality estimates and mortality.

METHODS

Participants

Between 1993 and 1995, a questionnaire was mailed to 4562 individuals older than 50 years who were registered with the Australian Twin Registry (27).

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The study was approved by Queensland Institute of Medical Research – Human Research Ethics Committee. The questionnaire assessed personality traits, somatic and mental health, demographic characteristics, and a wide range of other health and life-style domains. Of the registered twins, 3752 twin individuals (85%) aged between 50 and 94 years (mean [standard deviation {SD}] = 61 [8.8] years) were willing to participate, including 596 identical (monozygotic [MZ]) women, 209 MZ men, 360 nonidentical (dizygotic [DZ]) women, 114 DZ men, 365 DZ opposite-sex twin pairs, and 464 single twins without the co-twin participating. Zygosity was assessed using a standard questionnaire evaluating the degree of physical similarities during childhood. This tool has been shown to be very reliable (28,29). In 5% of the sample, we subsequently confirmed zygosity through genotyping microsatellite markers across the genome (30). Further details of the questionnaire, sampling methods, zygosity determination, and the questionnaire are described elsewhere (31,32).

Measures

Longevity

Because the present article is focusing on normal variation in life span, age at death is used as the primary phenotype of interest here. All participating twin individuals were matched to the Australian National Death Index in January 2010 (approximately 16 years after filling in the original questionnaire) to obtain age at death and cause of death (coded according to the *International Classification of Diseases, Tenth Revision*) for those who had died during the follow-up interval. Data on all-cause mortality were available for the sample until December 31, 2009.

Personality

Neuroticism, Psychoticism, Extraversion, and Social Desirability

The short version of the Revised Eysenck Personality Questionnaire consists of four subscales: neuroticism, psychoticism, extraversion, and social desirability (lie); each assessed with 12 items scored on a 2-point response set (yes/no) (33). The item score for each of the three subscales was imputed with the mean in cases where fewer than three items (25%) were missing; if more items were missing, the respondent's scale score was treated as missing. Scores more than 3 SD above the mean were Winsorized (i.e., psychoticism scores for 12 twins). Because the distribution of the neuroticism and psychoticism scales were skewed, a square root transformation was performed.

Optimism/Pessimism

The Revised Life Orientation Test of optimism and pessimism (34) consists of 10 items, with 3 items assessing optimism, 3 items assessing pessimism, and 4 acting as filler items, scored on a 3-point Likert scale ("yes," "don't know," and "no"). The optimism and pessimism items of the scale can either be examined separately or be treated as a one-dimensional scale (as in the present study) by simply reversing the scores of the three negatively worded items and then adding up the item scores of the six items (34). Final scores ranged between 6 and 18. Twenty-seven scores slightly below 3 SD of the mean were Winsorized. Because the scale was negatively skewed, it was reflected, and a log transformation was applied, so that a low total score indicated optimism, and a high total score indicated pessimism.

Covariates

Sociodemographic Factors (SES, Age, and Sex)

Age, sex, and SES were included as covariates. SES was coded using the Australian Socioeconomic Index 2006, an occupation status scale that provides the means for converting official occupational classifications into occupational status scores (35). As suggested by McMillan et al. (35), if the occupational status score was missing (i.e., no occupation at the time of survey), the past occupation was used to calculate the Australian Socioeconomic Index 2006 score (35), or if not available, the score was substituted with the current (or past) partner's occupational status score. If neither (past) occupation nor spouse's (past) occupation was available, the score was substituted with an individual's (or if not available, the partner's) educational attainment.

Health Behaviors (Cigarette and Alcohol Consumption, Exercise, and Body Mass Index)

The number of cigarettes smoked per day in different phases of life (teens, 20- to 29-year-olds, 30- to 39-year-olds, >40-year-olds, and during the last 12 months) and the number of years smoking (date of survey/quit date minus the initiation date) were assessed. The mean score of cigarettes per day was multiplied by the total number of years smoked to obtain an estimate representative of cigarettes smoked over the lifetime. Finally, the data were converted into a 3-point Likert scale: individuals who never smoked (Category 0), who had a score between 1 and 500 (Category 1), and who had a score higher than 500 (Category 2). Please note that the resulting score is arbitrary because we did not multiply it by days smoked but by years smoked; so to obtain the total number of cigarettes in a lifetime, the number would have to be multiplied by 365 days per year. Frequency of alcohol consumption (daily, three to four times a week, about weekly, about monthly, less often, and never) and the average amount (in standard drinks) of alcohol consumption during the past 12 months were assessed. From these items, the average number of drinks per month (during the last 12 months) was calculated and converted into the following categories: never drank (Category 0), up to 10 drinks per month (Category 1), 10 to 30 drinks per month (Category 2), 30 to 100 drinks per month (Category 3), and more than 100 drinks per month (Category 4). Finally, individuals were asked how they would rate their commitment to sport/exercise during life and were classified accordingly as nonexercisers (no commitment to sport/exercise), occasional exercisers (exercised at least once a week or had an occupation involving walking and manual work for at least 5 hours a week), or as exercisers (exercising at least three times a week). Body mass index (BMI) was calculated from the height and weight information obtained in the original mailed survey. Self-reported current height and weight have been shown to be highly accurate (36). In accordance with the BMI-based classification of the International Obesity Task Force (http://www.iotf.org), participants were categorized as underweight (BMI <18.5 kg/m^2), normal weight (18.5 kg/m$^2 \leq$ BMI < 25 kg/m^2), overweight (25 kg/m$^2 \leq$ BMI < 30 kg/m^2), and obese (BMI ≥ 30 kg/m^2).

Statistical Analysis

Survival Analysis

For mortality analyses, the number of months of survival from entering the study (between 1993 and 1995) to date of death or censoring (for individuals who were still alive at follow-up in 2010) was calculated. A variable indicating the censor state (deceased versus censored) was included to distinguish between individuals who were deceased during the study and individuals who were still alive. Cox proportional hazards regression analysis, a partial likelihood method of estimation taking the number and rank order of deaths into account, as implemented in Stata/SE 11.0 (37), was used to calculate hazard ratios (HRs). A HR represents the effect of a one-unit change in the predictor score (e.g., personality) on the base mortality risk (raw hazard of mortality) during the follow-up period. (Please note that the base mortality risk is relative and may be very small.) For categorical predictors (some of the covariates), the HR represents the difference in mortality risk compared with the reference group (e.g., obese versus normal). To correct for the possible association between the life spans of related individuals (e.g., twins), robust estimators of variance were computed using the cluster option in Stata (37).

Two models were fitted for every trait separately. The baseline model explored the relationship between personality and mortality containing only age and sex as covariates. The second model also included correction for the effects of SES, smoking, drinking, exercise behavior, and BMI to explore whether the relationship between personality and mortality may be mediated by any of these variables. Subsequently, we also fitted a multivariate model including all five traits in the same model to test whether the associations of the different personality traits with all-cause mortality were independent from each other. In line with the Stata guidelines (38), all categorical variables (smoking, drinking, BMI, and exercise) were included as dummy variables. The selection of covariates was based on previous studies showing that each of the variables may have a significant effect on mortality and may also be related to personality. To avoid losing data, missing entries for any of the covariates were substituted with the sample mean. The proportional hazards assumptions (the assumption that covariates multiply hazard) were tested based on Schoenfeld residuals (38). p values

for all four personality traits and for the covariates were nonsignificant (ranging between .19 and .92), indicating that the assumptions were not violated and that the model was specified correctly.

Genetic Modeling

For genetic modeling analyses, the liability distribution of a trait is assumed to be continuously normal, receiving contributions from both independent normally distributed genetic and environmental effects. In the classic twin design, variance in and covariance between traits can be partitioned into that due to genetic (additive [A] and dominant [D]) and environmental (shared within twin pairs [C] and nonshared [E]) influences. Normally, twin correlations can be explained by a combination of these four parameters, and structural equation modeling can be used to determine the combination that best explains the observed data. This is possible because different patterns of MZ and DZ twin pair correlations predict the portions of A, C, D, and E influences. However, C and D influences cannot both be estimated in the same model because they are confounded. In addition, this method is based on the assumption that trait-relevant environments are the same for MZ and DZ twin pairs and that the only difference between MZ and DZ twins is their genetic resemblance, with MZ twins sharing all their genes as opposed to DZ twins who share (on average) only half their genes.

To explore genetic and environmental influences on longevity accounting for censored data, a general mixed-effects Cox proportional hazards model (39) was fitted using the coxme function from the Kinship package for R (40). The makekinship function was used to create the relationship (A) matrix for the pedigree. Because this software does not consider MZ twin pairings and instead treats MZ twins as normal siblings, manual adjustments were made to correct these genetic relationships (i.e., doubling the genetic relationship for MZ twins). The C and D were created similarly. Specific hypotheses regarding the significance of particular parameters in the saturated model (estimating all parameters) can be tested statistically by comparing the goodness-of-fit of various models using likelihood ratio statistics. In the present study, we fitted an ACE and an ADE model first and subsequently compared the fit of more restricted models (e.g., AE only) to explore heritability of mortality.

To explore genetic and environmental influences underlying the covariation between personality and mortality, the cross-twin cross-trait HR has been calculated, meaning that the HR for mortality of one twin is predicted based on the co-twin's personality score for MZ and DZ twins, respectively. If the covariation between the traits was due to genes, we would expect a significantly stronger relationship between the personality score of one twin with the mortality risk of the co-twin in MZ twins (i.e., the prediction would be much more accurate) compared with DZ twins.

RESULTS

Survival Analysis

Table 1 shows summary statistics for the personality traits, risk factors, and survival time in the study. At follow-up, 485 women (19% of all women) and 296 men (25% of all men), 781 individuals in total (21% of the total sample), had died. Of these individuals, five were excluded because they died of nonnatural or violent causes. Of the remainder, 220 individuals had cancer as an underlying cause of death, 206 individuals had heart disease as an underlying cause of death, 26 individuals had both disorders as underlying causes of death, and the rest had either died of other causes or cause of death was not specified. Table 2 shows information on death status by zygosity.

Table 3 shows the results of survival analyses for the five personality traits adjusted for age and sex (Model 1) and for all covariates (Model 2). As expected, age and sex had a significant effect on mortality, with the HR increasing with age (12% per year) and being male (36%). We also tested for an age-sex interaction, but this was not significant. Aside from a slight decrease in significance with adjustment for all covariates, the findings did not differ between the two models. The fully adjusted model shows that extraversion was inversely related to all-cause mortality with the risk of death decreasing 3% per unit increase of the extraversion score. In contrast, psychoticism and pessimism (or optimism) were positively related to all-cause mortality with a 36% and 39% increase in risk of death per unit increase in psychoticism and pessimism, respectively. There was no significant effect of neuroticism or social desirability on all-cause mortality.

TABLE 1. Summary Statistics for the Different Traits and Risk Factors Showing Means (SDs) and Frequencies, Both for the Full and Deceased Samples

	Full Sample (N = 3752)	Only Deceased (n = 781)
Trait, M (SD)		
Months of survival from first testing	168.2 (41.0)	104.9 (53.2)
Neuroticism	4.0 (3.2)	4.0 (3.1)
Extraversion	6.6 (3.4)	6.2 (3.3)
Psychoticism	1.7 (1.4)	1.9 (1.4)
Lie (social desirability)	6.4 (2.8)	7.1 (2.7)
Optimism/pessimism[a]	15.4 (2.5)	15.1 (2.5)
Risk factors		
Sex, %		
Women	69	62
Age at baseline, M (SD)	61.3 (8.9)	69.4 (9.4)
Socioeconomic status, M (SD)	46.9 (20.2)	45.3 (19.1)
Smoking, %		
Nonsmoker	41	33
Light smoker	49	52
Heavy smoker	10	15
Alcohol consumption, %		
Never drank	20	22
10 drinks per month (category 1)	55	57
10–30 drinks per month (category 2)	14	9
30–100 drinks per month (category 3)	8	8
> 100 drinks per month (category 4)	3	4
BMI, %		
Underweight	2	4
Normal	60	61
Overweight	29	27
Obese	9	7
Exercise, %		
No exercise	9	9
Occasional exercisers	48	53
Exercisers	43	38

SD = standard deviation; M = mean; BMI = body mass index.
[a] Optimism and pessimism was scored as a one-factor bipolar scale, with a high score indicating optimism and a low score indicating pessimism. For further analyses (i.e., those reported in Tables 3 and 4), the score has been reflected.

TABLE 2. Composition of Twin Survival Data as of January 2010 Showing the Number of Pairs by Zygosity and Death Status for Complete Twin Pairs[a]

	Both Alive		One Dead		Both Dead		Total	
	n	Age, M (SD)	*n*	Age, M (SD)	*n*	Age, M (SD)	*n*	Age, M (SD)
MZF	425	58.6 (6.7)	115	65.3 (8.7)	56	75.7 (7.9)	596	61.5 (9.0)
DZF	261	58.4 (6.5)	71	66.3 (8.7)	28	76.4 (6.8)	360	61.3 (8.8)
MZM	137	57.7 (6.1)	50	65.4 (7.5)	22	74.1 (8.8)	209	61.2 (8.7)
DZM	62	58.4 (6.6)	34	64.9 (9.5)	18	68.8 (8.3)	114	62.0 (8.8)
DZ opposite sex	226	58.8 (7.0)	102	65.3 (8.1)	37	72.8 (7.4)	365	62.0 (8.7)
Total	1111	58.4 (6.7)	372	65.5 (8.4)	161	74.2 (8.0)	1644	61.6 (8.9)

M = mean; SD = standard deviation; MZF = monozygotic female; DZF = dizygotic female; MZM = monozygotic male; DZM = dizygotic male; DZ = dizygotic.
[a] There were 464 single twins without a participating co-twin, of whom 83 died.
The twins from the original study have been matched on mortality status by the Australian National Death Index.

Thus, overall, the association between personality trait and risk of death did not change after adjusting for the different covariates, indicating that the covariates only had a marginal effect. Apart from age and sex, only heavy smoking was a significant independent predictor of all-cause mortality in the fully adjusted model. No significant interaction effects among the covariates could be detected.

The results did not change when, subsequently, the analyses were repeated with all five variables in the same model (results not shown). When men and women were modeled separately (results not shown), none of the personality traits (when adjusted for all covariates) were significantly related to death, most likely because of the reduction in power.

TABLE 3. Hazard Ratios (95% Confidence Intervals) of a One-Unit Increase in the Personality Traits on the Risk of All-Cause Mortality Corrected for Age and Sex (Model 1) and Corrected for All Covariates (Model 2)

	Model 1	Model 2
Neuroticism	1.10 (0.97–1.25)	1.05 (0.92–1.19)
Extraversion	0.97 (0.94–1.00)*	0.97 (0.94–1.00)*
Psychoticism	1.45 (1.13–1.85)**	1.36 (1.07–1.75)*
Lie	1.00 (0.96–1.03)	1.00 (0.97–1.04)
Pessimism[a]	1.55 (1.17–2.06)**	1.39 (1.03–1.88)*
Risk factors		
Age	1.12 (1.11–1.13)**	1.12 (1.11–1.13)**
Sex	0.64 (0.53–0.76)**	0.64 (0.53–0.76)**
SES		1.00 (0.99–1.00)
Light smoker		1.19 (0.98–1.46)
Heavy smoker		1.85 (1.43–2.38)**
Drinks, Category 1		1.04 (0.85–1.28)
Drinks, Category 2		0.81 (0.60–1.10)
Drinks, Category 3		0.89 (0.63–1.26)
Drinks, Category 4		1.27 (0.79–2.03)
Underweight		1.44 (0.91–2.28)
Overweight		1.12 (0.93–1.36)
Obese		1.14 (0.82–1.57)
Occasional exercisers		0.91 (0.68–1.21)
Exercisers		0.89 (0.67–1.18)

SES = socioeconomic status.
* $p < .05$, ** $p < .01$.
[a] Optimism/pessimism was scored as a one-factor bipolar scale, with a high score indicating pessimism.
For categorical predictors (smoking, drinking, body mass index, and exercise), the hazard ratio represents the difference in mortality risk compared with the reference group (i.e., drinkers are compared with nondrinkers, smokers are compared with nonsmokers, overweight/underweight are compared with normal, and exercisers are compared with nonexercisers).

Genetic Modeling

C and D could be dropped without significant loss of model fit, indicating that an AE model fitted the mortality data best. Additive genetic influences were small but significant ($\Delta\chi^2 = 10.06$, $df = 1$, $p < .01$), with a heritability estimate of 7%.

Table 4 shows the cross-twin cross-trait HRs for extraversion, psychoticism, and pessimism/optimism. The only significant HR was for the pessimism/optimism measure for MZ twins. There was no significant difference in HRs between MZ and DZ twins; however, there was a trend for slightly higher HRs in MZ compared with DZ twins in psychoticism and pessimism (optimism). The wide and overlapping confidence intervals indicate low power for these analyses.

DISCUSSION

In the present study, we aimed to a) replicate and extend previous findings on the relationship between five personality traits (pessimism/optimism, extraversion, neuroticism, psychoticism,

TABLE 4. Cross-Twin Cross-Trait HRs for MZ and DZ Twins Corrected for Age and Sex Predicting Risk of Mortality for One Twin Based on the Personality Score of the Co-Twin

	MZ		DZ	
	HR (95% CI)	*p*	HR (95% CI)	*p*
Extraversion	0.99 (0.95–1.02)	.46	0.97 (0.93–1.00)	.09
Psychoticism	1.24 (0.87–1.78)	.23	1.02 (0.71–1.46)	.92
Pessimism[a]	1.58 (1.02–2.44)	.04	1.03 (0.68–1.56)	.90

HR = hazard ratio; MZ = monozygotic; DZ = dizygotic; CI = confidence interval.
[a] Optimism/pessimism was scored as a one-factor bipolar scale.

and social desirability) and all-cause mortality, b) estimate the heritability of longevity in an Australian sample, and c) investigate the extent that the personality-mortality relationship is due to common genetic or environmental influences. Cox proportional hazards regression model of the five personality traits adjusted for age and sex showed that mortality risk was significantly greater in individuals low in optimism or extraversion or high in psychoticism, whereas neuroticism and social desirability showed no association with all-cause mortality. This indicates that extraversion and optimism may serve as protective factors, whereas a high score in psychoticism is a risk factor. Our findings remained unchanged after adjusting for potential confounders, indicating that the personality-mortality relationship was not significantly mediated by behavioral choices such as exercise, smoking and drinking behavior, and BMI or by SES. Furthermore, when the analysis was repeated with all five variables in the model, results were comparable, suggesting that the associations of extraversion, psychoticism, and optimism with all-cause mortality were largely independent.

In line with the present results, previous studies have shown that extraversion and optimism serve as protective factors against mortality (2,3,11). This is generally explained by the following (3): first, high extraversion and optimism are associated with external attribution style, positive emotions, and higher self-esteem (41,42), which, in turn, may reduce the impact of environmental stressors (42,43). Second, a high level of extraversion is associated with reduced risk of incident disability, chronic diseases, and bereavement (44), strong risk factors for mortality in the elderly (45,46). Third, extraverted and optimistic individuals tend to be more sociable and active and have more social contacts (individuals with pleasant personality traits may attract more people) (42), which, in turn, has been shown to be a protective factor against stress and disease (42,43) and has been associated with longevity (47,48). Another explanation, supported by several studies, is the behavioral differences between optimistic and pessimistic individuals, such as optimistic individuals adopting more effective and favorable coping strategies in demanding situations (49,50) and more actively engaging in health-promoting behavior, that is, taking vitamins, having a better diet, and avoiding potential risk areas (42,46).

Our finding of no relationship between neuroticism and mortality is in line with some studies (3,12,15,17), whereas others report a positive relationship (5,11,16) and some even found a protective effect of neuroticism on mortality (7,8,13). An explanation for these contradicting findings could be that the harmful effect of stress or negative emotions on longevity in individuals scoring high in neuroticism may be canceled out by a protective role of this trait against mortality by increasing the motivation for physician visits (3).

Finally, the new finding of a relationship between psychoticism and all-cause mortality, suggesting psychoticism to be a risk factor, is in line with a study (51) reporting some indication of an increased risk for myocardial infarction in individuals scoring high on psychoticism. In addition, high scores on the psychoticism scale have been associated with a lack of health protective behavior (52), which, in turn, may explain the higher mortality risk. Furthermore, individuals high in psychoticism have been described as solitary, not caring, insensitive, having difficulty fitting in, troublesome, inhumane, cruel, and lacking feelings such as empathy (53), all traits associated with social stress, negative emotions, and low level of social support. Interpreted in line with the "diathesis-stress to resilience model" (54,55) – proposing that there are important associations between personality traits, stress, and health outcomes – extraversion as well as optimism may serve as enhancers of resilience, while psychoticism may decrease resilience and immune functions which in turn may decrease life-expectancy. Despite neuroticism not being related to mortality in the present study, it has been known to be associated with an increased vulnerability to stress. As explained previously, this expected negative effect might be in conflict, although with a protective role of neuroticism resulting in mixed study findings.

Social desirability is generally not included in studies using the Eysenck Personality Questionnaire because, apart from measuring social conformity, it is mainly used to detect malingering or inaccurate self-assessment. We decided to include the trait because social conformity or desirability may also be related to mortality. However, our results showed that it seemed to have no effect on longevity.

Heritability of life span/longevity was 7% in our sample, which is rather small compared with past studies (mainly conducted in Scandinavian cohorts), reporting heritability estimates ranging between 20% and 54% (22–26). There is no other Australian study exploring heritability of life span. However, this small estimate is most likely due to the high proportion of censoring (79%) in the present sample effectively increasing our E component. The fact that the present sample consisted of a relatively young cohort (born between 1899 and 1944) may also affect heritability. For example, the health system, medical and pharmaceutical products, and quality of life have much improved during the last 100 years, meaning that much more “life-prolonging” methods are available, which, in turn, might decrease the twin correlations for date of death, increasing nonshared environmental influences. Similarly, many disorders fatal not too many years ago may now easily be diagnosed and treated (e.g., diabetes).

The present study is the first to explore the genetic architecture underlying the relationship between personality traits with longevity. The only significant cross-twin cross-trait HR was for pessimism in MZ twins, indicating that the pessimism/optimism score of one twin was a good predictor of the mortality risk of the co-twin, with a 58% risk increase for one twin, with each score increase in pessimism of the co-twin. Although not significantly different, most likely due to a lack of power caused by subdividing a sample, already short on individuals who actually have died during follow-up, into even smaller subgroups (MZ/DZ twins), the trend toward higher MZ compared with DZ HRs in pessimism/optimism and psychoticism suggests possible genetic influences in the covariation of these traits with longevity.

Based on our study design, we cannot draw any conclusions regarding the causality of the relationship between personality and survival. Although personality may directly influence

longevity, there also could be underlying pathways mediating the relationship, and finally, approaching death may influence personality. However, the latter explanation is the least likely because personality has been shown to be a relatively stable trait throughout one's lifetime (34,53), and also, the mean survival time in the present study (of the individuals who died during the study time) was approximately 9 years, which would mean that personality started to change on an average of 9 years before death, which would seem to be a long period and therefore rather unlikely. This highlights the importance of a long follow-up period for future studies exploring the personality-survival relationship.

Our results must be interpreted recognizing the limitations of our study. Despite the relatively large study sample, the low prevalence of mortality (21%) necessarily reduced precision of estimated parameters. The overlapping confidence intervals for MZ and DZ cross-twin cross-trait HRs indicate that partitioning the variance into genetic and environmental components would be compromised. However, we intend to keep following up the present sample, so power will increase eventually. Furthermore, the fact that, of all covariates (apart from age and sex), only heavy smoking had a significant effect on longevity could also be influenced by the way the covariates were scored. For example, because alcohol consumption was assessed during the past 12 months only, individuals who engaged in heavy drinking before that period and then stopped would have been categorized as nondrinkers. In addition, the exercise questions included in the baseline questionnaire focused on exercise behavior over lifetime. Although physical activity patterns have been shown to be fairly stable over lifetime (56), they can change in different periods of life, which may result in misclassification of those individuals who exercised a lot when they were younger and stopped when they got older or vice versa. Another reason for the nonsignificant effect of the covariates could be that, of the final sample, between 11% (BMI) and 24% (drinking) of the scores, depending on the variable, were missing and were replaced with the sample mean of the specific variable to prevent the loss of valuable information. However, despite being nonsignificant, the covariates clearly showed a strong trend toward the expected influence. Finally, another possible limitation of this study is that, with the model applied to the data, we did not account for the possibility of gene-environment interaction or correlation but only explored the influence of main genetic and environmental effects.

Findings of the present study may assist the development of effective intervention strategies in the elderly to facilitate healthy aging and longevity. The finding that some personality traits, independently of life-style/behavioral choices, serve as protective or risk factors for mortality, and the indication that the covariation between personality and mortality may largely be due to environmental influences shows that it potentially could be possible to influence longevity by facilitating change in these traits. For example, therapeutic interventions (e.g., cognitive-behavior therapy) may help individuals with unfavorable personality traits in longevity to change their thought processes and resulting behavior toward more positive or favorable qualities and thereby enhancing their ability to cope with stress. This, in turn, may facilitate healthy aging.

In summary, we found a significant relationship between extraversion, psychoticism, and optimism and life span, with extraversion and optimism significantly reducing and psychoticism increasing mortality risk. Neuroticism and social desirability seemed to be unrelated to longevity. Heritability of life span in the present sample was estimated to be rather low with 7%. There was some indication for genetic influences underlying the relationship between the psychoticism and optimism, and longevity, although larger studies are needed to confirm this.

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REFERENCES

1. Pervin LA, Cervone D, John OP. Personality Theory and Research. 9th ed. Hoboken, NJ: Wiley; 2005.
2. Fry PS, Debats DL. Perfectionism and the five-factor personality traits as predictors of mortality in older adults. J Health Psychol 2009;14:513–24.
3. Iwasa H, Masui Y, Gondo Y, Inagaki H, Kawaai C, Suzuki T. Personality and all-cause mortality among older adults dwelling in a Japanese community: a five-year population-based prospective cohort study. Am J Geriatr Psychiatry 2008;16:399–405.
4. Martin LR, Friedman HS, Schwartz JE. Personality and mortality risk across the life span: the importance of conscientiousness as a biopsychosocial attribute. Health Psychol 2007;26:428–36.
5. Shipley BA, Weiss A, Der G, Taylor MD, Deary IJ. Neuroticism, extraversion, and mortality in the UK Health and Lifestyle Survey: a 21-year prospective cohort study. Psychosom Med 2007;69:923–31.
6. Taylor MD, Whiteman MC, Fowkes GR, Lee AJ, Allerhand M, Deary IJ. Five factor model personality traits and all-cause mortality in the Edinburgh Artery Study cohort. Psychosom Med 2009;71:631–41.
7. Weiss A, Costa PT. Domain and facet personality predictors of all-cause mortality among Medicare patients aged 65 to 100. Psychosom Med 2005; 67:724–33.
8. Ploubidis GB, Grundy E. Personality and all cause mortality: evidence for indirect links. Pers Individ Differ 2009;47:203–8.
9. Chapman BP, Fiscella K, Kawachi I, Duberstein PR. Personality, socioeconomic status, and all-cause mortality in the United States. Am J Epidemiol 2009;171:83–92.
10. Almada SJ, Zonderman AB, Dyer AR, Shekelle RB, Daviglus ML, Stamler J, Costa PT. Cynicism, neuroticism, and 25-year mortality in the Western Electric Company Study. Psychosom Med 1990;52:222.
11. Wilson RS, Krueger KR, Gu LP, Bienias JL, De Leon CFM, Evans DA. Neuroticism, extraversion, and mortality in a defined population of older persons. Psychosom Med 2005;67:841–5.
12. Huppert FA, Whittington JE. Symptoms of psychological distress predict 7-year mortality. Psychol Med 1995;25:1073–86.
13. Korten AE, Jorm AF, Jiao Z, Letenneur L, Jacomb PA, Henderson AS, Christensen H, Rodgers B. Health, cognitive, and psychosocial factors as predictors of mortality in an elderly community sample. J Epidemiol Community Health 1999;53:83–8.
14. Grossardt BR, Bower JH, Geda YE, Colligan RC, Rocca WA. Pessimistic, anxious, and depressive personality traits predict all-cause mortality: the Mayo Clinic cohort study of personality and aging. Psychosom Med 2009; 71:491–500.
15. Wilson RS, de Leon CFM, Bienias JL, Evans DA, Bennett DA. Personality

and mortality in old age. J Gerontol Ser B Psychol Sci Soc Sci 2004;59: P110–6.
16. Christensen AJ, Ehlers SL, Wiebe JS, Moran PJ, Raichle K, Ferneyhough K, Lawton WJ. Patient personality and mortality: a 4-year prospective examination of chronic renal insufficiency. Health Psychol 2002;21:315–20.
17. Almada SJ, Zonderman AB, Shekelle RB, Dyer AR, Daviglus ML, Costa PT, Stamler J. Neuroticism and cynicism and risk of death in middle-aged men—the Western Electric Study. Psychosom Med 1991;53:165–75.
18. Keller MC, Coventry WL, Heath AC, Martin NG. Widespread evidence for non-additive genetic variation in Cloninger's and Eysenck's personality dimensions using a twin plus sibling design. Behav Genet 2005;35: 707–21.
19. Jang KL, Livesley WJ, Vernon PA. Heritability of the big five personality dimension and their facets: a twin study. J Pers 1996;64:577–91.
20. Gillespie NA, Johnstone SJ, Boyce P, Heath AC, Martin NG. The genetic and environmental relationship between the Interpersonal Sensitivity Measure (IPSM) and the personality dimensions of Eysenck and Cloninger. Pers Individ Differ 2001;31:1039–51.
21. Heath AC, Cloninger CR, Martin NG. Testing a model for the genetic structure of personality—a comparison of the personality systems of Cloninger and Eysenck. J Pers Social Psychol 1994;66:762–75.
22. Hjelmborg JV, Iachine I, Skytthe A, Vaupel JW, McGue M, Koskenvuo M, Kaprio J, Pedersen NL, Christensen K. Genetic influence on human lifespan and longevity. Hum Genet 2006;119:312–21.
23. Skytthe A, Pedersen NL, Kaprio J, Stazi MA, Hjelmborg JVB, Iachine I, Vaupel JW, Christensen K. Longevity studies in GenomEUtwin. Twin Res 2003;6:448–54.
24. Ljungquist B, Berg S, Lanke J, McClearn GE, Pedersen NL. The effect of genetic factors for longevity: a comparison of identical and fraternal twins in the Swedish Twin Registry. J Gerontol Ser A Biol Sci Med Sci 1998;53:M441–6.
25. McGue M, Vaupel JW, Holm N, Harvald B. Longevity is moderately heritable in a sample of Danish twins born 1870–1880. J Gerontol 1993; 48:B237–44.
26. Herskind AM, McGue M, Holm NV, Sorensen TIA, Harvald B, Vaupel JW. The heritability of human longevity: a population-based study of 2872 Danish twin pairs born 1870–1900. Hum Genet 1996;97:319–23.
27. Hopper JL. The Australian Twin Registry. Twin Res 2002;5:329–36.
28. Kasriel J, Eaves L. Zygosity of twins—further evidence on agreement between diagnosis by blood-groups and written questionnaires. J Biosoc Sci 1976;8:263–6.
29. Martin NG, Martin PG. Inheritance of scholastic abilities in a sample of twins. 1. Ascertainment of sample and diagnosis of zygosity. Ann Hum Genet 1975;39:213–8.
30. Cornes BK, Medland SE, Ferreira MAR, Morley KI, Duffy DL, Heijmans BT, Montgomery GW, Martin NG. Sex-limited genome-wide linkage scan for body mass index in an unselected sample of 933 Australian twin families. Twin Res Hum Genet 2005;8:616–32.
31. Bucholz KK, Heath AC, Madden PAF, Slutske WS, Statham DJ, Dunne MP, Martin NG. Drinking in an older population: cross-sectional and longitudinal data from the Australian Twin Registry. In: Gomberg ESL, Hegedus AM, Zucker RA, editors. Alcohol Problems and Aging. Bethesda, MD: National Institutes of Health; 1998:41–62.
32. Mosing M, Zietsch B, Shekar S, Wright M, Martin N. Genetic and environmental influences on optimism and its relationship to mental and self-rated health: a study of aging twins. Behav Genet 2009;39:597–604.
33. Eysenck SBG, Eysenck HJ, Barrett P. A revised version of the psychoticism scale. Pers Individ Differ 1985;6:21–9.
34. Scheier MF, Carver CS. Optimism, coping, and health—assessment and implications of generalized outcome expectancies. Health Psychol 1985;4: 219–47.
35. McMillan J, Beavis A, Jones FL. A new socioeconomic index for Australia. J Sociol 2009;45:123–49.
36. Romero-Corral A, Montori VM, Somers VK, Korinek J, Thomas RJ, Allison TG, Mookadam F, Lopez-Jimenez F. Association of bodyweight with total mortality and with cardiovascular events in coronary artery disease: a systematic review of cohort studies. Lancet 2006;368:666–78.
37. StataCorp. Stata Statistical Software: Release 11. College Station, TX: StataCorp LP; 2009.
38. Cleves M, Gould WW, Gutierrez RG, Marchenko YU. An Introduction to Survival Analysis Using Stata. 2nd ed. College Station, TX: Stata Press; 2008.
39. Therneau TM, Grambsch PM. Modeling Survival Data: Extending the Cox Model. New York, NY: Springer-Verlag; 2000.
40. R Development Core Team. R: A Language and Environment for Statistical Computing. Vienna, Austria: R Foundation for Statistical Computing; 2010.
41. Marshall GN, Wortman CB, Vickers RR, Kusulas JW, Hervig LK. The 5-factor model of personality as a framework for personality-health research. J Pers Soc Psychol 1994;67:278–86.
42. Carver CS, Scheier MF, Segerstrom SC. Optimism. Clin Psychol Rev 2010;30:879–89.
43. Swickert RJ, Rosentreter CJ, Hittner JB, Mushrush JE. Extraversion, social support processes, and stress. Pers Individ Differ 2002;32:877–91.
44. Krueger KR, Wilson RS, Shah RC, Tang YX, Bennett DA. Personality and incident disability in older persons. Age Ageing 2006;35:428–33.
45. Ramos LR, Simoes EJ, Albert MS. Dependence in activities of daily living and cognitive impairment strongly predicted mortality in older urban residents in Brazil: a 2-year follow-up. J Am Geriatr Soc 2001;49:1168–75.
46. Diener E, Chan MY. Happy people life longer: subjective well-being contributes to health and longevity. Appl Psychol Health Well-being 2011;3:1–43.
47. Rasulo D, Christensen K, Tomassini C. The influence of social relations on mortality in later life: a study on elderly Danish twins. Gerontologist 2005;45:601–8.
48. Lyyra TM, Heikkinen RL. Perceived social support and mortality in older people. J Gerontol B Psychol Sci Soc Sci 2006;61:S147–52.
49. Nes LS, Segerstrom SC. Dispositional optimism and coping: a meta-analytic review. Pers Soc Psychol Rev 2006;10:235–51.
50. Rasmussen HN, Wrosch C, Scheier MF, Carver CS. Self-regulation processes and health: the importance of optimism and goal adjustment. J Pers 2006;74:1721–47.
51. Sturmer T, Hasselbach P, Amelang M. Personality, lifestyle, and risk of cardiovascular disease and cancer: follow-up of population based cohort. BMJ 2006;332:1359–62.
52. Brayne C, Do KA, Green L, Green AC. Is health protective behaviour in adolescents related to personality? A study of sun protective behaviour and the Eysenck Personality Questionnaire (junior version) in Queensland. Pers Individ Differ 1998;25:889–95.
53. Eysenck HJ, Eysenck SBG. Manual of the Eysenck Personality Scales (EPS Adult). London, UK: Hodder & Stoughton; 1991.
54. Gatz M. Toward a developmentally informed theory of mental disorder in older adults. In: Lomrantz J, editor. Handbook of Aging and Mental Health: An Integrative Approach. New York, NY: Plenum Press; 1998: 101–20.
55. Hobfoll SE. Conservation of resources, stress, and aging: why do some slide and some spring? In: Lomrantz J, editor. Handbook of Aging and Mental Health: An Integrative Approach. New York, NY: Plenum Press; 1998:121–34.
56. Friedman HS, Martin LR, Tucker JS, Criqui MH, Kern ML, Reynolds CA. Stability of physical activity across the lifespan. J Health Psychol 2008; 13:1092–104.