Published in final edited form as:

J Res Pers. 2017 October ; 70: 174–186. doi:10.1016/j.jrp.2017.07.005.

Personality Predicts Mortality Risk: An Integrative Data Analysis of 15 International Longitudinal Studies

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Abstract

This study examined the Big Five personality traits as predictors of mortality risk, and smoking as a mediator of that association. Replication was built into the fabric of our design: we used a Coordinated Analysis with 15 international datasets, representing 44,094 participants. We found that high neuroticism and low conscientiousness, extraversion, and agreeableness were consistent predictors of mortality across studies. Smoking had a small mediating effect for neuroticism. Country and baseline age explained variation in effects: studies with older baseline age showed a pattern of protective effects (HR<1.00) for openness, and U.S. studies showed a pattern of protective effects for extraversion. This study demonstrated coordinated analysis as a powerful approach to enhance replicability and reproducibility, especially for aging-related longitudinal research.

Keywords

Personality; Mortality; Health Behaviors; Replicability; Generalizability

1. Introduction

Personality traits are important predictors of health outcomes, including mortality risk (Friedman et al., 1995; Jokela et al., 2013), however, several questions remain with respect to this association. First, are all five of the Big Five traits related to mortality? Some recent studies have concluded that only conscientiousness predicts longevity (Jokela et al., 2013), although some have disagreed with this position (Chapman, Hampson, & Clarkin, 2014; P. T. Costa, Jr., Weiss, Duberstein, Friedman, & Siegler, 2014). Certainly, the effects of high conscientiousness and low neuroticism are well-established (Friedman, Kern, Hampson, & Duckworth, 2014) yet with the exception of Jokela et al., (2012) few large-scale investigations have examined the other traits. Second, to what extent do health-detrimental or health-promoting factors mediate the personality-mortality association? There has been some work on such mediation models (Mroczek, Spiro, & Turiano, 2009; Turiano, Chapman, Gruenewald, & Mroczek, 2015), but never in a large-scale, multiple-study context. This study addressed both of these questions, and did so using a novel methodological framework designed to enhance replicability: Coordinated Analysis. Rather than analyze different data sets one or two at a time, in what could be many separate papers, a Coordinated Analysis (a form of Integrative Data Analysis, or IDA; (Curran & Hussong, 2009; Hofer & Piccinin, 2009; Shrout, 2009)) seeks to harness many data sets at once, thereby leveraging power and sample diversity to create a more complete picture of an effect or set of effects than would otherwise be possible. In essence, we had two foci, one substantive and the methodological and replication-oriented.

Personality, Health Behavior, and Mortality Risk

Our substantive research questions were guided by the health-behavior model of personality (Friedman et al., 1995), a theoretical framework positing that personality traits predispose individuals to engage in health-beneficial and refrain from health-detrimental behaviors, such as neglecting to visit a doctor regularly, smoking, or physical inactivity (Mroczek et al.,

2009; Turiano et al., 2015). Behaviors such as smoking are one set of potential mechanisms, or mediators (MacKinnon, 2008), that connect personality traits to long-term downstream outcomes of disease and mortality. Newer formulations of this model (Friedman et al., 2014) have emphasized dynamic, changing aspects of both personality and health behaviors over the lifespan (Chapman et al., 2014; Shanahan, Hill, Roberts, Eccles, & Friedman, 2014). This is a development we endorse, however the logistical constraints of a large scale (15-study) Coordinated Analysis limited us to basic tests of the personality-health behavior model. The current study sought to provide a set of (up to) 15 tests of the association between the full Big 5 personality traits and mortality, with data sets from around the world, as well as a set of mediation tests using the key health-detrimental behavior of smoking. Over the 15 studies, we had a wider range of follow up times (42.75 years) than has been used in most prior investigations. This is important because, despite a well-articulated theoretical model of personality and health behaviors (Friedman et al., 2014; Friedman et al., 1995), there has been very little longitudinal work that connects traits to mediating mechanisms (such as health behaviors) and then, within the same sample, link even further downstream to long-term outcomes such as mortality. Part of the reason for this paucity has to do with the demands of obtaining such long-term data, and when a qualifying extant study is identified, it is often the case the full desired set of mediators are unmeasured.

This work also has practical or applied significance in that it could demonstrate the role of personality traits as psychosocial or behavioral “vital signs” that predict long-term health risks for individuals. Traits may be useful to health care professionals to identify those who are at greater risk for early health problems and earlier mortality, even without knowing what future health-detrimental behaviors they may be likely to engage in. Using discrete-time longitudinal mixture analysis under a structural equation modeling (SEM) framework (B. O. Muthen & Masyn, 2005), we simultaneously tested both direct effects of traits on mortality risk, and indirect effects of traits (mediation) through smoking on mortality risk (CDC, 2008).

Coordinated Analysis: A Technique to Enhance Replication

Complementing the above substantive goal, we had an additional methodological goal of this study that was focused on enhancing replication of results. There is great concern at present with replicability of findings in psychological science (Open Science Collaboration, 2015). Much of this concern has focused on research that uses experimental design. However, areas that use other techniques, such as longitudinal designs, have unique replication challenges that have largely gone unaddressed in the more experimentally-oriented debates about replication in psychology. It is not easy to replicate a long-term longitudinal finding, especially one that uses a large N. An experiment that deploys a relatively small N, a cross-sectional design, and a convenience sample can be run again on a new sample quickly. Replications cannot be done quickly with mortality follow-ups or other long-term longitudinal investigations, which can span years or decades. In addition, groupings of longitudinal studies tend to be different enough from one another (different measures of the same constructs, samples of different ages or from different countries) that exact replications are often impossible, although these study-level differences can often greatly enhance generalizability and external validity. In addition, many recent replication

efforts are comprised of a single attempt to replicate a given result. However, two studies do not necessarily make – or break – a replicable result. Hofer and Piccinin (2009) proposed Coordinated Analysis as a possible solution to this problem of robustness and replication in hard-to-obtain longitudinal studies.

Coordinated Analysis is a form of Integrative Data Analysis (IDA; (Curran & Hussong, 2009; Hofer & Piccinin, 2009) has two main forms, Coordinated Analysis and Pooled Analysis. Coordinated analysis marshals multiple datasets, estimates identical data-analytic models (using the same code) to answer a given research question, and then summarizes effect sizes using tools borrowed from meta-analysis such as forest plots of effects sizes and weighted summary effects. In contrast, pooled analysis, another form of IDA merges data sets to obtain a single effect size. Coordinated Analysis approach promotes and accelerates the process of obtaining the multiple replications required to have confidence in a finding. In lieu of waiting for the investigators of longitudinal studies to test and publish results on a given research question, then waiting further still for someone to meta-analyze that literature, Coordinated Analysis can expedite the process. With coordinated analysis, it is also possible to maximize the comparability of the models, including operationalizing of measurement and conditioning on a similar set of covariates.

In the area of personality and mortality, Pooled Analysis investigations have been successfully carried out using 2 or 3 merged data sets (Jokela et al., 2013; Jokela, Pulkki-Raback, Elovainio, & Kivimaki, 2014; Jokela et al., 2010). However, Pooled Analysis requires the same measures of constructs and ultimately obtains a single effect size (per research question). Coordinated Analysis preserves the heterogeneity of effect sizes across studies, and because it doesn't pool data, can accommodate studies that do not have the same measures of constructs, or other key differences. This permits a larger total number of studies to be included. Despite these advantages, most Coordinated Analyses are based on 3 to 6 studies. In the current investigation, we opted for a much larger-scale attempt, and included 15 studies from 5 different countries, representing up to 44,094 participants depending on the construct. This is perhaps the largest Coordinated Analysis attempted to date. It draws upon a cooperative network of studies: the IALSA (Integrative Analysis of Longitudinal Studies of Aging). We tested the effects of the full Big Five dimensions of personality (together and separately) on mortality risk, as well as the role of smoking as a mediator of the personality-mortality association. Lastly, we attempted to explain the heterogeneity in effect sizes among studies by considering study-level predictors.

2. Methods

2.1 Studies and Participants

The data analyzed were part of the Integrative Analysis of Longitudinal Studies on Aging (IALSA) network, including 5 (see below) that were publically available independent of their affiliation with the IALSA network. In November 2013, we began to search for the studies in the IALSA network that contained at least one measurement of personality traits (the full Big Five or a subset), mortality data (death status, month and year of death at minimum), as well as smoking behaviors measured at or a short time after the personality assessment (to maximize available mortality data). We initiated contact with the

corresponding investigator for each study (each of whom is a co-author) in February 2014, first to establish interest in participating in the project, and second to determine whether the analyses would be carried out at Northwestern, or at the investigator's institution. Based on their response, investigators were sent either 1) a detailed variable list with specific coding instructions, by which to prepare a data set for analysis at Northwestern, or 2) sent the syntax for the analyses, with specific instructions on how to prepare their data, to conduct analyses at a site where a given study's data resides. Regardless of the option a given study chose, all models were run using the same set of Mplus syntax files. Investigators at Northwestern then exponentiated the parameter estimates into hazard ratios, and summarized these via meta-analysis and forest plots. The DerSimonian-Laird technique, a non-iterative method of estimating inter-study variance by weighting the point estimates with each study's sample variance was used to estimate the hazard ratios.

Of the 15 longitudinal datasets used for this project, 10 are available via the IALSA network (EAS, CLS, NAS, SATSA, NSHD, LASA, ILSE, SLS, ROS, MARS, OCTO-Twin). The remaining 5 are archived data sets available to qualified researchers, and are also part of IALSA (WLS-G, WLS-S, MIDUS, HRS, LBLS). Data sharing agreements were put into place for the non-archived studies listed above in order for us to include them.

1 **Einstein Aging Study (EAS).** The primary objective of the EAS is to study the aging brain, with a particular interest in health aging. This study is a longitudinal cohort study of community-based adults who were systematically recruited from Bronx County, NY beginning in 1993. Participants undergo annual assessment including psychosocial measures. The sample is composed of participants aged 70 years and older (mean age=81.49(5.24)), and 59.2% are female (Katz et al., 2012; Mathiesen & Tambs, 1999; Naerde, Roysamb, & Tambs, 2004). The current analysis used 1,530 participants with the requisite data. Personality was assessed via IPIP measures in 2005. EAS analyses were completed at Northwestern.

2 **Health & Retirement Study (HRS).** The objective of the HRS is to understand health processes of adults after retirement. The HRS is a nationally representative longitudinal sample of over 20,000 participants who are surveyed every two years since 1992 (Juster & Suzman, 1995). The average age of the sample is 67.97(11.12), and 59% are female. A total of 7,533 participants had the requisite data. Personality was assessed via IPIP measures in 2006 Analyses were completed at Northwestern.

3 **Long Beach Longitudinal Study (LBLS).** The LBLS started in 1978, and consisted of 589 adults aged 28–84. This sample was resurveyed in 1994–1995, and has since been reassessed two additional times (2000–2002 and 2008–2013). Additional cohorts were added in the second two waves of data collections (Zelinski & Kennison, 2001). The average age of the sample used in this study was 69.34 (13.83), and 52.3% are female. The current study contains a subsample of 348 individuals with requisite data for the current analysis. Personality was assessed using the NEO in 1994/1995. Analyses were completed at Northwestern.

4 **Midlife in U.S. Study (MIDUS).** MIDUS is an ongoing nationally representative study of 7,108 participants in the U.S. that began in 1994/1995, and has since added two additional waves of data collection, in 2004/2005, and 2013/2014. The average of this sample at baseline was 46.38(13), and is 51% female. Personality was derived from IPIP items in 1994. MIDUS investigators completed these analyses (Brim, Ryff, & Kessler, 2004).

5 **The Minority Aging Research Study (MARS)**. MARS is a cohort study of 750 individuals in the Chicago area aged 65 and older (Barnes, Shah, Aggarwal, Bennett, & Schneider, 2012). Initiated in 2004, it studies aging and risk factors of cognitive decline. Participants all self-identify as African American, and contribute data annually, including questionnaires, physical measures, and biological samples. Personality data were collected at baseline using the NEO (2004). The average age at baseline was 73.29(6.45), and is 23.9% female. A total of 632 individuals have data for the current study. Analyses were completed at Northwestern.

6 **Veteran Affairs Normative Aging Study (NAS).** The NAS was founded at the Boston VA outpatient clinic in 1963, as a longitudinal study of aging in men (Bosse, Ekerdt, & Silbert, 1984). The original sample of 2,280 men have been tracked and tested every few years. In 1990–1991, the investigators first collected the Big Five personality measurements, using Goldberg's Big Five items (Goldberg, 1990). The current analysis was conducted on the 1,286 men who were alive in 1990 to complete the personality inventory and report smoking status. The average age at baseline was 64.91(.91). NAS investigators completed these analyses.

7 **The Religious Orders Study (ROS).** The ROS is a longitudinal cohort study of Catholic priests, brothers, and sisters from more than 40 religious communities across the United States that began in 1994 (Bennett, Schneider, Arvanitakis, & Wilson, 2012). Personality was assessed via the NEO in 1994. The average age at baseline was 75.66(7.45), and was 30.4% female. The current study had data for 1,185 participants, and these analyses were completed at Northwestern.

8 **Seattle Longitudinal Study (SLS).** The SLS began in 1956 and contains multiple longitudinal samples as a part of a cross-sequential design. The sample used for the current analysis comes from the 2001 wave of data collection, when the Big-Five personality inventory was introduced to the study (Schaie, Willis, & Caskie, 2004). The average age at this measurement was 63.4(15.64), and was 55.3% female. A total of 1,331 participants had data for the current analyses, and the SLS investigators completed the analyses.

9/10 **Wisconsin Longitudinal Study Grads and Sibs (WLS).** WLS is a study of 22,334 participants in the U.S. started in 1957 to track the class of 1957 high school graduates in Wisconsin. Several years later a sample of these graduates' siblings was collected and both samples are still being followed up on periodically (Herd, Carr, & Roan, 2014; Sewell, Hauser, Springer, & Hauser, 2003). The full big five personality traits were assessed via the BFI (John,

Donahue, & Kentle, 1991) in 1992–1993 for the graduate sample, and in 1993–1994 for the sibling sample. The graduate sample had an average age of 53.7(.67) and was 51.6% female at baseline, while the sibling sample had an average age of 54.9(.52) and was 49.5% female at baseline. For the current analysis we had the requisite data for 8,471 graduates and 4,779 siblings. These analyses were completed at Northwestern.

11 **MRC National Survey of Health & Development (NSHD).** The NSHD is an ongoing cohort study of 5,362 participants from Great Britain who have been followed since birth in March 1946 (Wadsworth et al., (2006). Personality was assessed in 1972 when participants were aged 26, using the Eysenck Personality Inventory (Neuroticism and Extraversion), as well as health behaviors. A total of 3,398 participants had the requisite data. At baseline, the sample had an average age of 26 and was 48% female. NSHD investigators completed these analyses.

12 **OCTO-Twin.** The OCTO-Twin study began in 1991 and included 351 twin pairs (702 individuals) aged 80 years and older. The data include five cycles of longitudinal data collected every two years (McClearn et al., 1997). At baseline, the sample had an average age of 83.58(3.17) and was 66.6% female. The current analysis included 653 participants who had data for this analysis. The OCTO-Twin investigators completed these analyses.

13 **Swedish Adoption Twin Study of Aging (SATSA).** SATSA was begun in 1984 with sample of twins reared apart and a matched sample of twins reared together (Finkel & Pedersen, 2004; Pedersen et al., 1991). Questionnaires including measures of personality, health, environments and lifestyle were collected from 2,019 twins at the initial assessment. For the current project we randomly selected one member from each pair and the final analysis sample included 991 twins. At baseline, the sample had an average age of 60.0(13.95), and was 59.4% female. Personality was assessed via the Eysenck, as well as the NEO. The SATSA investigators completed these analyses.

14 **Longitudinal Study of Amsterdam (LASA).** LASA is an ongoing study of 5,132 participants in the Netherlands started in 1992, and focuses on physical, emotional, cognitive, and social functioning in older adulthood, with three-year measurement cycles (Huisman et al., 2011)(Huisman et al., 2011). Personality (neuroticism only) was assessed in 1993 using the Dutch Personality Questionnaire. For the current analysis we had data for 4,057 individuals. At baseline, the sample had an average of 68.13(9.05) and was 51.8% female. These analyses were completed at Northwestern.

15 **Canberra Longitudinal Study (CLS).** The CLS is a longitudinal epidemiological survey of community-based adults aged 70 years or older living in or near Canberra, Australia that began in 1990 (Christensen, Mackinnon, & Jorm, 2004). Personality was assessed in 1990 via the EPQ (Eysenck). At baseline, the sample had an average age of 76,55(4.94) and was 49.1% female. The sample consists of 894 participants with data for the current analysis. CLS investigators completed these analyses.

2.2 Measures

2.2.1 Covariates—All models were adjusted for age, sex, and education since these variables have known associations with mortality risk. All continuous variables (age, education, personality) were standardized (mean=0, SD=1) for these analyses to have comparable scales across measures, and discrete variables (smoking status) were dummy coded such that 0=no and 1=yes.

2.2.2 Smoking—All 15 studies gathered information about whether participants had ever smoked regularly (at least a few cigarettes every day), were currently smoking, or had quit smoking. We used assessments of "current smoking" as the mediator. In each study a variable was created to represent those who were currently smoking cigarettes (compared to those who were not current smokers). Across the 15 studies, the current smoking measurement was taken from as near as possible in time to the personality assessment that was used. This was done to maximize the usable mortality follow up data (if we insisted on lengthy temporal separation between predictor and mediator, we would miss a considerable number of mortality events).

2.2.3 Personality—Personality traits were assessed using various measures of the Big 5 (neuroticism, conscientiousness, extraversion, agreeableness, and openness to experience). Several of the 15 data sets had the full Big 5, while others only had a subset of the traits (e.g., LASA and MARS had only neuroticism). All studies administered short versions of the following personality measures: the NEO Five Factor Inventory (NEO) (P. T. Costa & McCrae, 1992), The Eysenck Personality Inventory (EPI) (Eysenck & Eysenck, 1968), the Big Five Inventory (BFI) (John et al., 1991), short measures derived from the International Personality Item Pool (IPIP) (Goldberg, 1992; Saucier, 1994), or the Dutch Personality Questionnaire (DPQ) (Lutejin, Starren, & Van Dijk, 2000). Most used English-language personality measures but others used the language of the country in which the study was based (e.g., Swedish, Dutch). Regardless of the exact measure, all assessed the Big 5 traits or a subset, meaning our studies were aligned on construct if not exact measure. Past studies have established high correlations between different measures of the same trait across the aforementioned scales and inventories (McCrae & Costa, 1997). For example, neuroticism as assessed by the DPQ is strongly correlated with neuroticism in other measures (Barelds & Luteijn, 2002). While this does not eliminate method variance, the Big 5 constructs align with one another quite well across the instruments used over the 15 studies.

2.2.4 Mortality—All 15 data sets contain mortality information (death status, death dates [at least month/year of death]), obtained through a reliable source, such as national mortality databases (e.g., U.S. National Death Index, Swedish Death Index) or official death certificates. Some studies that include considerable numbers of participants born 80 or more years ago (NAS, ROS, CLS, EAS, OCTO-Twin) have large percentages that are decedents, and as noted earlier, OCTO-Twin is entirely deceased. Others that include larger groups of younger respondents have lower rates of mortality (the MIDUS has a decedent rate of approximately 8.6%). Yet all have enough decedents (more than 5%) to carry out models using mortality risk as an outcome. See Figure 1 for a visual timeline of data collection for each study.

2.3 Data Analysis

A series of discrete-time survival models were used to examine the association between the personality traits and mortality risk using Mplus® 6.0 and 7.2 software (L. K. Muthen & Muthen, 2012). These analyses were modeled assuming proportional hazards (i.e. Cox Models) within a structural equation modeling (SEM) framework in order to determine the likelihood of death conditioned on personality, smoking, and the other independent variables. Proportional hazards models are ideal for estimating mortality risk because they can account for varying survival times, censoring (those who have not yet died), ages at study entry, and occurrence of a discrete outcome (e.g., death) (Cox, 1972). These models provide estimates, in the form of hazard ratios, that indicate how much the odds of surviving to a given point over the follow-up period increases or decreases given a one-unit difference in a predictor (expressed in standard deviation units in the current study). We also tested mediation models in order to evaluate indirect effects of smoking. Our models combine SEM with proportional hazards models, which we have done in prior work (Turiano et al., 2015). The models estimated the effects of all three pathways in the mediation models (personality → mortality, personality → smoking, smoking → mortality) (see Figure 2 for theoretical model) as well as the mediating effects of personality on mortality through smoking (Fig 5, A–E). See Tables 2–5 for the hazard ratios for each pathway across study.

Testing mediation in proportional hazards modeling through an SEM framework allows simultaneous assessment of both the direct and indirect effects on survival time (Asparouhov, Masyn, & Muthen, 2006), and only in the past 10 years has SEM been adapted for use with censored outcomes such as mortality status and survival time. We used a maximum likelihood robust (MLR) estimator to estimate survival times and Monte Carlo integration to simulate the distribution of these times conditioned on smoking status. The model calculates indirect effects that are comparable to the Sobel method. A *product-of-coefficients* approach computes the ratio of the path from the predictor (personality) to the mediator (smoking) and the path from the mediator (smoking) to the outcome (mortality) (Gunzler, Chen, Wu, & Zhang, 2013). Importantly, this technique provides standard errors, confidence limits, and significance tests of the indirect effects, permitting the statistical interpretation of mediation. All estimates for the direct and indirect effects were exponentiated to provide hazard ratios, and can be interpreted as the increased or decreased percent risk of dying over the study follow up by standard deviation unit difference in personality.

Tools from meta-analysis were employed to provide weighted summary effect size across the 15 studies for all main and mediating effects. These were done in SAS 9.3 using the SAS METAANAL Macro, which provides the DerSimonian-Laird estimates of inter-study confidence intervals (Hertzmark & Spiegelman, 2012). Forest plots were produced using the R function **forest.plot.or** (Belisle, 2014).

Testing the full Big Five in the same models: All models reported were tested for each trait individually to deduce its unique effect, independent of the others. However, due to the inter-trait correlations often observed among the Big Five traits, we also tested models that included all 5 traits simultaneously. Only 10 of the 15 studies had measures of the full Big

Five. Of these we selected a convenience subset (ROS, LBLS, WLS-G, WLS-S, SATSA) and ran mediator models that included the full Big Five, thereby adjusting for the effects of the other traits. In these analyses, the direct and indirect effects of personality on mortality risk were consistent with the separated-trait models, and the latter are reported below. That said, in these 5 data sets, it was often the case that in both models that included the full Big Five and those that separated them, confidence intervals passed through 1.00. The weighted effect sizes in both separated and non-separated models showed CIs that were outside 1.00, again highlighting the usefulness of using many data sets at once. See the Supplemental Materials (Table S1) for a summary of these models.

We also estimated all models with and without education as a covariate, as some traits (especially conscientiousness) can be highly correlated with education. Overall the results were unchanged by this modification, so the results below are reported with education included in the models. See Supplemental Materials (Tables S2–S5) for a summary of the models unadjusted for education.

3. Results

The weighted hazard ratios for direct effects of personality and smoking on mortality, and the indirect effects for personality through smoking are composites of the individual study-level hazard ratios, and they are illustrated in Figures 3–6.

3.1 Personality and Mortality

Results indicated that conscientiousness (Fig 3, B) extraversion (Fig. 3, C), and agreeableness (Fig. 3, D) were associated with lower risk of mortality. The hazard ratios represent direct effects in the mediation model and are adjusted for the indirect effects of smoking, as well as controlling for gender, age and education. Note that in each forest plot, there is a distribution of effect sizes around the overall weighted hazard ratio. Across the various studies, there is a range of effect sizes, and in some case no effect. This heterogeneity of effects is a phenomenon one would expect to see in most scientific content areas, but is often understated in published literatures.

Conscientiousness had the largest effect with a weighted hazard ratio across the 15 studies of .89 (95% CI: .85–.95), indicating that higher levels of conscientiousness were associated with a lower mortality rate. Given that the vast majority of people fall within two standard deviations (−2 SD to +2 SD) of the population mean on any trait (assuming a normal distribution), this represents a 44% difference in mortality risk between a person 2 SDs above the mean and a person 2 SDs below the mean. Extraversion and agreeableness had somewhat smaller effects, representing a 24% and 20% lower risk of mortality, respectively. Openness (Figure 3, A) did not have an effect on mortality risk (HR=.97, CI=.92–1.03), at least with respect to the overall weighted hazard ratio. Neuroticism (Fig 3, E) was associated with increased risk of mortality, with a weighted hazard ratio of 1.05 (95% CI: 1.03–1.07), even after accounting for smoking as a mediating factor, representing a 20% difference in mortality risk.

3.2 Personality and Smoking

The direct effects of personality on current smoking status were observed most robustly for neuroticism and extraversion. The forest plots summary of the odds ratios for these results are presented in Figure 4. High levels of both traits were associated with greater likelihood of being a smoker (a 2–7% increased risk per standard deviation). Openness, conscientiousness, and agreeableness did not appear to be associated with smoking; however there was a trend towards lower odds of smoking for those high in agreeableness. For all five traits, but most strongly for these three traits, there is a split in the direction of effects across studies with some showing lower odds and others showing greater odds, which accounts for the relatively small weighted average effect.

3.3 Mediating Effects of Smoking

The direct effects of smoking on mortality, as expected, supported the well-tested and widely accepted notion that smoking is associated with greater risk of death (Fig 5). Individuals who reported being current smokers had a markedly increased risk of mortality (weighted HR= 1.70 95% CI= 1.45–2.00), regardless of the length of follow up, which ranged from 0 to 42 years. The indirect effects were evaluated for current smoking status (Fig 6). These hazard ratios were interpreted as the effect of a trait on mortality, *through* smoking. It is rare for any mediator (such as smoking) to fully account for the effect of a predictor on an outcome (full mediation). Rather, partial mediation is more common and this is what we observed. There were small mediation effects of smoking on the neuroticism-mortality association (0–3%). For conscientiousness, extraversion, openness, and agreeableness, the confidence intervals for the indirect effect included 1.0, indicating that current smoking likely does not mediate the trait-mortality association for these personality traits.

3.4 Heterogeneity of Effects

A central step in traditional meta-analysis is to test between-study heterogeneity. It is natural to expect some level of variation, but if we detect statistical heterogeneity, this would affect how we interpret our results, including any discrepant results (Pereira, Patsopoulos, Salanti, & Ioannidis, 2010). While the current study is not a traditional meta-analysis, we still found it prudent to explore whether the effects are statistically heterogeneous, and explore study-level factors that might explain the variation. We calculated Cochrane's Q and I^2, which describes the percentage of variability in the effects that is due to heterogeneity (rather than sampling error, or chance) (Higgins & Green, 2008). Table 6 summarizes the heterogeneity of the effects of 1) personality on smoking, 2) personality on mortality, 3) smoking on mortality, and 4) the overall indirect effects. Calculating these statistics is relatively simple, but interpreting them are admittedly "arduous tasks in practice"(Pereira et al., 2010). We had between 9 and 15 studies (fewer for some traits, only the full 15 only for neuroticism), and up to 44,094 total cases, thus we had adequate power to detect heterogeneity (Pereira et al., 2010). For the statistically heterogeneous effects, we broke out the results by average baseline age (+/−65) and country of origin (U.S. vs. Non-U.S.) to examine whether the effects consistently varied by these two study-level variables.

3.4.1 Heterogeneity of effects: Effects of personality on smoking—Results indicated significant heterogeneity for the direct effects of personality on smoking (I^2=91.6–98.8), meaning that the differences in these effects across studies was due to heterogeneity between the studies, and not due to sampling error. For neuroticism, we observed that the average effect across the two age groups did not differ between the younger (OR=1.05[0.98–1.14]) and older (OR=1.05[0.89–1.25]) samples. However, we did see a difference between U.S. (OR=1.01[0.90–1.15]) and Non-U.S. (1.14[1.00–1.31]) samples. This suggests that the association between neuroticism and greater odds of smoking is stronger in non-U.S. samples. For extraversion, we observed a small difference between younger (OR=1.08[.100–1.16]) and older (OR=1.02[.86–1.24]) samples, and a bigger difference between the U.S (1.02[.90–1.16]) and Non-U.S. (OR=1.11[.96–1.29]). This suggests that the association between extraversion and greater odds of smoking is stronger for non-U.S. samples.

For openness, conscientiousness, and agreeableness we were only to compare the samples by age, not country of origin, due to a low number of studies from Europe containing the full Big Five. We found that for conscientiousness, there was relatively little difference between the younger (OR=1.01[.93–1.10]) and older (OR=1.03[.86–1.25]) samples. For openness we observed overall greater odds of smoking in the younger samples (OR=1.13[1.05–1.23], and a small protective effect for older samples (OR=0.95[0.79–1.16]). For agreeableness we observed no effect for the younger samples (OR=1.01[.93–1.10]), and a protective effect for the older samples (OR=0.92[.78–1.11]).

3.4.2 Heterogeneity of effects: Personality on mortality—We detected statistically significant heterogeneity in the direct effects of personality on mortality for conscientiousness (I^2=62.35%), openness (I^2=74.24%), and extraversion (I^2=76.56%). When we stratified these effects by baseline age (<65 vs. >65), conscientiousness and extraversion show consistently lower risk of mortality across sample age, indicating that baseline age does not account for the heterogeneity in these effects. The effects for openness however do vary, such that for the older samples (those over 65 on average at baseline), the effects are consistently below 1.00, indicating lower risk of mortality, with an average effect of 0.90 (a stronger effect than the overall average), while the younger samples see a small effect (1.02, CI=.99–1.06), indicating greater risk of mortality. This suggests that openness may be associated with lower risk of mortality, but only in older age. For the country-of-origin stratification, we could only do reliable comparisons for extraversion, as the other traits were not measured in more than one non-U.S. sample (SATSA being the exception). For extraversion, we do see a difference: for U.S. samples the average HR was 0.92, and for non-U.S. samples the average HR was 1.01. This indicates that extraversion may be associated with lower risk of mortality, but only in the United States. Future studies should explore cultural differences in the personality-mortality association, and consider the societal pressure to be extraverted in the U.S. as a potential contributor to these effects. This indicates that the variation in effects of openness on mortality can be (at least partly) explained by baseline age, and the variation in effects of extraversion on mortality can (at least partly) be explained by country of origin.

3.4.3 Heterogeneity of effects: Effects of smoking on mortality—The heterogeneity of the effect of smoking on mortality was also quite high (I^2=98.5). We explored the heterogeneity in smoking effects by baseline age and country. Although there was wide variation in the effects of smoking on mortality, smoking was associated with greater odds of mortality in all studies. For the age stratification, samples that were under 65 on average baseline had a somewhat higher average hazard ratio (HR=1.86) than the older samples (HR=1.56). This suggests that the risk of dying associated with smoking is larger the longer someone smokes (CDC, 2008) (although this interpretation needs to be further explored with appropriate data that includes length of smoking time). For country of origin, we found that U.S. studies had a higher risk of mortality associated with smoking (HR=1.90) than non-U.S. studies (HR=1.40), suggesting that smoking in the U.S. poses a higher risk of mortality than in other countries. It is not clear why this is the case, again suggesting future work.

3.4.4 Heterogeneity of effects: Indirect effects of personality on mortality via smoking—The indirect effects of personality on mortality via smoking were significantly heterogeneous for openness (I^2=63.0%)s, extraversion (I^2= 50.90%), and agreeableness (I^2=67.64%). For the age stratification, the effects were very similar between younger and older sample for extraversion (HR=1.02 [0.98–1.07] vs.1.00 [0.99–1.01]), and agreeableness (HR=1.03 [0.97–1.10] vs. 0.99[0.98–1.01]). We saw a slight difference in the openness effects, such that the older samples had essentially no effect (HR=.99[.98–1.00], while the younger samples showed greater risk of mortality (HR=1.05 [1.00–1.11]), suggesting that the mediating effect of openness on mortality via smoking is only prevalent in younger samples. The indirect effect of extraversion did not vary across country of origin. For extraversion, the indirect effect for both the U.S. and non-U.S. samples were similarly negligible.

4. Discussion

The effects of personality and mortality revealed a wider array of associations between personality traits and mortality risk than had previously been observed (Jokela et al., 2013; Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007). Extraversion and agreeableness emerged as factors associated with lower risk of mortality, which were novel findings. These results held even when these traits were considered along with the other Big Five dimensions in fully adjusted models. Two factors may account for the detection of these effects in our multi-study format. First, the focus of the personality and mortality literature has primarily been on conscientiousness and neuroticism, so tests of extraversion and agreeableness as predictors are infrequent. Second, if analyzed within a single study or data set, an investigator may not have attained traditional statistical significance (the .05 level) and thus chosen not to publish. Compiling these effects across multiple data sets reveals a mix of significant and non-significant effects. This occurred even when the overall weighted effect size showed a confidence interval entirely above or below 1.00. Not every study will return a significant finding – indeed this is a hallmark of significance testing. Additionally, although associations of conscientiousness and neuroticism with mortality have been shown in single-study reports, our Coordinated Analysis indicated that these effects not only replicate, but

are also robust in the sense that they were observed in five different countries of origin, a wide range of follow-up periods, and different measures of the same construct. This addresses the issue of external validity, as we note below.

We considered mechanisms in this study as well, showing that smoking mediated the neuroticism-mortality relationship, thereby partially accounting for the association. This suggests that health behaviors, specifically smoking, are at least one potential explanation for why this trait is associated with mortality. Individuals high in neuroticism are more likely to be current smokers, and these smokers were in turn less likely to survive. The relatively small mediating effects tell us two things: one, that smoking is just one health behavior in a much bigger health-behavior model picture. What we showed here, across studies, is that smoking does indeed play a role, but other factors need to be account for as well in future studies. Additionally, the lack of smoking-related mediation detected for traits like extraversion and agreeableness (even though we found them to be directly associated with mortality), tells us that perhaps different health behaviors, or a different model altogether, may account for associations for these two traits. The characteristics of extraversion and agreeableness are very social in nature, so the links to mortality are more likely to be explained with more social mediators (e.g. social support, relationship status etc…).

In addition, these results have great importance for ongoing debates about replication and reproducibility in the psychological sciences, in health and medical research, and across a variety of other fields. Our multi-study Coordinated Analysis (or IDA) illustrates the utility of many analyses harnessed together to allow comparisons of effects obtained across multiple data sets (which can be construed as multiple replications), with different kinds of samples, varying in countries, ages, and using different measures of the same construct) to better capture a fuller range of effect sizes. The latter addresses issues of external validity and generalizability, concepts often ignored in psychology.

Friedman et al. (2014) has recently argued that investigations of the personality-mortality association should use “full models” or “third generation” models when testing mediation hypotheses. These refer to large numbers of mediators considered simultaneously, along with incorporation of dynamic or changing elements in both predictors and mediators. It is often not possible to carry out such models in a Coordinated Analysis format as not all constituent studies have assessed the relevant predictors and mediators. This typically reduces the scope of testable models, but is countered by the larger number of studies. For example, in the current Coordinated Analysis, smoking was the only universal health-detrimental behavior that was assessed in all data sets. That said, the third-generation models described by Friedman et al. (2014) lay out a potential path for future work.

A related issue concerns control variables not used or unavailable for use in Coordinated Analysis modeling. Epidemiological studies often utilize large numbers of control variables, but this gives rise to issues of over-control and high overlap among predictors and covariates. One set of potential controls we did not use were self-rated and objective indicators of health status. This was in part due to availability and longitudinal timing of such measures, but also reflected concerns we had regarding overlap and over-control. Many self-rating (and objective) health ratings tap into common sources of variance as personality

traits, especially neuroticism. Placing them within the same models often creates a murkier picture rather than clarifying.

Another issue touches on heterogeneity across studies. The effects reported above reflect the weighted hazard ratios computed in the meta-analytic portion of the analysis. The many effect sizes resolved into an overall weighted effect, and paint a more comprehensive picture than any one individual effect. However, for many individual studies there were direct effects of personality on mortality, despite the meta-analytic summary showing no effect. We observed a similar pattern for the indirect effects. For example, there were clear mediation effects of current smoking on the extraversion-mortality association in the Seattle Longitudinal Study (Fig. 6C), of current smoking on the openness-mortality association in the MIDUS sample (Fig. 6A), and of current smoking on the agreeableness-mortality association in the WLS-Grad sample (Fig. 6D). Yet, when the individual mediating effect is averaged across 14 other samples, this claim of mediation is less clear. A single effect from a single sample is not sufficient for a confident scientific claim. Even when many large-N samples were used, there was still a range of effect sizes, although most were within each other's 95% confidence intervals. Even so, there was a distribution of effect sizes across studies that were likely in part due to either random or systematic factors (e.g. differences by countries or baseline age in how personality manifests behaviorally). Among potential systematic factors that may be influencing effect sizes across studies, we found that effects varied somewhat by country, such that the effects of neuroticism on smoking were stronger in non-U.S. studies, and the effects of extraversion on mortality were more protective in U.S. studies. We also found that the effects varied by baseline age (+/−65). Younger samples had an average risk effect of openness on smoking, and of the indirect effect of openness on mortality via smoking. Older samples showed a pattern of openness being associated with lower odds of smoking and mortality, as well as agreeableness on smoking.

Lastly, although Coordinated Analysis is not without limitations, its benefits outweigh the drawbacks. Perhaps its greatest benefit is preservation of the rich heterogeneity of effect sizes, in contrast to estimation of a single effect size, based on pooled samples. This enhances the evaluation of generalizability (external validity) of the personality-mortality association. Coordinated Analysis also allows inclusion of a larger numbers of studies in comparison to Pooled Analysis, which limits the number of data sets due to requirements of measurement harmonization (Kern, Hampson, Goldberg, & Friedman, 2014).

Our analysis also indicates that more work is needed on health behaviors as mediators. Smoking only accounts for a small portion of the association between personality and mortality, and other health behaviors almost certainly also play a role. Furthermore, other mediators, such as social behaviors (social support, strain, network density, marital status, relationship quality), or physiological pathways (Turiano et al., 2015) may play a role in explaining the agreeableness and extraversion effects. Coordinated Analyses that are smaller than the one attempted here (perhaps 6–7 studies) could test a large array of additional mediators and other pathways, and also begin to develop more complex, dynamic models (Shanahan et al., 2014). Additionally, the current study controlled for main effects of gender, but future studies should formally address possible gender effects, and stratify similar models by sex. Furthermore, objective or subjective health status may also be related to both

smoking and mortality. It is reasonable to hypothesize that health measures may mediate the personality-mortality association in similar ways as smoking behavior, such that higher levels of certain traits (e.g. neuroticism) would be associated with worse health, and therefore greater risk of mortality. Health could also act as a moderator, such that the association between personality and mortality is stronger among those with better or worse health. For example, we could expect to find that conscientiousness is associated with lower odds of mortality, but only among those in better health. In order to formally test these associations, we would need to establish the subset of studies that have comparable measures of subjective health (i.e. self-rated health) or objective measures (e.g., current heart conditions, diabetes etc.). Future work will extend the results of this paper to include both subjective and objective health as a moderator or mediator of the personality-mortality association.

This paper has provided a "proof of concept" for a large-scale Coordinated Analysis (smaller studies involve 5–6 studies had been done; (Piccinin et al., 2013)) along with presenting novel substantive findings. We hope other researchers will use the Coordinated Analysis technique to answer other research questions in personality, thereby generating new findings that are simultaneously more robust, replicable, and authoritative.

4.1 Conclusions

The current study found that neuroticism was associated with a higher risk of mortality, while conscientiousness, extraversion, and agreeableness were associated with lower risk of mortality. Additionally, we found that smoking mediates the association between neuroticism and mortality, such that individuals reporting higher neuroticism were more likely to be smokers, and thus had greater risk of dying in the follow up period. Coordinated Analysis (a form of Integrative Data Analysis) is a useful technique for addressing issues of replicability in many scientific fields. It is especially useful for long-term longitudinal studies where fresh-data collection replication attempts can take years or decades to carry out. In the current study, the individual findings derived from 15 different long-term studies indicated that 4 of the 5 "Big Five" personality traits have a direct effect on mortality risk and that smoking is one explanation as to why this is the case. People with certain levels of personality traits (e.g., high neuroticism) are more likely to smoke, and have a greater likelihood of dying sooner. The Coordinated Analysis process is time-consuming (although less so than conducting a longitudinal study over a decade or two), but provides richer quality results than single-study designs, providing many replications within a single set of studies. The results presented here point to the benefits of multi-study analyses in promoting reproducibility, replicability, and cumulative science. Moreover, the use of multiple, diverse studies enhances the external validity of findings on a given topic.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Data for the analyses reported in this paper are available in a number of locations. Information about specific data sets can be provided upon request. We would like to thank all participating studies for sharing data and, providing support with respect to data management and execution of analyses. Each study has at least one investigator who is an author on this paper. Funding support for this project was provided by the National Institute on Aging: P01-AG043362 (*Integrative Analysis of Longitudinal Studies of Aging (IALSA)*, [Scott M. Hofer (PI)]), and Daniel K. Mroczek (Co-Investigator and Project Leader of the IALSA Personality & Health Project, as well as R01-AG018436 [*Personality & Well-Being Trajectories in Adulthood*, Daniel K. Mroczek, PI]).

1. Religious Orders Study (ROS) – P30-AG10161, R01-AG15819, Bennett (PI)
2. Minority Aging Research Study (MARS) – R01-AG22018, Barnes (PI)
3. VA Normative Aging Study. The VA Normative Aging Study is a research component of the Massachusetts Veterans Epidemiology Research and Information Center, and is supported by the US Department of Veterans Affairs Cooperative Studies Program / Epidemiological Research and Information Centers. Spiro (Associate Director)
4. Midlife in the United States Study (MIDUS)- P01-AG020166 Ryff (PI)
5. Canberra Longitudinal Study (CLS)- NHMRC Unit Grants 973301, 933301, NHMRC Program Grant 179805. Batterham: supported by NHMRC fellowship 1083311.
6. Swedish Adoption Twin Study of Aging (SATSA)- R01-AG04563, and R01-AG10175, the MacArthur Foundation Research Network on Successful Aging, the Swedish Council for Working Life and Social Research (FAS) (97:0147:1B, 2009-0795), and the Swedish Research Council (825-2007-7460, 825-2009-6141).
7. Einstein Aging Study (EAS)- P01-AG03949, and R03-AG045474, the Leonard and Sylvia Marx Foundation, and the Czap Foundation.
8. The Longitudinal Aging Study Amsterdam (LASA)- Ministry of Health, Welfare and Sports of the Netherlands.
9. MRC National Survey of Health and Development (NSHD)- Medical Research Council Unit Programme number MC_UU_12019/1.
10. Long Beach Longitudinal Study (LBLS)- R01-AG10569
11. Seattle Longitudinal Study (SLS)- RC1-AG035645, R21-AG032379, and R21-AG033109
12. Origins of Variance in the Oldest-Old (OCTO-Twin)- NIA (AG08861), the Swedish Council for Working Life and Social Research, the Adlerbertska Foundation, the Wenner-Gren Foundations, and the Wilhelm and Martina Lundgrens Foundation.
13. Wisconsin Longitudinal Study (WLS)- AG-9775, AG-21079, AG-033285, and AG-041868
14. Health and Retirement Study (HRS)- NIA U01AG009740

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Highlights

1. Neuroticism is associated with higher risk of mortality
2. Extraversion, Agreeableness, Conscientiousness are associated with lower mortality
3. Smoking has a small mediating effect on the neuroticism-mortality association
4. These effects are consistent across 15 long term longitudinal studies
5. Baseline age and country-of-origin partially explain heterogeneity in effects

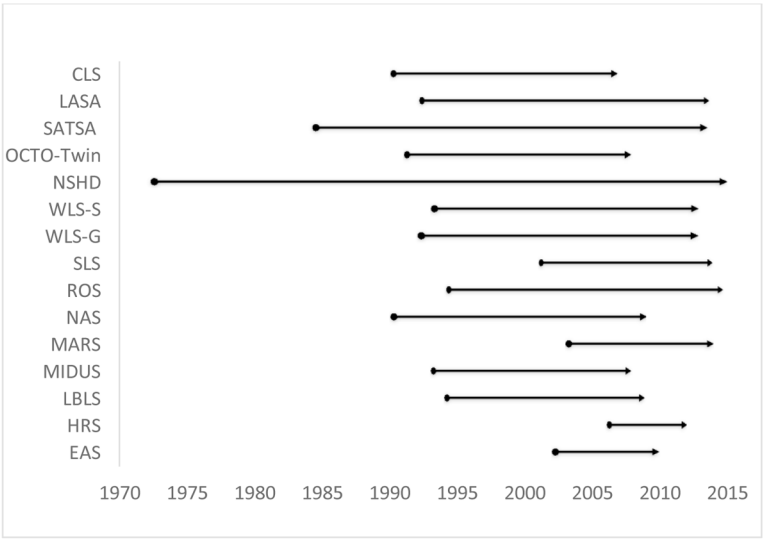

Figure 1.
Visual time line of data collection for each study (personality measurement to mortality follow-up)

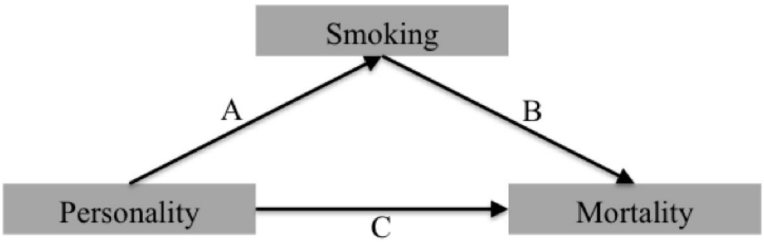

Figure 2.
Theoretical mediation model of personality in relation to mortality directly (path C), and indirectly via smoking (paths A and B).

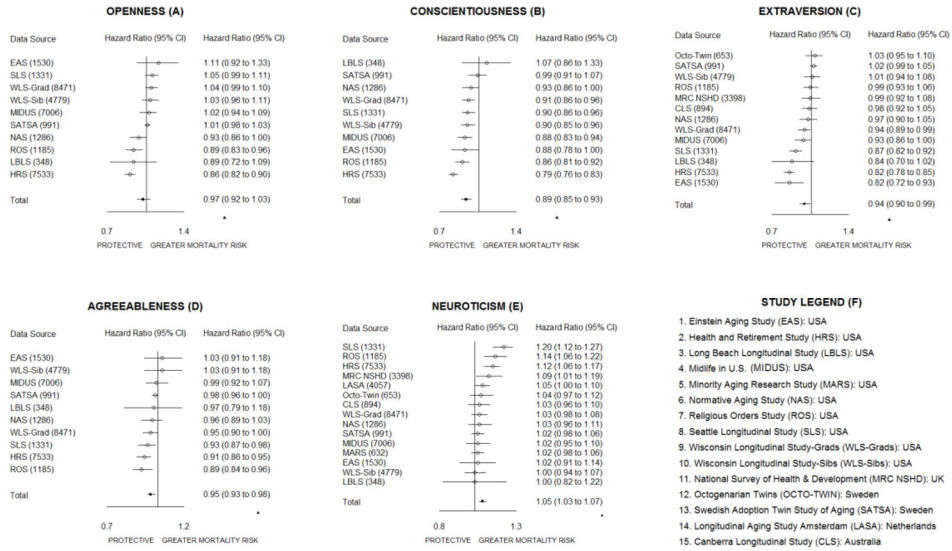

Figure 3.

The direct effects of personality on mortality. Forest plots summarizing the effect for each study, separated by trait (A–E). Each data point on the forest plot reflects the hazard ratio for a study (sorted by strength and direction of the effect), and the error bars represent 95% confidence intervals. The total effect is the average hazard ratio across all of the studies, weighted by N. Panel F provides the full name of each study.

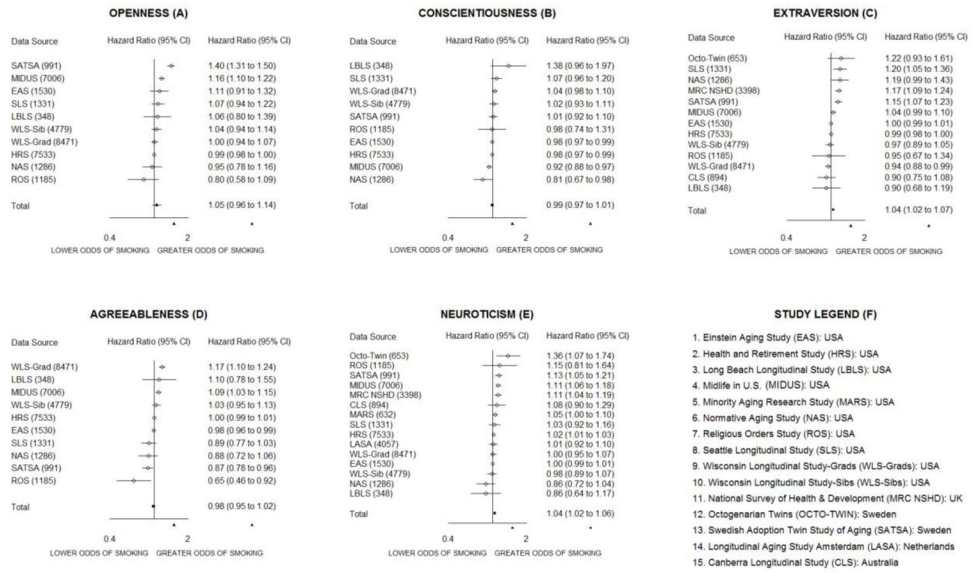

Figure 4.

The direct effects of personality traits on current smoking status. Forest plots summarizing the effect for each study, separated by trait (A–E). Each data point reflects the odds ratio for a study, and error bars represent 95% confidence intervals. The total effect is the average odds ratio, weighted by N.

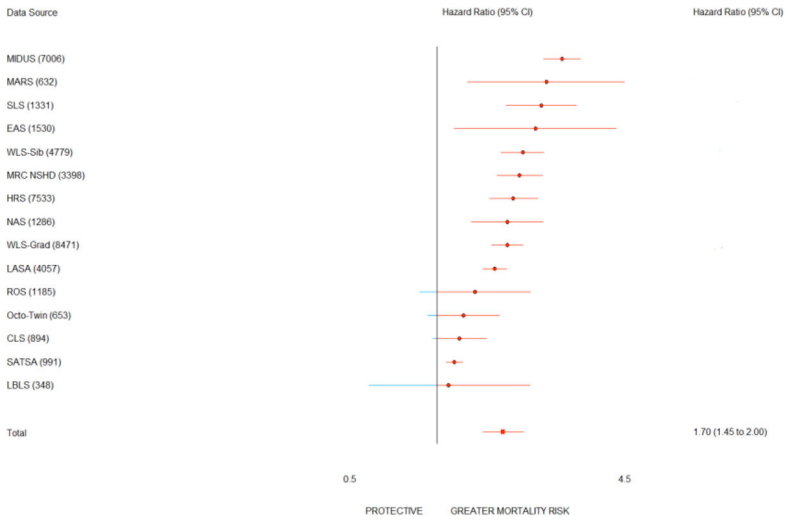

Figure 5.

The direct effects of current smoking on mortality. Forest plot summarizing the effect for each study. Each data point reflects the hazard ratio for each study, and the error bars represent 95% confidence intervals. The bottom (total) effect is the average hazard ratio, weighted by N.

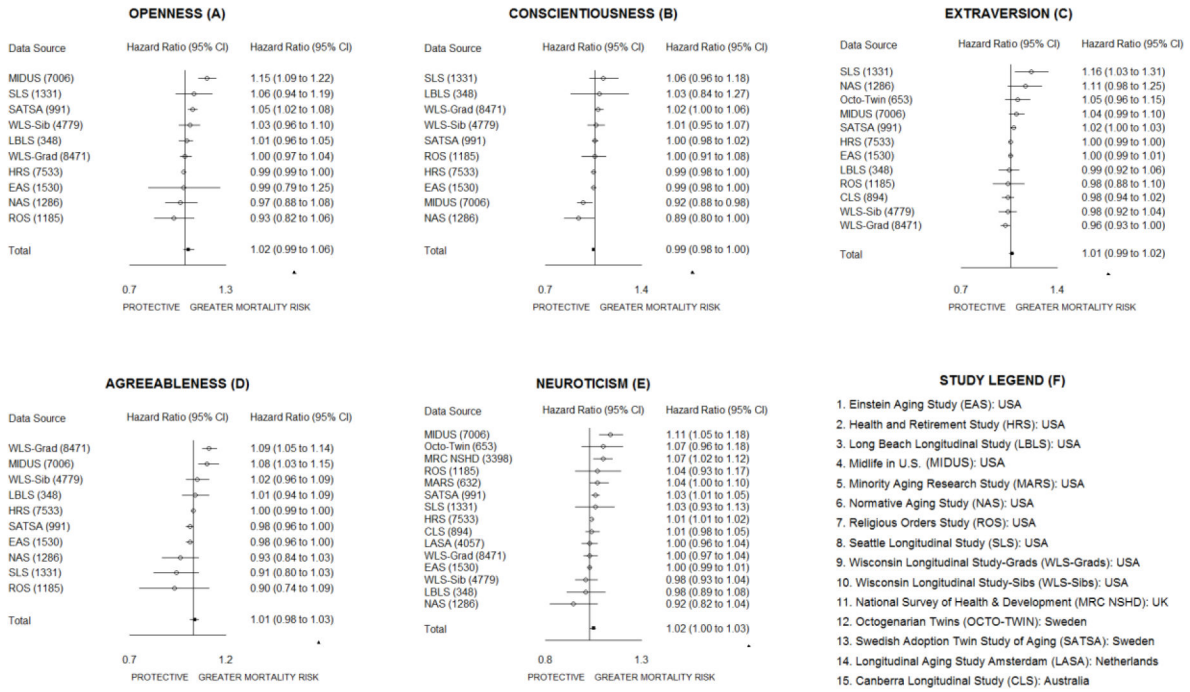

Figure 6.

Indirect effects of personality on mortality, via current smoking status. Forest plots summarizing the effect for each study, separated by trait (A–E). Each data point reflects the hazard ratio for each study, and the error bars represent the 95% confidence intervals. The bottom (total) effect is the average hazard ratio, weighted by N.

Table 1

Descriptive statistics for each dataset

	N	Age M(SD) Range	Ed	Sex (%F)	Current Smoker (%)	Dead (%)	Survival Time: Years M(SD) Range	Personality Scale (Year)
1. EAS	1,530	81.49 (5.24) 70.41– 99.01	14.39 (3.30) 3–24	59.2	3.4	9.8	7.75 (.97) .001–8.00	IPIP (2002)
2. HRS	7,533	67.97 (11.12) 25–104	75.1% HS+	59.0	60.8	31.6	5.66 (0–6)	IPIP (2006)
3. LBLS	348	69.34 (13.83) 30–95	86.3% 12+ yrs	52.3	12.6	45.8	13.18(2.34) 0–14.16	NEO (1994)
4. MIDUS	7,006	46.38 (13) 20–75	38.5% college+	51.0	23	8.6	8.01 (3.9) 0.2–14	IPIP (1994/95)
5. MARS	632	73.29 (6.45) 57.97	69.1% some college+	23.9	7.28	13.97	9.25 (2.17) .26–10	NEO (2004)
6. NAS	1,286	64.91 (7.75) 45–89	28.65% some college+	0	8	41	11.01 (4.55) 0.8–18.5	IPIP (1990/91)
7. ROS	1,185	75.66 (7.45) 55.78–102.15	18.17 (3.35) 3–30	30.4	1.8	56.2	14.13 (7.14) .09–21	NEO (1994)
8. SLS	1,331	63.4 (15.64) 25–99	15.49 (2.63) 7–20	55.3	4.58	28.32	11.71 (2.80) 3–13	NEO (2001)
9. WLS-G	8,471	53.7 (.67) 51.03–82.3	23% college+	51.6	32.6	22.3	19.92 (4.15) 0–21.5	BFI (1992)
10. WLS-S	4,779	54.9 (.52) 52.66–60	28% college+	49.5	30.7	22.2	18.47 (4.03) 0–20	BFI (1993)

	N	Age M(SD) Range	Ed	Sex (%F)	Current Smoker (%)	Dead (%)	Survival Time: Years M(SD) Range	Personality Scale (Year)
11. NSHD	3,398	26 (0)	11.08 (1.29) 10–14.25	48.0	41.7	14.6	40.78 (5.43) 1.17–42.75	Eysenck (1972)
12. OCTO- Twin	653	83.58 (3.17) 79–98	7.14 (2.29) 0–23	66.6	7.7	99.6	6.34 (3.86) 0–16.49	Eysenck (1991)
13. SATSA	991	60 (13.95) 26.07–92.88	5.95% College+	59.4	31.3	65.49	20.7 (9.57) .43–29.58	EPI/NEO (1984)
14. LASA	4,057	68.13 (9.05) 54.8–85.6	13.9% Higher Vocational+	51.8	25.2	60.3	14.93 (7.49) 0–22.08	DPQ (1992/93)
15. CLS	894	76.55 (4.94) 70–97	28.6% College+	49.1	11.5	76.7	9.67 (5.41) 0–16.75	EPQ (1990)

Table 2

Direct Effects of Personality on Mortality (Hazard Ratio [HR], 95% CI)

	Neuroticism			Conscientiousness			Openness			Extraversion			Agreeableness		
	HR	**CI (−)**	**CI (+)**	**HR**	**CI (−)**	**CI (+)**	**HR**	**CI (−)**	**CI (+)**	**HR**	**CI (−)**	**CI (+)**	**HR**	**CI (−)**	**CI (+)**
EAS	1.02	0.91	1.14	0.88	0.78	1.00	1.11	0.92	1.33	0.82	0.71	0.92	1.03	0.91	1.18
HRS	1.12	1.06	1.17	0.79	0.76	0.83	0.86	0.82	0.90	0.82	0.78	0.85	0.91	0.87	0.95
LBLS	1.00	0.82	1.22	1.07	0.86	1.33	0.89	0.73	1.09	0.84	0.70	1.02	0.97	0.79	1.18
MIDUS	1.02	0.95	1.10	0.88	0.83	0.94	1.02	0.94	1.09	0.93	0.86	1.00	0.99	0.92	1.07
MARS	1.02	0.98	1.06	--	--	--	--	--	--	--	--	--	--	--	--
NAS	1.03	0.96	1.11	0.93	0.86	1.00	0.93	0.86	1.00	0.97	0.90	1.05	0.96	0.89	1.03
ROS	1.14	1.06	1.22	0.86	0.81	0.92	0.89	0.83	0.96	0.99	0.93	1.06	0.89	0.84	0.96
SLS	1.20	1.12	1.27	0.90	0.86	0.96	1.05	0.99	1.11	0.87	0.82	0.92	0.93	0.87	0.98
WLS-G	1.03	0.98	1.08	0.91	0.87	0.96	1.05	0.99	1.10	0.94	0.89	0.99	0.95	0.90	1.00
WLS-S	1.00	0.94	1.07	0.90	0.85	0.96	1.03	0.96	1.11	1.01	0.94	1.08	1.03	0.95	1.13
NSHD	1.09	1.01	1.19	--	--	--	--	--	--	0.99	0.92	1.08	--	--	--
OCTO	1.04	0.97	1.12	--	--	--	--	--	--	1.03	0.95	1.11	--	--	--
SATSA	1.02	0.98	1.06	0.99	0.91	1.07	1.01	0.98	1.03	1.02	0.99	1.05	0.98	0.96	1.00
LASA	1.05	1.01	1.10	--	--	--	--	--	--	--	--	--	--	--	--
CLS	1.03	0.96	1.10	--	--	--	--	--	--	0.98	0.92	1.05	--	--	--

Note. These are the C pathways of the full mediation models; -- = not estimated, trait not assessed in study.

Table 3

Direct Effects of Personality on Current Smoking (Odds Ratios [OR], 95% CI)

	Neuroticism			Conscientiousness			Openness			Extraversion			Agreeableness		
	OR	CI (−)	CI (+)	OR	CI (−)	CI (+)	OR	CI (−)	CI (+)	OR	CI (−)	CI (+)	OR	CI (−)	CI (+)
EAS	1.00	0.99	1.01	0.98	0.97	1.00	0.98	0.94	1.02	1.00	0.99	1.01	0.97	0.96	0.99
HRS	1.02	1.02	1.03	0.98	0.97	0.99	0.99	0.98	1.00	0.99	0.99	1.00	1.00	0.99	1.01
LBLS	0.86	0.64	1.17	1.38	0.96	1.97	1.06	0.80	1.39	0.90	0.68	1.19	1.10	0.78	1.55
MIDUS	1.11	1.06	1.18	0.92	0.88	0.97	1.16	1.10	1.22	1.04	0.99	1.10	1.09	1.03	1.15
MARS	1.05	1.00	1.10	--	--	--	--	--	--	--	--	--	--	--	--
NAS	0.86	0.72	1.04	0.81	0.67	0.98	0.95	0.78	1.16	1.19	0.99	1.43	0.88	0.72	1.06
ROS	1.15	0.81	1.64	0.98	0.74	1.31	0.80	0.58	1.09	0.95	0.67	1.34	0.65	0.46	0.92
SLS	1.03	0.92	1.16	1.07	0.96	1.20	1.07	0.94	1.22	1.20	1.05	1.36	0.89	0.77	1.03
WLS-G	1.00	0.95	1.07	1.04	0.98	1.10	1.00	0.94	1.07	0.94	0.88	0.99	1.17	1.10	1.24
WLS-S	0.98	0.89	1.07	1.02	0.93	1.11	1.04	0.94	1.14	0.97	0.89	1.05	1.03	0.95	1.13
NSHD	1.11	1.04	1.19	--	--	--	--	--	--	1.17	1.09	1.24	--	--	--
OCTO	1.36	1.07	1.74	--	--	--	--	--	--	1.22	0.93	1.61	--	--	--
SATSA	1.13	1.05	1.21	1.01	0.92	1.10	1.40	1.31	1.50	1.15	1.07	1.23	0.87	0.78	0.96
LASA	1.01	0.92	1.10	--	--	--	--	--	--	--	--	--	--	--	--
CLS	1.08	0.90	1.29	--	--	--	--	--	--	0.90	0.75	1.08	--	--	--

Note. These are the A pathways of the full mediation models --= not estimated

Table 4

Direct Effects of Current Smoking on Mortality (Hazard Ratio [HR], 95% CI)

	HR	CI (−)	CI (+)
EAS	2.20	1.15	4.20
HRS	1.84	1.52	2.24
LBLS	1.10	0.58	2.10
MIDUS	2.72	2.35	3.15
MARS	2.40	1.28	4.49
NAS	1.76	1.32	2.34
ROS	1.36	0.87	2.11
SLS	2.31	1.74	3.06
WLS-G	1.76	1.55	1.99
WLS-S	1.99	1.67	2.35
NSHD	1.94	1.62	2.33
OCTO	1.24	0.93	1.65
SATSA	1.15	1.08	1.23
LASA	1.59	1.44	1.75
CLS	1.20	0.97	1.49

Note. These are the B pathways of the full mediation models

Table 5

Indirect Effects of Personality on Mortality via Current Smoking (Hazard Ratio [HR], 95% CI)

	Neuroticism			Conscientiousness			Openness			Extraversion			Agreeableness		
	HR	**CI (−)**	**CI (+)**	**HR**	**CI (−)**	**CI (+)**	**HR**	**CI (−)**	**CI (+)**	**HR**	**CI (−)**	**CI (+)**	**HR**	**CI (−)**	**CI (+)**
EAS	1.00	0.99	1.01	0.99	0.98	1.00	0.99	0.79	1.25	1.00	0.99	1.01	0.98	0.96	1.00
HRS	1.01	1.01	1.02	0.99	0.98	1.00	0.99	0.99	1.00	1.00	0.99	1.00	1.00	0.99	1.00
LBLS	0.98	0.89	1.08	1.03	0.84	1.27	1.01	0.96	1.06	0.99	0.92	1.07	1.01	0.94	1.09
MIDUS	1.11	1.05	1.18	0.93	0.88	0.98	1.15	1.09	1.22	1.04	0.99	1.10	1.09	1.03	1.15
MARS	1.05	0.99	1.10	--	--	--	--	--	--	--	--	--	--	--	--
NAS	0.92	0.82	1.04	0.89	0.80	1.00	0.97	0.88	1.08	1.11	0.98	1.25	0.93	0.84	1.03
ROS	1.04	0.93	1.17	1.00	0.91	1.08	0.93	0.82	1.06	0.98	0.88	1.10	0.90	0.74	1.09
SLS	1.03	0.93	1.13	1.06	0.96	1.18	1.06	0.95	1.19	1.16	1.04	1.31	0.91	0.80	1.03
WLS-G	1.00	0.97	1.04	1.03	1.00	1.06	1.00	0.97	1.04	0.96	0.93	1.00	1.09	1.05	1.14
WLS-S	0.98	0.93	1.04	1.01	0.95	1.07	1.03	0.96	1.10	0.98	0.92	1.04	1.02	0.96	1.09
NSHD	1.07	1.02	1.12	--	--	--	--	--	--	1.12	1.05	1.17	--	--	--
OCTO	1.07	0.97	1.18	--	--	--	--	--	--	1.05	0.96	1.15	--	--	--
SATSA	1.03	1.01	1.05	1.00	0.98	1.03	1.05	1.02	1.08	1.02	1.01	1.04	0.98	0.96	1.00
LASA	1.00	0.96	1.05	--	--	--	--	--	--	--	--	--	--	--	--
CLS	1.01	0.98	1.05	--	--	--	--	--	--	0.98	0.94	1.02	--	--	--

Note. --= not estimated, trait not assessed in study.

Table 6

Heterogeneity of effects

	Effect	Q	df	I^2
A-Paths	Consc → Smoke	**107.12**	9	91.6
	Open → Smoke	**661.66**	8	98.8
	Extra → Smoke	**329.4**	12	96.4
	Agree → Smoke	**327**	9	97.2
	Neuro → Smoke	**240.09**	14	94.2
B-Path	Smoking → Mortality	**941.77**	14	98.5
C-Path	Neuro → Mortality	18.36	14	23.7
	Consc → Mortality	**23.91**	9	62.4
	Open → Mortality	**34.17**	9	74.2
	Extra → Mortality	**51.2**	12	76.6
	Agree → Mortality	10.3	9	12.6
Mediation	Neuro → Smoke → Mortality	21.41	14	34.6
	Consc → Smoke → Mortality	10.73	9	16.1
	Open → Smoke → Mortality	**24.31**	8	63.0
	Extra → Smoke → Mortality	**24.44**	12	50.9
	Agree → Smoke → Mortality	**27.81**	9	67.6

Bold denotes statistically significant heterogeneity