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Pessimistic, Anxious, and Depressive Personality Traits Predict All-Cause Mortality: The Mayo Clinic Cohort Study of Personality and Aging

Brandon R. Grossardt, MS, James H. Bower, MD, MSc, Yonas E. Geda, MD, MSc, Robert C. Colligan, PhD, and Walter A. Rocca, MD, MPH

Division of Biomedical Statistics and Informatics (B.R.G.), Department of Health Sciences Research; Department of Neurology (J.H.B., W.A.R.); Department of Psychiatry and Psychology (Y.E.G., R.C.C.); Division of Epidemiology (Y.E.G., W.A.R.), Department of Health Sciences Research, College of Medicine, Mayo Clinic, Rochester, MN

Abstract

Objective—To study the association between several personality traits and all-cause mortality.

Methods—We established a historical cohort of 7216 subjects who completed the Minnesota Multiphasic Personality Inventory (MMPI) for research at the Mayo Clinic from 1962 to 1965, and who resided within a 120-mile radius centered in Rochester, MN. A total of 7080 subjects (98.1%) were followed over four decades either actively (via a direct or proxy telephone interview) or passively (via review of medical records or by obtaining their death certificates). We examined the association of pessimistic, anxious, and depressive personality traits (as measured using MMPI scales) with all-cause mortality.

Results—A total of 4634 subjects (65.5%) died during follow-up. Pessimistic, anxious, and depressive personality traits were associated with increased all-cause mortality in both men and women. In addition, we observed a linear trend of increasing risk from the first to the fourth quartile for all three scales. Results were similar in additional analyses considering the personality scores as continuous variables, in analyses combining the three personality traits into a composite neuroticism score, and in several sets of sensitivity analyses. These associations remained significant even when personality was measured early in life (ages 20 to 39 years).

Conclusions—Our findings suggest that personality traits related to neuroticism are associated with an increased risk of all-cause mortality even when they are measured early in life.

Keywords

personality; mortality; pessimism; anxiety; depression; neuroticism; MMPI

INTRODUCTION

Several studies investigated the association of neuroticism-related personality traits with mortality (1). However, previous findings are mixed because a majority of studies showed that neuroticism is a risk factor (2–11), one study showed that neuroticism is protective (12), and another study showed no association (13). These conflicting findings may be due to important methodological differences across studies. For example, investigators used

Address correspondence and reprint requests to Dr. W.A. Rocca, Division of Epidemiology, Department of Health Sciences Research, Mayo Clinic, 200 First Street SW, Rochester, MN 55905, USA; [telephone: (507) 284-3568; fax: (507) 284-1516; rocca@mayo.edu].

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different instruments to measure neuroticism, tested subjects at different ages, and followed them for different lengths of time. Age at the time of testing is particularly important because anxious or depressive traits measured late in life could be consequences of existing health problems rather than predictors (8).

Prompted by these uncertainties, we investigated the relationship of neuroticism-related personality traits with all-cause mortality in the Mayo Clinic Cohort Study of Personality and Aging (14). Special features of our historical cohort study were the large sample size, the approximately equal number of men and women, and the long follow-up of the study participants. This allowed us to stratify analyses by age at the time of personality testing and to address the possibility of a cause-effect inversion (8). We also explored possible differences in men and women and conducted several sets of sensitivity analyses.

MATERIALS AND METHODS

Participants

The Mayo Clinic Cohort Study of Personality and Aging is a geographically defined subset of a previously established Minnesota Multiphasic Personality Inventory (MMPI) cohort. The established cohort was described extensively elsewhere (11,15–18). In brief, approximately 50,000 consecutive Mayo Clinic outpatients were administered the MMPI from 1962 to 1965 for research purposes (not because of clinical indication). The study included ambulatory patients from anywhere in the U.S. who were referred to the Mayo Clinic for any medical reason other than an acute illness, a surgical procedure, a psychiatric disorder, or for hospitalization. The participation rate was 95% (15).

For the present study, we restricted the established cohort to subjects who resided within a cluster of 72 counties located approximately within a 120-mile radius centered in Rochester, MN, as described elsewhere (14). The restriction to a geographic region proximate to Rochester increased the likelihood that cohort members had used the Mayo Clinic as a primary source of medical care following the baseline MMPI testing (contributing to passive follow-up). Details regarding the final inclusion of 7216 subjects in the current cohort are reported elsewhere (14).

Personality Testing

The MMPI includes 550 different statements concerning thoughts, feelings, attitudes, physical and emotional symptoms, and life experiences. Patients are asked to respond “true” or “false” to each statement in a self-administered questionnaire (19). Hathaway and McKinley originally intended the MMPI to be used as a case finding instrument, and MMPI data provide a standardized and quantitative measure of personality traits (19). Specific clinical scales on the MMPI were developed through cross-validation with independent samples of diagnostic groups.

Pessimistic, anxious, and depressive personality traits were selected as scales of primary interest because they have been found to be related to aging and aging-related diseases (11,17,18,20–24). Elevated scores on the three primary scales indicate personality traits consistent with the respective clinical entities. We have shown previously that the 75th percentile cut-off corresponds to a clinically meaningful classification of the personality traits (14). However, the interpretation of low scores is clinically uncertain (e.g., a low pessimistic trait score may not necessarily indicate an optimistic person).

As a measure of pessimistic personality, we used the raw CoNeg scale as described by Colligan and colleagues (23). An elevation on this scale is associated with feelings of negativity, pessimism, and a personality style consistent with the theory of learned

helplessness (Seligman's theory) (21–24). The 192 items in this scale and their corresponding weights were reported elsewhere (score ranging from 0 to 2797) (23). We will refer to this scale as the “pessimism scale”. For the anxious personality scale (historically called Scale 7 [Pt, Psychasthenia], 48 items with equal weight), an elevation indicates generalized feelings of anxiety and discomfort. Furthermore, rumination about personal inadequacies, either real or imagined, is often excessive. For the depressive personality scale (historically called Scale 2 [D, Depression], 60 items with equal weight), an elevation indicates feelings of depression, sadness, pessimism, guilt, passivity, and a tendency to give up hope easily. The items of the anxious and depressive personality scales were reported verbatim elsewhere (20). We tested the internal consistency of the three scales in the current study using the Cronbach's coefficient alpha, and all three scales exhibited moderate to good values (pessimistic trait alpha = 0.95, anxious trait alpha = 0.89, depressive trait alpha = 0.72).

In addition to these three previously established scales, we also developed a new composite neuroticism score. This score is an equally weighted average of the pessimistic, anxious, and depressive scores, and identifies subjects with elevations on multiple neurotic trait scores (co-occurrence of pessimistic, anxious, and depressive personality traits). In particular, we averaged the three standardized scores (mean of 0, standard deviation of 1) within each person. We then further standardized this composite score to a mean of 0 and a standard deviation of 1 for comparability with the three independent trait scores.

Follow-up Procedures

The primary objective of follow-up in the Mayo Clinic Cohort Study of Personality and Aging was to detect incident cases of parkinsonism and dementia (14). We will report methods for tracking subjects to their address at the time of the study and for detecting parkinsonism or dementia separately. We describe here only the methods relevant to the assessment of mortality. All individuals eligible for inclusion were followed historically until death, the time of study contact (one contact for each cohort participant staggered over 2001 to 2006), or the time when they were lost to follow-up, using two methods.

First, we contacted subjects actively via telephone interview using a standardized questionnaire to assess their vital status and the presence of the diseases of interest. The telephone contact was direct whenever possible and conducted by one of six specifically trained research assistants. For deceased or incapacitated subjects (e.g., deaf, cognitively impaired, or terminally ill), we contacted a surrogate informant (proxy interview). Second, independent of the telephone contact, we followed all subjects passively through review of inpatient and outpatient medical records in the records-linkage system of the Rochester Epidemiology Project (for subjects residing in Olmsted County, MN) (25) and in the medical records archive of the Mayo Clinic (for subjects residing outside of Olmsted County, MN) (14). For each deceased subject, we obtained a copy of the death certificate from the records-linkage system, from the vital statistics offices of individual states, or from the National Death Index (NDI Plus; National Center for Health Statistics, Hyattsville, MD).

All study procedures were approved by the Institutional Review Board of the Mayo Clinic and of Olmsted Medical Center. In addition, all subjects who were examined as part of the study signed an informed consent form.

Primary Statistical Analyses

The primary statistical analyses made no *a priori* assumptions about the relationship between personality trait scores and mortality. We defined exposure for each personality trait using age-specific (by decades of age at the time of MMPI testing) and sex-specific

quartiles of the score distribution (14). Secondary analyses considered scores as continuous variables standardized to a mean of 0 and standard deviation of 1 to allow for comparison of results across scales with different numbers of items. This standardization was done separately by decades of age, and for men and women.

For all study subjects, we computed the time (in days) between the date of MMPI administration and death (or censoring). We censored surviving subjects at the time of study contact or at the time of last contact, and we obtained survival curves using the Kaplan-Meier method, and hazard ratios (HR) and 95% confidence intervals (CI) using Cox proportional hazards models. The assumption of proportional hazards was assessed by graphical methods and by introducing a time-dependent coefficient in the Cox models (26). The assumption was not violated in any of the analyses presented. Cox models used age as the time scale to allow for complete age-adjustment and included sex (whenever appropriate).

Analyses were conducted overall, separately for men and women, and in three broad strata based on age at the time of MMPI administration (ages 20 to 39, 40 to 49, 50 to 69 years). To formally test for a trend in the HRs across age at time of MMPI administration, we added an interaction term to the Cox models (age as a continuous variable by personality trait as a continuous variable).

To address the overlap of items across the three scales and the correlation across the personality traits measured by the scales, we fit several regression models including two or three scale scores simultaneously (as continuous variables). All analyses were completed using SAS[®] (SAS Institute, Cary, NC), and tests of statistical significance were conducted at the two-tailed alpha level of 0.05.

Analyses Addressing Confounding

To address possible confounding variables, we conducted a set of secondary analyses with adjustment for education as a marker of socioeconomic status (four classes: <9; 9 to 12; 13 to 16; >16 years of school), for alcohol use as a marker of lifestyle, and for self-assessed overall health. Information about alcohol use and self-assessed overall health was derived from two items in the MMPI (item 215 for excessive use of alcohol and item 51 for feelings of good health compared to peers). However, these analyses were considered secondary because they were restricted to the 4268 cohort members who provided education information at the time of an interview (1517 direct and 2751 proxy interviews).

Sensitivity Analyses

To exclude deaths not due to a natural cause or to a disease process, we performed a set of sensitivity analyses censoring deaths due to suicide, accidents, and homicide. To exclude a possible cause-effect inversion, we performed sensitivity analyses excluding the 256 subjects who died or were lost to follow-up within 5 years after the time of MMPI administration, and whose personality profiles may have been related to some severe diseases preceding the testing.

In addition, we investigated the association of anxious and pessimistic personality with mortality after excluding all subjects who scored in the top quartile of the depression scale at baseline. These analyses were conducted to eliminate the possible effect of depressive states in the measurement of pessimistic and anxious traits. Finally, because the MMPI scores at baseline may have been influenced by diseases or symptoms related to aging, we conducted additional sensitivity analyses that removed all MMPI items related to somatic complaints.

RESULTS

Study Sample

The baseline characteristics of the study cohort of 7216 subjects were reported in detail elsewhere (14). We were unable to follow 136 (1.9%) subjects beyond the date of the MMPI assessment. The remaining 7080 subjects (98.1%) were followed for a median of 32.4 years (range 19 days to 44.3 years). The vital status of subjects included was assessed via direct or proxy interview for 4422 subjects (62.5%). An additional 1176 subjects (16.6%) were contacted successfully either directly or by proxy via telephone or letter, but refused to participate in the active part of the study. This contact was inadequate to assess disease outcomes; however, it was sufficient to ascertain the vital status of subjects. In addition, by utilizing the records-linkage system of the Rochester Epidemiology Project, the medical records archive of the Mayo Clinic, and state-specific or national death indices, we were able to obtain vital status information for an additional 1482 subjects (20.9%). A total of 4634 (65.5%) of the cohort members died during follow-up.

Table 1 shows the baseline distribution of demographic characteristics, possible confounding variables, and personality traits in subjects who died compared with those who survived during the study. Subjects who died were older, were more often men, had used alcohol more often, had lower education, and reported less frequently to feel in good health compared with subjects who survived (crude comparisons unadjusted by age and sex). By design, our cohort included subjects with a broad range of ages at baseline (ages 20 to 69 years) and a similar number of men (3446, 48.7%) and women (3634, 51.3%).

We observed an important degree of pairwise correlation across the personality traits measured by the three scales (14). This is expected to a certain extent because the scales have some items in common that are scored in the same direction. Of the 60 items on the depressive trait scale, 13 (21.7%) overlap with the anxious trait scale and 15 (25%) with the pessimistic trait scale. Of the 48 items on the anxious trait scale, 13 (27.1%) overlap with the depressive trait scale and 32 (66.7%) with the pessimistic trait scale. Of the 192 items on the pessimistic trait scale, 15 (7.8%) overlap with the depressive trait scale and 32 (16.7%) with the anxious trait scale. Using the raw scores (not standardized, and not adjusted by age and sex), we observed the highest Spearman rank correlation for anxious trait and pessimistic trait ($\rho = 0.90$), followed by anxious trait and depressive trait ($\rho = 0.60$), and by depressive trait and pessimistic trait ($\rho = 0.50$). Significant correlations remained even after excluding overlapping items. We reported our analyses for scale overlap and scale correlation more extensively elsewhere (14).

Personality Scales and Mortality

Table 2 shows the results of survival analyses across the three personality scales of interest and for the composite neuroticism score, separately for men and women, and overall. We found an increased risk of death for subjects with elevated scores on the scales for pessimistic, anxious, and depressive personality traits considered separately, and for subjects with elevated composite neuroticism scores (top quartile compared with lower quartile; Figure 1). In addition, we observed a linear trend of increasing risk from quartile 1 through quartile 4 for pessimistic (trend $p < .001$), anxious (trend $p < .001$), and depressive traits (trend $p < .001$), and for the composite neuroticism score (trend $p < .001$). Analyses adjusted for education, alcohol use, and self-assessed general health yielded smaller HRs, and the analysis for depressive trait no longer reached statistical significance (Table 2, footnotes c, d, e, and f).

Analyses considering the personality trait scores as continuous variables yielded consistent results. In these analyses, the HR represents the change in risk of death for an increase of

one standard deviation in the personality scale (assuming a linear effect; Table 2). Table 3 shows multivariable regression models including personality trait scores one at a time, two at a time, and all three simultaneously. Models including two personality traits at a time suggested that the effect of the anxious trait was largely explained by the pessimistic trait. This was confirmed in a regression model including all three trait scores simultaneously and sex (Table 3).

Sensitivity Analyses

For the pessimistic trait, the HR was 1.32 (95% CI 1.21–1.43, $p < .001$) in the primary analysis and remained consistent in sensitivity analyses removing the 256 subjects with less than 5 years of follow-up (HR = 1.31, 95% CI 1.20–1.42, $p < .001$), censoring the 105 deaths of un-natural cause (homicide, suicide, and accidental; HR = 1.31, 95% CI 1.20–1.42, $p < .001$), and excluding all subjects who scored in the top quartile of the depression scale at baseline (HR = 1.24, 95% CI 1.13–1.37, $p < .001$). For the anxious trait, the HR was 1.30 (95% CI 1.20–1.42, $p < .001$) in the primary analysis and remained consistent in sensitivity analyses removing the 256 subjects with less than 5 years of follow-up (HR = 1.30, 95% CI 1.19–1.41, $p < .001$), censoring the 105 deaths of un-natural cause (HR = 1.30, 95% CI 1.20–1.41, $p < .001$), and excluding all subjects who scored in the top quartile of the depression scale at baseline (HR = 1.22, 95% CI 1.09–1.35, $p < .001$).

For the depressive trait, the HR was 1.36 (95% CI 1.25–1.47, $p < .001$) in the primary analysis and remained consistent in sensitivity analyses removing the 256 subjects with less than 5 years of follow-up (HR = 1.33, 95% CI 1.22–1.44, $p < .001$), and censoring the 105 deaths of un-natural cause (HR = 1.36, 95% CI 1.25–1.48, $p < .001$). For the composite neuroticism score, the HR was 1.42 (95% CI 1.31–1.54, $p < .001$) in the primary analysis and remained consistent in sensitivity analyses removing the 256 subjects with less than 5 years of follow-up (HR = 1.41, 95% CI 1.30–1.53, $p < .001$), and censoring the 105 deaths of un-natural cause (HR = 1.42, 95% CI 1.31–1.54, $p < .001$). Finally, results did not vary for any of the three scales in sensitivity analyses repeated after removing all MMPI items related to somatic complaints (data not shown).

Analyses Stratified by Age at Time of Testing

Table 4 shows the results of analyses stratified by age at the time of MMPI administration. A pessimistic personality trait measured early in life (ages 20 to 39 years) was a particularly strong predictor of increased mortality (HR = 1.86, 95% CI 1.46–2.38, $p < .001$), and a formal test of interaction between age at MMPI administration and pessimistic personality was significant ($p = .02$). By contrast, the anxious trait and the depressive trait were consistently associated with increased mortality regardless of the age at time of personality testing (Table 4, footnote a).

DISCUSSION

Comparison with Previous Studies

We found that pessimistic, anxious, and depressive personality traits considered separately or combined into a composite neuroticism score were associated with an increased risk of death in men and women. Our findings were not due to a cause-effect inversion because the associations with increased mortality remained similar even when personality traits were measured at younger ages.

Table 5 summarizes studies on the association between neuroticism-related personality traits and mortality published since 2000. Our findings are consistent with findings from several previous studies (2–11); however, they are discrepant with the findings from two additional

studies (13,27). The first discrepant study showed that older frail subjects with high neuroticism and agreeableness scores experienced reduced mortality. The sample included 1076 subjects age 65 years or older (mean age 79.6 years), the follow-up was approximately 5 years, and personality was measured using the NEO Five-Factor Inventory (12,28). A trend toward reduced mortality was also observed for neuroticism in an additional set of analyses from the same study using the Revised NEO Personality Inventory (NEO-PI-R) (12). A second discrepant study from Japan showed no association between neuroticism and death in 1228 men and women. The mean follow-up was 5 years (13). Visual inspection of the plots obtained in our study suggests that the curves for low and high scores diverge appreciably only after 10 to 15 years of follow-up (quartiles 4 vs. 1 in Figure 1). These patterns suggest that studies with short follow-up and small sample size are unlikely to detect a difference in survival.

Possible Mechanisms of Causality

The mechanisms underlying the associations between pessimistic, anxious, and depressive personality traits and mortality remain uncertain and are probably complex (1,8). The association may be non-causal and due to confounding (Figure 2, panel A). Thus, a set of genetic variants, events in early life, or a combination of both genetic and early life factors could influence the development of personality and, independently, could cause increased morbidity and mortality later in life (29). This hypothesis is consistent with the recent emphasis on the possible developmental origin of health and diseases and with the possible involvement of epigenetic processes (30). Under this hypothesis, preventive interventions targeting personality traits may be ineffective to improve health and to increase survival. Whether confounding explains part of the observed associations cannot be confirmed at this time (31,32); however, we propose three other mechanisms in which personality traits may be either directly or indirectly causal (Figure 2, panels B, C, and D).

Under the first proposed causal mechanism, personality traits may lead to increased morbidity and mortality later in life through some direct biological mechanism. For example, these personality traits could influence neurological, endocrinological, or immunological pathways and trigger disease processes leading to functional impairments or lesions in specific tissues or organs (Figure 2, panel B). Indeed, stress and depression have been linked to hippocampal atrophy and other brain lesions (33,34). Similarly, stress and depression have been linked to increased heart rate, hypertension, increased plasma norepinephrine levels, or changes in blood coagulation that may increase the risk of cardiovascular disease (35–38). Stress has also been implicated in lower immune system responsiveness and in higher risk of cancer (39–41). Under this hypothesis, the personality traits are directly causal, and interventions aimed at modifying them would be preventive (e.g., cognitive-behavioral treatments or pharmacological treatments) (42).

Under the second proposed causal mechanism, personality traits may lead to poor self-care or to unhealthy behaviors such as inadequate diet, excessive use of substances, excessive use of medications, lack of physical activity, or poor living conditions. These risk factors may, in turn, trigger disease processes leading to functional impairments or lesions in specific tissues or organs (Figure 2, panel C) (43). Under this hypothesis, preventive interventions could target either the personality traits directly (42), or the intervening behavioral risk factors.

Under a third causal mechanism, personality traits may modify the prognosis of diseases after their onset. After the symptoms of diseases have appeared, negative personality traits may cause underutilization of health care facilities and low compliance with treatment regimens (Figure 2, panel D) (1). Under this hypothesis, preventive interventions could target the management and care of subjects with particular diseases to prolong survival.

Unfortunately, the findings from our study are insufficient to determine which one of the three causal mechanisms is most important. However, it is likely that all three mechanisms may be involved in combination. Additional research is needed to address these important hypotheses because they have major implications for disease prevention and health promotion.

Strengths of the Study

Our study has several strengths. First, the size of our cohort was large and the median length of follow-up was 32 years. This long follow-up and the large number of deaths provided adequate statistical power to investigate strata by sex and by age at time of MMPI administration. A study of this magnitude was only possible because we used a previously established cohort with extensive personality testing.

Second, the MMPI was administered as part of a research project and not because of any specific medical indication. This reduced the possible confounding effect of pre-existing conditions. However, details about the specific medical conditions or symptoms that brought the participants to the Mayo Clinic are not available. Sensitivity analyses that excluded subjects who died or were lost to follow-up within the initial 5 years after the time of MMPI administration yielded consistent results.

Third, by restricting the cohort to those subjects residing within the 120-mile radius, and by using the records-linkage system of the Rochester Epidemiology Project, the medical records archive of the Mayo Clinic, and state-specific and national death indices, we were able to obtain vital status information for almost all original cohort members. Only 1.9% of subjects were not followed beyond the initial MMPI assessment.

Limitations of the Study

The study also has weaknesses. First, the sample was not recruited from a well-defined population through randomization; therefore, it was a convenience sample (14,44). On the other hand, efforts were made to recruit patients referred to the Mayo Clinic sequentially, and to avoid the possible confounding effect of the reason for testing. Patients tested because of psychiatric indications were excluded, and sensitivity analyses excluding subjects who died or were lost to follow-up within the initial 5 years after MMPI testing yielded consistent results.

Second, we observed fewer deaths in our cohort than were expected using age-, sex-, and calendar year-specific Minnesota life table values (standardized mortality ratio [SMR]: 0.91, 95% CI 0.88–0.93, $p < .001$). This reduced mortality may indicate a survival benefit for persons who had access to care at the Mayo Clinic or for subjects who accepted to participate in a research study (volunteer bias) (44). Alternatively, our capture methods to ascertain death may have been incomplete. However, the interpretation of our study findings for mortality remains valid because the ascertainment of death should be similar across strata defined by the personality scores at baseline (non-differential underascertainment).

Third, personality scales have changed and will continue to change over time. The MMPI has a long history and is familiar to both clinicians and researchers (14,15). However, the MMPI is a relatively old instrument, is long and time-consuming, only permits true or false answers, and was not specifically designed to assess normal personality variation. New instruments based on recent personality theories have achieved recognition both in clinical practice and in research, e.g., the Revised NEO Personality Inventory (NEO-PI-R) (45). On the other hand, the value of our cohort is the long time-span between personality testing and the assessment of outcomes. It is impossible to simultaneously have a contemporary personality instrument and long-term follow-up. In addition, it has been shown that

performance on the MMPI correlates well with contemporary instruments such as the NEO-PI scale (46).

CONCLUSIONS

Our findings suggest that pessimistic, anxious, and depressive personality traits are associated with an increased risk of all-cause mortality even when they are measured early in life. The findings are similar for men and women. Further research is needed to confirm that the associations observed are either directly or indirectly causal. If the causal role of neuroticism-related traits is confirmed, our findings may prompt interventions to improve health and increase survival.

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Acronyms

CES-D	Center for Epidemiologic Studies – Depression
CI	confidence interval
EPI	Eysenck Personality Inventory
HADS	Hospital Anxiety and Depression Scale
HR	hazard ratio
IQR	interquartile range
MMPI	Minnesota Multiphasic Personality Inventory
NEO-FFI	NEO Five-Factor Inventory
NEO-PI-R	Revised NEO Personality Inventory
SD	standard deviation
SMR	standardized mortality ratio

References

1. Cloninger CR. How does personality influence mortality in the elderly? *Psychosom Med.* 2005; 67:839–40. [PubMed: 16314586]
2. 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. [PubMed: 17991814]
3. Mykletun A, Bjerkeset O, Dewey M, Prince M, Overland S, Stewart R. Anxiety, depression, and cause-specific mortality: the HUNT study. *Psychosom Med.* 2007; 69:323–31. [PubMed: 17470669]
4. Brummett BH, Helms MJ, Dahlstrom WG, Siegler IC. Prediction of all-cause mortality by the Minnesota Multiphasic Personality Inventory Optimism-Pessimism Scale scores: study of a college sample during a 40-year follow-up period. *Mayo Clin Proc.* 2006; 81:1541–4. [PubMed: 17165632]
5. Ostir GV, Goodwin JS. High anxiety is associated with an increased risk of death in an older tri-ethnic population. *J Clin Epidemiol.* 2006; 59:534–40. [PubMed: 16632143]
6. Wilson RS, Krueger KR, Gu L, Bienias JL, Mendes de Leon CF, Evans DA. Neuroticism, extraversion, and mortality in a defined population of older persons. *Psychosom Med.* 2005; 67:841–5. [PubMed: 16314587]

7. Wilson RS, Mendes de Leon CF, Bienias JL, Evans DA, Bennett DA. Personality and mortality in old age. *J Gerontol B Psychol Sci Soc Sci.* 2004; 59:P110–6. [PubMed: 15118013]
8. Wilson RS, Bienias JL, Mendes de Leon CF, Evans DA, Bennett DA. Negative affect and mortality in older persons. *Am J Epidemiol.* 2003; 158:827–35. [PubMed: 14585760]
9. McCarron P, Gunnell D, Harrison GL, Okasha M, Davey Smith G. Temperament in young adulthood and later mortality: prospective observational study. *J Epidemiol Community Health.* 2003; 57:888–92. [PubMed: 14600116]
10. 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. [PubMed: 12090673]
11. Maruta T, Colligan RC, Malinchoc M, Offord KP. Optimists vs pessimists: survival rate among medical patients over a 30-year period. *Mayo Clin Proc.* 2000; 75:140–3. [PubMed: 10683651]
12. Weiss A, Costa PT Jr. Domain and facet personality predictors of all-cause mortality among Medicare patients aged 65 to 100. *Psychosom Med.* 2005; 67:724–33. [PubMed: 16204430]
13. 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. [PubMed: 18403571]
14. Rocca WA, Grossardt BR, Peterson BJ, Bower JH, Trenerry MR, Ahlskog JE, Sanft KR, de Andrade M, Maraganore DM. The Mayo Clinic Cohort Study of Personality and Aging: design and sampling, reliability and validity of instruments, and baseline description. *Neuroepidemiology.* 2006; 26:119–29. [PubMed: 16424675]
15. Swenson, WM.; Pearson, JS.; Osborne, D. An MMPI Source Book: Basic Item, Scale, and Pattern Data on 50,000 Medical Patients. Minneapolis: University of Minnesota Press; 1973.
16. Maruta T, Hamburg ME, Jennings CA, Offord KP, Colligan RC, Frye RL, Malinchoc M. Keeping hostility in perspective: coronary heart disease and the Hostility Scale on the Minnesota Multiphasic Personality Inventory. *Mayo Clin Proc.* 1993; 68:109–14. [PubMed: 8423689]
17. Malinchoc M, Rocca WA, Colligan RC, Offord KP, Kokmen E. Premorbid personality characteristics in Alzheimer's disease: an exploratory case-control study. *Eur J Neurol.* 1997; 4:227–30.
18. Maruta T, Colligan RC, Malinchoc M, Offord KP. Optimism-pessimism assessed in the 1960s and self-reported health status 30 years later. *Mayo Clin Proc.* 2002; 77:748–53. [PubMed: 12173709]
19. Hathaway, SR.; McKinley, JC. The Minnesota Multiphasic Personality Inventory. Minneapolis: The University of Minnesota Press; 1943. Rev
20. Dahlstrom, WG.; Welsh, GS.; Dahlstrom, LE. An MMPI Handbook. Minneapolis: University of Minnesota Press; 1972. Rev
21. Peterson C, Seligman ME, Vaillant GE. Pessimistic explanatory style is a risk factor for physical illness: a thirty-five-year longitudinal study. *J Pers Soc Psychol.* 1988; 55:23–7. [PubMed: 3418489]
22. Seligman, MEP. Learned Optimism. New York: A.A. Knopf; 1991.
23. Colligan RC, Offord KP, Malinchoc M, Schulman P, Seligman ME. CAVEing the MMPI for an Optimism-Pessimism Scale: Seligman's attributional model and the assessment of explanatory style. *J Clin Psychol.* 1994; 50:71–95. [PubMed: 8150997]
24. Hermann BP, Trenerry MR, Colligan RC. Learned helplessness, attributional style, and depression in epilepsy. Bozeman Epilepsy Surgery Consortium. *Epilepsia.* 1996; 37:680–6. [PubMed: 8681901]
25. Melton LJ 3rd. History of the Rochester Epidemiology Project. *Mayo Clin Proc.* 1996; 71:266–74. [PubMed: 8594285]
26. Therneau, TM.; Grambsch, PM. Modeling Survival Data: Extending the Cox Model. New York, NY: Springer; 2000.
27. Weiss A, Costa PT Jr, Karuza J, Duberstein PR, Friedman B, McCrae RR. Cross-sectional age differences in personality among Medicare patients aged 65 to 100. *Psychol Aging.* 2005; 20:182–5. [PubMed: 15769223]

28. Costa, PT.; McCrae, RR. Psychological Assessment Resources Inc. Revised NEO Personality Inventory (NEO PI-R) and NEO Five-Factor Inventory (NEO-FFI). Odessa, Fla. (P.O. Box 998, Odessa 33556): Psychological Assessment Resources; 1992.
29. Keltikangas-Jarvinen L, Raikkonen K, Ekelund J, Peltonen L. Nature and nurture in novelty seeking. *Mol Psychiatry*. 2004; 9:308–11. [PubMed: 14569271]
30. Hanson MA, Gluckman PD. Developmental origins of health and disease: new insights. *Basic Clin Pharmacol Toxicol*. 2008; 102:90–3. [PubMed: 18226060]
31. McCaffery JM, Frasure-Smith N, Dube MP, Theroux P, Rouleau GA, Duan Q, Lesperance F. Common genetic vulnerability to depressive symptoms and coronary artery disease: a review and development of candidate genes related to inflammation and serotonin. *Psychosom Med*. 2006; 68:187–200. [PubMed: 16554382]
32. Su S, Miller AH, Snieder H, Bremner JD, Ritchie J, Maisano C, Jones L, Murrah NV, Goldberg J, Vaccarino V. Common genetic contributions to depressive symptoms and inflammatory markers in middle-aged men: The Twins Heart Study. *Psychosom Med*. 2008 in press.
33. Sapolsky RM. Depression, antidepressants, and the shrinking hippocampus. *Proc Natl Acad Sci U S A*. 2001; 98:12320–2. [PubMed: 11675480]
34. Sheline YI, Wang PW, Gado MH, Csernansky JG, Vannier MW. Hippocampal atrophy in recurrent major depression. *Proc Natl Acad Sci U S A*. 1996; 93:3908–13. [PubMed: 8632988]
35. Carney RM, Freedland KE, Rich MW, Jaffe AS. Depression as a risk factor for cardiac events in established coronary heart disease: A review of possible mechanisms. *Ann Behav Med*. 1995; 17:142–9. [PubMed: 18425665]
36. Carney RM, Freedland KE, Veith RC, Cryer PE, Skala JA, Lynch T, Jaffe AS. Major depression, heart rate, and plasma norepinephrine in patients with coronary heart disease. *Biol Psychiatry*. 1999; 45:458–63. [PubMed: 10071718]
37. Musselman DL, Evans DL, Nemeroff CB. The relationship of depression to cardiovascular disease: epidemiology, biology, and treatment. *Arch Gen Psychiatry*. 1998; 55:580–92. [PubMed: 9672048]
38. von Kanel R, Mills PJ, Fainman C, Dimsdale JE. Effects of psychological stress and psychiatric disorders on blood coagulation and fibrinolysis: a biobehavioral pathway to coronary artery disease? *Psychosom Med*. 2001; 63:531–44. [PubMed: 11485106]
39. Levenson JL, Bemis C. The role of psychological factors in cancer onset and progression. *Psychosomatics*. 1991; 32:124–32. [PubMed: 2027934]
40. Laudenslager ML, Ryan SM, Drugan RC, Hyson RL, Maier SF. Coping and immunosuppression: inescapable but not escapable shock suppresses lymphocyte proliferation. *Science*. 1983; 221:568–70. [PubMed: 6603018]
41. Visintainer MA, Volpicelli JR, Seligman ME. Tumor rejection in rats after inescapable or escapable shock. *Science*. 1982; 216:437–9. [PubMed: 7200261]
42. Dozois, DJA.; Dobson, KS. The Prevention of Anxiety and Depression: Theory, Research, and Practice. 1. Washington, DC: American Psychological Association; 2004.
43. Everson SA, Kauhanen J, Kaplan GA, Goldberg DE, Julkunen J, Tuomilehto J, Salonen JT. Hostility and increased risk of mortality and acute myocardial infarction: the mediating role of behavioral risk factors. *Am J Epidemiol*. 1997; 146:142–52. [PubMed: 9230776]
44. Sackett DL. Bias in analytic research. *J Chronic Dis*. 1979; 32:51–63. [PubMed: 447779]
45. Costa PT Jr, McCrae RR. Normal personality assessment in clinical practice: The NEO Personality Inventory. *Psychol Assess*. 1992; 4:5–13.
46. McCrae RR. The five-factor model and its assessment in clinical settings. *J Pers Assess*. 1991; 57:399. [PubMed: 1757868]

Figure 1. Survival among subjects in the top quartile (thick line) and bottom quartile (thin line) of MMPI scales for pessimistic, anxious, and depressive personality traits and for the composite neuroticism score.

Figure 2.

Four hypothetical mechanisms linking personality traits with increased overall mortality. A. Genetic variants or early life risk factors influence personality traits and, independently, cause increased mortality later in life (association due to confounding). B. Personality traits cause direct neurological, endocrinological, or immunological effects leading to functional impairments or lesions in specific tissues or organs. C. Personality traits lead to poor self-care or to unhealthy behaviors causing, in turn, functional impairments or lesions in specific tissues or organs. D. Personality traits may cause underutilization of health care facilities and low compliance with treatment after the onset of diseases, leading to poor prognosis. Mechanisms B, C, and D may be involved in combination and may be amenable to behavioral or pharmacological intervention.

TABLE 1
Comparison of Baseline Characteristics in Subjects Who Died Versus Subjects Who Survived During the Study

Characteristic	Total cohort <i>n</i> = 7080 median (IQR) ^a	Subjects who died <i>n</i> = 4634 median (IQR) ^a	Subjects who survived <i>n</i> = 2446 median (IQR) ^a	<i>p</i> value ^b
Age at MMPI testing, in years	48.1 (38.2–57.3)	53.9 (46.4–60.6)	37.0 (29.5–43.3)	<.001
Men, <i>n</i> (%)	3446 (48.7%)	2441 (52.7%)	1005 (41.1%)	<.001
Alcohol use, <i>n</i> (%) ^c	1026 (14.5%)	732 (15.8%)	294 (12.0%)	<.001
Good general health, <i>n</i> (%) ^d	4957 (70.0%)	3139 (67.7%)	1818 (74.3%)	<.001
Education, in years ^e	12 (12–15)	12 (8–14)	13 (12–16)	<.001
Pessimistic trait score	578.5 (369.0–830.5)	595.7 (382.3–845.1)	539.0 (349.2–786.4)	<.001
Quartile 4, <i>n</i> (%)	1763 (24.9%)	1210 (26.1%)	553 (22.6%)	0.001
Anxious trait score	10 (6–16)	10 (6–16)	10 (6–16)	0.84
Quartile 4, <i>n</i> (%)	1625 (23.0%)	1118 (24.1%)	507 (20.7%)	0.001
Depressive trait score	22 (18–26)	22 (19–26)	21 (18–25)	<.001
Quartile 4, <i>n</i> (%)	1538 (21.7%)	1049 (22.6%)	489 (20.0%)	0.01
Composite neuroticism score	−0.20 (−0.74–0.55)	−0.14 (−0.72–0.64)	−0.27 (−0.78–0.42)	<.001
Quartile 4, <i>n</i> (%)	1767 (25.0%)	1244 (26.9%)	523 (21.4%)	<.001

^aIQR = interquartile (or 25th to 75th percentile) range.

^bTest comparing subjects who died with subjects who survived.

^cSubjects responding positively to the MMPI item “I have used alcohol excessively” (item 215).

^dSelf-assessed general health. Subjects responding positively to the MMPI item “I am in just as good health as most of my friends” (item 51).

^eEducation expressed as years of schooling was restricted to 4268 subjects who participated in a direct or proxy interview.

TABLE 2
Survival Analyses Across Quartiles of Scores for Three Personality Traits and for the Composite Neuroticism Score in Men and Women

Personality trait	Women				Men				Both men and women ^a			
	No. of subjects at risk	No. of deaths	HR (95% confidence interval)	p value	No. of subjects at risk	No. of deaths	HR (95% confidence interval)	p value	No. of subjects at risk	No. of deaths	HR (95% confidence interval)	p value
Pessimistic												
Quartile 1	912	528	1.00 (ref.)	---	865	583	1.00 (ref.)	---	1777	1111	1.00 (ref.)	---
Quartile 2	905	549	1.23 (1.09, 1.39)	<.001	864	577	1.01 (0.90, 1.13)	.87	1769	1126	1.10 (1.02, 1.20)	.02
Quartile 3	910	552	1.16 (1.03, 1.31)	.02	861	635	1.16 (1.04, 1.30)	.01	1771	1187	1.15 (1.06, 1.25)	<.001
Quartile 4	907	564	1.29 (1.14, 1.45)	<.001	856	646	1.34 (1.20, 1.50)	<.001	1763	1210	1.32 (1.21, 1.43) ^c	<.001
Continuous ^b	3634	2193	1.11 (1.06, 1.15)	<.001	3446	2441	1.13 (1.09, 1.18)	<.001	7080	4634	1.12 (1.09, 1.15)	<.001
Anxious												
Quartile 1	938	542	1.00 (ref.)	---	971	660	1.00 (ref.)	---	1909	1202	1.00 (ref.)	---
Quartile 2	923	556	1.10 (0.98, 1.24)	.10	867	598	1.00 (0.90, 1.12)	.99	1790	1154	1.05 (0.96, 1.13)	.28
Quartile 3	949	574	1.13 (1.00, 1.27)	.04	807	586	1.08 (0.97, 1.21)	.18	1756	1160	1.10 (1.01, 1.19)	.02
Quartile 4	824	521	1.31 (1.16, 1.48)	<.001	801	597	1.30 (1.16, 1.45)	<.001	1625	1118	1.30 (1.20, 1.42) ^d	<.001
Continuous ^b	3634	2193	1.10 (1.06, 1.15)	<.001	3446	2441	1.11 (1.07, 1.15)	<.001	7080	4634	1.11 (1.08, 1.14)	<.001
Depressive												
Quartile 1	1031	605	1.00 (ref.)	---	1052	711	1.00 (ref.)	---	2083	1316	1.00 (ref.)	---
Quartile 2	882	514	0.96 (0.85, 1.08)	.45	799	563	1.07 (0.95, 1.19)	.26	1681	1077	1.02 (0.94, 1.10)	.68
Quartile 3	920	574	1.10 (0.98, 1.24)	.09	858	618	1.16 (1.04, 1.30)	.006	1778	1192	1.14 (1.05, 1.23)	.001
Quartile 4	801	500	1.32 (1.17, 1.48)	<.001	737	549	1.39 (1.24, 1.55)	<.001	1538	1049	1.36 (1.25, 1.47) ^e	<.001
Continuous ^b	3634	2193	1.11 (1.07, 1.16)	<.001	3446	2441	1.14 (1.10, 1.19)	<.001	7080	4634	1.13 (1.10, 1.16)	<.001
Composite neuroticism score												
Quartile 1	910	535	1.00 (ref.)	---	864	581	1.00 (ref.)	---	1774	1116	1.00 (ref.)	---
Quartile 2	908	534	1.17 (1.04, 1.32)	.01	860	590	1.08 (0.96, 1.21)	.21	1768	1124	1.11 (1.03, 1.21)	.01
Quartile 3	909	540	1.11 (0.98, 1.25)	.10	862	610	1.20 (1.07, 1.34)	.002	1771	1150	1.16 (1.07, 1.26)	<.001
Quartile 4	907	584	1.40 (1.25, 1.58)	<.001	860	660	1.44 (1.29, 1.61)	<.001	1767	1244	1.42 (1.31, 1.54) ^f	<.001
Continuous ^b	3634	2193	1.12 (1.08, 1.17)	<.001	3446	2441	1.15 (1.10, 1.19)	<.001	7080	4634	1.13 (1.10, 1.17)	<.001

^a Analyses for men and women combined were adjusted for sex.

^b Scores were treated as continuous variables and standardized to a mean of 0 and standard deviation of 1 to allow for comparison of results across scales with different numbers of items. This standardization was done separately by decades of age, and for men and women. The HR represents the change in risk of death for an increase of one standard deviation in the personality scale.

^c Results were consistent in secondary analyses adjusted for education, alcohol use, and self-assessed general health (HR = 1.17, 95% CI 1.04–1.30, $p = .007$).

^d Results were consistent in secondary analyses adjusted for education, alcohol use, and self-assessed general health (HR = 1.12, 95% CI 1.01–1.25, $p = .04$).

^e Results were consistent, but no longer statistically significant, in secondary analyses adjusted for education, alcohol use, and self-assessed general health (HR = 1.08, 95% CI 0.96–1.22, $p = .19$).

^f Results were consistent in secondary analyses adjusted for education, alcohol use, and self-assessed general health (HR = 1.19, 95% CI 1.06–1.33, $p = .003$).

TABLE 3

Multivariable Regression Models Using Continuous Variables

Model	Pessimistic trait		Anxious trait		Depressive trait	
	HR (95% confidence interval) ^a	p value	HR (95% confidence interval) ^a	p value	HR (95% confidence interval) ^a	p value
Pessimistic only	1.12 (1.09, 1.15)	<.001				
Anxious only	---	---	1.11 (1.08, 1.14)	<.001		
Depressive only	---	---			1.13 (1.10, 1.16)	<.001
Pessimistic and anxious	1.12 (1.05, 1.20)	<.001	1.00 (0.93, 1.07)	0.91		
Pessimistic and depressive	1.07 (1.04, 1.11)	<.001	---	---	1.09 (1.06, 1.13)	<.001
Anxious and depressive	---	---	1.04 (1.01, 1.08)	.02	1.10 (1.06, 1.14)	<.001
Full model ^b	1.18 (1.10, 1.26)	<.001	0.89 (0.82, 0.96)	.003	1.12 (1.08, 1.17)	<.001
Adjusted full model ^c	1.14 (1.04, 1.25)	.005	0.90 (0.82, 1.00)	.05	1.04 (0.99, 1.10)	.14

^a All models were adjusted by sex. The HR represents the change in risk of death for an increase of one standard deviation in the personality trait score.^b The full model included all three standardized personality trait scores simultaneously.^c The adjusted full model included all three standardized personality trait scores, education, alcohol use, and self-assessed general health.

TABLE 4
Survival Analyses Across Quartiles of Scores for Three Personality Traits Stratified by Age at the Time of Testing^a

Personality trait	Aged 20 to 39 years at testing				Aged 40 to 49 years at testing				Aged 50 to 69 years at testing			
	No. of subjects at risk	No. of deaths	HR (95% confidence interval)	p value	No. of subjects at risk	No. of deaths	HR (95% confidence interval)	p value	No. of subjects at risk	No. of deaths	HR (95% confidence interval)	p value
Pessimistic												
Quartile 1	519	104	1.00 (ref.)	---	453	256	1.00 (ref.)	---	805	751	1.00 (ref.)	---
Quartile 2	515	120	1.24 (0.95, 1.61)	.11	457	265	1.02 (0.86, 1.21)	.81	797	741	1.11 (1.00, 1.23)	.045
Quartile 3	519	154	1.60 (1.25, 2.06)	<.001	455	282	1.12 (0.95, 1.33)	.18	797	751	1.09 (0.99, 1.21)	.09
Quartile 4	513	166	1.86 (1.46, 2.38)	<.001	457	297	1.28 (1.08, 1.51)	.004	793	747	1.24 (1.12, 1.37)	<.001
Continuous ^b	2066	544	1.22 (1.13, 1.32)	<.001	1822	1100	1.12 (1.06, 1.19)	<.001	3192	2990	1.09 (1.06, 1.14)	<.001
Anxious												
Quartile 1	573	131	1.00 (ref.)	---	495	284	1.00 (ref.)	---	841	787	1.00 (ref.)	---
Quartile 2	505	122	1.07 (0.84, 1.37)	.60	474	278	1.03 (0.87, 1.22)	.73	811	754	1.04 (0.94, 1.15)	.46
Quartile 3	524	147	1.29 (1.02, 1.64)	.03	408	248	1.07 (0.90, 1.27)	.44	824	765	1.07 (0.97, 1.18)	.21
Quartile 4	464	144	1.49 (1.17, 1.89)	.001	445	290	1.29 (1.09, 1.52)	.003	716	684	1.27 (1.15, 1.41)	<.001
Continuous ^b	2066	544	1.14 (1.05, 1.23)	.002	1822	1100	1.11 (1.05, 1.18)	<.001	3192	2990	1.10 (1.06, 1.14)	<.001
Depressive												
Quartile 1	624	144	1.00 (ref.)	---	519	296	1.00 (ref.)	---	940	876	1.00 (ref.)	---
Quartile 2	493	123	1.12 (0.88, 1.42)	.37	419	255	1.04 (0.88, 1.23)	.67	769	699	1.00 (0.90, 1.10)	.98
Quartile 3	485	137	1.29 (1.02, 1.64)	.03	481	282	1.06 (0.90, 1.25)	.50	812	773	1.14 (1.03, 1.25)	.01
Quartile 4	464	140	1.42 (1.13, 1.80)	.003	403	267	1.24 (1.05, 1.46)	.01	671	642	1.39 (1.25, 1.54)	<.001
Continuous ^b	2066	544	1.13 (1.04, 1.23)	.003	1822	1100	1.10 (1.03, 1.16)	.003	3192	2990	1.14 (1.10, 1.18)	<.001

^a All analyses were adjusted by sex. A formal test of interaction between age at MMPI administration and personality (both used as continuous variables) was significant for the pessimistic trait ($p = .02$); however, tests were not significant for the anxious trait ($p = .48$) and for the depressive trait ($p = .41$).

^b Scores were treated as continuous variables and standardized to a mean of 0 and standard deviation of 1 to allow for comparison of results across scales with different numbers of items. This standardization was done separately by decades of age, and for men and women. The HR represents the change in risk of death for an increase of one standard deviation in the personality scale.

TABLE 5
Published Studies on the Association Between Neuroticism-Related Personality Traits and Overall Mortality

Study	Setting	Size of study	Men, %	Ages, in years mean (range)	Years of follow-up mean	Personality instrument	Main results and comments
Studies supporting an association							
Maruta et al., 2000 (11)	Olmsted County, MN, USA	839	36.9	35 ^a (15–84)	29	MMPI	Pessimism was associated with increased mortality.
Christensen et al., 2002 (10)	Iowa, USA	174	57.5	54.4 (SD 16.0)	4.1	NEO-FFI	Neuroticism was associated with increased mortality
Wilson et al., 2003 (8)	Religious Orders Study, USA	851	31.8	75.4 (SD 6.9)	4.7	CES-D, anxiety trait scale	Depression and anxiety were associated with increased mortality.
McCarron et al., 2003 (9)	Glasgow, Scotland	9239	100	20.5 (16–30)	40.9 ^a	Physician assessment	Depression and anxiety were associated with increased mortality.
Wilson et al., 2004 (7)	Religious Orders Study, USA	883	30.9	75.1 (SD 7.1)	5.1	NEO-FFI	Neuroticism was associated with increased mortality.
Wilson et al., 2005 (6)	Chicago, IL, USA	6158	39.3	75.0 (SD 7.2)	6.2	NEO-FFI	Neuroticism was associated with increased mortality.
Brummett et al., 2006 (4)	North Carolina, USA	6958	82.6	18.5 (SD 2.6)	40	MMPI	Pessimism was associated with increased mortality.
Ostir and Goodwin, 2006 (5)	Texas, USA	506	49.2	80.8 (75–100)	5	Zung anxiety scale	Anxiety was associated with increased mortality.
Shipley et al., 2007 (2)	United Kingdom	5424	55.1	44.7 (18–94)	21	EPI	Neuroticism was associated with increased mortality.
Mykletun et al., 2007 (3)	Norway	61349	49.6 ^b	48 (19–101)	4.4	HADS	Depression was associated with increased mortality, anxiety was not.
Current study	Region surrounding Rochester, MN, USA	7080	48.7	48.1 ^a (20–69)	32.4 ^a	MMPI	Pessimism, anxiety, and depression were associated with increased mortality.
Studies not supporting an association							
Weiss and Costa, 2005 (12)	Eastern USA	1076	27.1	79.6 (65–100)	5	NEO-FFI and NEO-PI-R ^c	Neuroticism was associated with decreased mortality.
Iwasa et al., 2008 (13)	Tokyo, Japan	1228	39.6	72.4 (≥65)	5	NEO-FFI	Neuroticism was not associated with mortality.

CES-D = Center for Epidemiologic Studies – Depression; EPI = Eysenck Personality Inventory; HADS = Hospital Anxiety and Depression Scale; MMPI = Minnesota Multiphasic Personality Inventory; NEO-FFI = NEO Five-Factor Inventory; NEO-PI-R = Revised NEO Personality Inventory; SD = standard deviation

^aMedian

^b Based on the overall population of Norway. Counts separate for men and women were not reported in the paper.

^c An additional set of analyses from the same study used the NEO-PI-R.