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Original Contribution

Examining the Long-Term Association of Personality With Cause-Specific Mortality in London: Four Decades of Mortality Surveillance in the Original Whitehall Smoking Cessation Trial

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The personality domains of extraversion and neuroticism are regarded as being stable individual psychological characteristics, yet it remains unclear whether they are associated with chronic disease over an extended period of time. In a randomized controlled trial of smoking cessation nested within the original prospective Whitehall Study (1967–2012), the Eysenck Personality Questionnaire was administered to 832 male self-declared smokers who had undergone a medical examination during which their levels of extraversion and neuroticism were quantified. In the 42-year follow-up period, there were 781 deaths. In analyses in which participants from both trial arms were pooled, there was little evidence of a robust relation of either personality domain with death from all causes, coronary heart disease, stroke, respiratory disease, or cancer in any of our analyses. We therefore found no support for a role of either extraversion or neuroticism as determinants of long-term mortality risk.

cancer; cohort study; coronary heart disease; extraversion; mortality; neuroticism; personality type; respiratory disease; stroke

Abbreviations: International Classification of Disease (ICD)

The notion that an individual's personality may have an impact on his or her health can be traced back to ancient writings (1). Over the past 2 decades, it has been suggested in a series of empirical reports that people with low levels of neuroticism and high levels of agreeableness, extraversion, and conscientiousness may experience a lower risk of total mortality (2), although these are not universal observations (3).

As the cohort studies on which these observations were based have matured, it has become possible for investigators to explore the association of personality traits with specific chronic disease outcomes of public health importance, such as cardiovascular disease and cancer. The modest evidence base to date suggests that, for cardiovascular disease endpoints—in particular coronary heart disease—neuroticism is, with rare exception (4), typically associated with an elevated risk (5). On the other hand, in large-scale studies (6) and meta-analyses (7), there has been no link shown between this personality domain and risk of all cancers combined. For extraversion, a recent aggregation of effect estimates across previously unpublished studies indicated no association with coronary heart disease but a positive relation with stroke (5). Again, the best evidence does not imply any extraversion-malignancy relationship (6, 7). Plausible mechanisms advanced for positive results with respect to cardiovascular disease outcomes, where they exist, include a more favorable risk factor profile—a lower prevalence of smoking (8), harmful level of alcohol intake (9), and existing disease (clinical and subclinical) (10) and lower levels of weight (11), blood pressure, and blood lipids—in the lower risk personality groups.

There has been some suggestion that certain personality types are more consistently associated with chronic disease risk in studies with a shorter duration of mortality surveillance (12, 13), with the strength of these relationships diminished or lost when a longer period of follow-up is utilized (4, 14). Rather than supporting the stability of personality as a risk factor for mortality (15), this finding is consistent with a reverse causality explanation, which posits that hidden or unmeasured disease may unfavorably influence self-rated

personality disposition (16) and simultaneously raise short-term mortality risk. Studies with several decades of mortality surveillance have the advantage of minimizing the influence of reverse causality because the proportion of persons with existing disease at study entry relative to chronic disease events accumulation decreases over time (17). Given the relative recency of research activity in personality epidemiology, however, such studies are rare.

A further consideration when interpreting the personality and health literature is the influence of publication bias, which is the notion that positive results have a better chance of being published. Thus, in an individual meta-analysis of unpublished raw data, the association between personality type and survival (3) was weaker than that seen in the published literature. Similar observations have been made in the context of other psychological predictors of health, such as job strain and coronary heart disease (18).

Against this background of inconsistent findings, limited evidence, potential publication bias, and the problem of reverse causality, we analyzed data from a smoking cessation trial nested within the original Whitehall Study. With surviving study members now entering their fifth decade of follow-up after personality measurement, this study is well placed to address some of these methodological considerations.

METHODS

The original Whitehall Study is a prospective cohort study with ongoing mortality surveillance (19, 20). Between 1967 and 1970, baseline data were collected on 19,019 male civil service (government) employees who were based in London, United Kingdom, and were 40–69 years of age. This represented a 77% response proportion from the target population. Data collection involved the completion of a study questionnaire and participation in a medical examination, both of which have been described in detail previously (19). In brief, during the medical examination, blood pressure (21), height, weight, pulmonary function (assessed using forced expiratory volume in 1 second) (22), and, after an overnight fast, plasma cholesterol (23) were measured using standard protocols of the period. Body mass index was computed using the usual formula (weight (kg)/height $(m)^2$) (24). The questionnaire included enquiries about employment grade (an indicator of socioeconomic status) (25), smoking habits (26), physical activity level (inactivity was defined as inactive leisure activity or no travel activity) (27), and existing disease (1 or more of the following: electrocardiographic evidence of ischemia, self-reported dyspnea, intermittent claudication, unexplained weight loss, bronchitis, use of blood pressure–lowering medication, or being under the care of a physician because of heart trouble or hypertension).

The data on personality traits were collected from a subgroup of male cigarette smokers who participated in a trial of smoking cessation, respiratory health, and mortality that was nested within the original cohort study, which served as a screening study (see Web Figure 1, available at http://aje.oxfordjournals.org/, for derivation of trial sample). As outlined elsewhere (28–31), 1,500 cigarette smokers with additional risk factors for cardiovascular disease—based on an early version of the Framingham index (32)—were targeted for recruitment into a randomized controlled trial of smoking cessation in which advice about quitting was dispensed by a physician. In an era in which the health effects of cigarette smoking were not fully understood, the usual care group received no cessation advice. In addition to the baseline medical examination that was carried out as part of the cohort study, detailed respiratory health measurements (lung function, self-reported phlegm and dyspnea, angina, etc.) were taken 1 and 3 years after the initial intervention.

Eysenck Personality Questionnaire

One year into the trial, presumably with the aim of examining whether personality traits were associated with smoking cessation, interviewers administered the 12-item Eysenck Personality Questionnaire (33) to 846 of the 1,204 attending men. Six items were used to quantify neuroticism, including, "Are you frequently 'lost in thought' even when supposed to be taking part in a conversation?" and an additional 6 items were used to assess extraversion, including, "Are you happiest when you get involved in some project that calls for rapid action?" The interviewers allocated 1 point for each positive response, −1 points for each negative response, and no points for answers that could not be clearly classified as either a positive or negative response. The possible scores on either factor therefore ranged from 6 to −6, with a higher score indicating a higher level in each personality domain. This version of the Eysenck Personality Questionnaire has a long research pedigree and has been incorporated into longer, contemporary neuroticism and extraversion scales. Abbreviated versions of the Eysenck Personality Questionnaire had high correlation coefficients with full versions of the questionnaire for both neuroticism (0.88) and extraversion (0.90) (34). There is also no apparent relationship between the 2 personality types ($r = -0.05$) (33), an observation that was also made in the present sample ($r = -0.03$).

Mortality ascertainment

Using the procedures of UK National Health Service Central Registry, we traced the vital status of 99% of the men to whom the personality questionnaire was administered. Based on the underlying cause, deaths were classified as resulting from coronary heart disease (*International Classification of Disease* Eighth or Ninth Revision codes 410–414 and Tenth Revision codes I20–I25), stroke (Eighth or Ninth Revision codes 430–438; Tenth Revision codes I60–I69), respiratory illness (Eighth or Ninth Revision codes 460–519; Tenth Revision codes J00–J99), or cancer (Eighth or Ninth Revision codes 140–209; Tenth Revision codes C00–C97).

Statistical analyses

In total, we had full data on personality type, mortality, and covariates for 832 of the 846 trial participants, and this represented our analytical sample. Neuroticism (≤−4, −3 to 0, or ≥1) and extraversion (≤−1, 0 to 2, or ≥3) scores were grouped into tertiles, with the lowest tertile serving as the referent. Using an approach we have taken elsewhere (35, 36), with no strong evidence that trial arm had a modifying effect on the personality type–mortality relationship, we pooled data, and effect estimates were adjusted for arm allocation.

Table 1. Hazard Ratios for the Associations of Neuroticism With Total Mortality, Coronary Heart Disease, Stroke, Respiratory Disease, and Cancer Among Study Participants ($n = 832$), 42-Year Mortality Surveillance in the Original Whitehall Smoking Cessation Trial, 1967–2012

Cause of Mortality and Adjustment	No. of Deaths	Neuroticism Tertiles						*P* for Linear Relation	*P* for Quadratic Relation
		Low ($n = 311$)		Medium ($n = 293$)		High ($n = 228$)			
		HR	95% CI	HR	95% CI	HR	95% CI		
Total mortality									
Age-adjusted	781	1.00	Referent	0.94	0.80, 1.11	1.03	0.86, 1.23	0.84	0.30
Multiply adjusted[a]	781	1.00	Referent	1.02	0.86, 1.21	1.08	0.90, 1.30	0.43	0.79
Multiply adjusted (20-year follow-up)[a]	372	1.00	Referent	1.00	0.77, 1.28	1.24	0.95, 1.61	0.13	0.33
Coronary heart disease mortality									
Age-adjusted	215	1.00	Referent	0.78	0.57, 1.09	1.14	0.82, 1.58	0.56	0.04
Multiply adjusted[a]	215	1.00	Referent	0.88	0.63, 1.23	1.23	0.87, 1.72	0.30	0.12
Multiply adjusted (20-year follow-up)[a]	129	1.00	Referent	0.95	0.62, 1.46	1.23	0.79, 1.90	0.40	0.42
Stroke mortality									
Age-adjusted	66	1.00	Referent	0.95	0.54, 1.65	0.91	0.48, 1.70	0.75	0.98
Multiply adjusted[b]	66	1.00	Referent	1.27	0.70, 2.30	1.15	0.59, 2.22	0.63	0.54
Respiratory disease mortality									
Age-adjusted	139	1.00	Referent	1.14	0.78, 1.68	1.03	0.67, 1.61	0.83	0.50
Multiply adjusted[a]	139	1.00	Referent	1.19	0.79, 1.80	1.05	0.66, 1.66	0.80	0.41
Multiply adjusted (20-year follow-up)[a]	41	1.00	Referent	1.52	0.66, 3.47	1.60	0.69, 3.69	0.28	0.60
Cancer mortality		1.00	Referent	0.96	0.71, 1.31	0.98	0.70, 1.38	0.88	0.84
Age-adjusted	219	1.00	Referent	0.96	0.71, 1.31	0.98	0.70, 1.38	0.88	0.84
Multiply adjusted[a]	219	1.00	Referent	0.97	0.70, 1.34	0.99	0.69, 1.41	0.93	0.86
Multiply adjusted (20-year follow-up)[a]	113	1.00	Referent	0.90	0.57, 1.42	1.12	0.69, 1.81	0.70	0.42

Abbreviations: CI, confidence interval; HR, hazard ratio.

[a] Multiple adjustment is adjustment for age, employment grade, number of cigarettes smoked per day at the start of the trial, smoking habit at administration of the personality questionnaire (year 1 of trial), physical activity level, arm of smoking trial, body mass index, systolic blood pressure, plasma cholesterol level, height-adjusted forced expiratory volume in 1 second, and disease at study entry.

[b] There were only 24 stroke deaths in the first 20 years of follow-up, which was insufficient for subgroup analyses.

Hazard ratios with accompanying 95% confidence intervals were computed using Cox's proportional hazards regression model (37) with calendar time as the time scale. Participants were censored at date at death, date at loss to follow-up, or end of follow-up (September 30, 2012), whichever came first. Hazard ratios were initially adjusted for age and thereafter for a series of covariates: employment grade, smoking habit (number of cigarettes smoked per day at the start of the trial and smoking status at the time of completion of the personality questionnaire after 1 year), physical activity level, body mass index, systolic blood pressure, plasma cholesterol, height-adjusted forced expiratory volume in 1 second, and disease at study entry. We performed separate significance tests for linear trend and quadratic-shaped association across the neuroticism and extraversion tertiles.

RESULTS

In the Web Tables 1 and 2, we show levels of covariates according to categories of personality type. Higher extraversion scores were associated with a slightly higher body mass index, but there were no associations with any of the other covariates. For neuroticism, findings were mixed: Somewhat lower levels of plasma cholesterol and systolic blood pressure were apparent in men with high neuroticism scores; conversely, disease at study entry was 20% more common in this group. In Table 1, we show the relationship between neuroticism and a range of mortality outcomes. In the 42-year follow-up period, there were 781 deaths (219 from cancer, 215 from coronary heart disease, 139 from respiratory disease, 66 from stroke, and 142 from other causes). There was little evidence that neuroticism was linked to death from all causes, stroke, respiratory disease, or cancer. Thus, in comparing the risk among participants in highest tertile of neuroticism with that among participants in the lowest tertile over the 42-year follow-up period, hazard ratios ranged between 0.78 (95% confidence interval: 0.57, 1.09) for stroke mortality and 1.60 (95% confidence interval: 0.69, 3.69) for respiratory disease mortality. When coronary heart disease was the endpoint of interest, in univariate analyses, there was a suggestion of a shallow U-shaped relationship with neuroticism such that the lowest risk was seen in men with moderate values, whereas those with low or

Table 2. Hazard Ratios for the Associations of Extraversion With Total Mortality, Coronary Heart Disease, Stroke, Respiratory Disease, and Cancer Among Study Participants ($n = 832$), 42-Year Mortality Surveillance in the Original Whitehall Smoking Cessation Trial, 1967–2012

Cause of Mortality and Adjustment	No. of Deaths	Extraversion Tertiles						*P* for Linear Relation	*P* for Quadratic Relation
		Low ($n = 206$)		Medium ($n = 376$)		High ($n = 250$)			
		HR	95% CI	HR	95% CI	HR	95% CI		
Total mortality									
Age-adjusted	781	1.00	Referent	0.86	0.72, 1.02	0.95	0.79, 1.16	0.72	0.07
Multiply adjusted[a]	781	1.00	Referent	0.89	0.74, 1.07	0.94	0.77, 1.14	0.60	0.24
Multiply adjusted (20-year follow-up)[a]	372	1.00	Referent	0.87	0.67, 1.12	1.04	0.79, 1.36	0.72	0.14
Coronary heart disease mortality									
Age-adjusted	215	1.00	Referent	0.68	0.49, 0.95	1.00	0.71, 1.41	0.83	0.006
Multiply adjusted[a]	215	1.00	Referent	0.71	0.50, 1.00	0.99	0.69, 1.40	0.90	0.02
Multiply adjusted (20-year follow-up)[a]	129	1.00	Referent	0.71	0.46, 1.10	0.98	0.63, 1.54	0.95	0.07
Stroke mortality									
Age-adjusted	66	1.00	Referent	0.74	0.39, 1.38	0.97	0.51, 1.87	0.98	0.26
Multiply adjusted[b]	66	1.00	Referent	0.76	0.41, 1.42	1.00	0.52, 1.92	0.93	0.29
Respiratory disease mortality									
Age-adjusted	139	1.00	Referent	0.78	0.51, 1.19	1.08	0.69, 1.68	0.61	0.09
Multiply adjusted[a]	139	1.00	Referent	0.86	0.55, 1.36	1.13	0.71, 1.80	0.49	0.25
Multiply adjusted (20-year follow-up)[a]	41	1.00	Referent	0.52	0.24, 1.15	0.90	0.41, 1.99	0.85	0.08
Cancer mortality									
Age-adjusted	219	1.00	Referent	1.16	0.82, 1.62	0.94	0.64, 1.39	0.96	0.20
Multiply adjusted[a]	219	1.00	Referent	1.12	0.79, 1.58	0.89	0.60, 1.32	0.47	0.22
Multiply adjusted (20-year follow-up)[a]	113	1.00	Referent	1.10	0.68, 1.79	1.11	0.65, 1.87	0.73	0.80

Abbreviations: CI, confidence interval; HR, hazard ratio.

[a] Multiple adjustment is adjustment for age, employment grade, number of cigarettes smoked per day at the start of the trial, smoking habit at administration of the personality questionnaire (year 1 of trial), physical activity level, arm of smoking trial, body mass index, systolic blood pressure, plasma cholesterol level, height-adjusted forced expiratory volume in 1 second, and disease at study entry.

[b] There were only 24 stroke deaths in the first 20 years of follow-up, which was insufficient for subgroup analyses.

high scores had slightly higher rates (for quadratic association, $P = 0.04$); however, this became less evident after multiple adjustment for covariates. When extraversion was the exposure of interest (Table 2), U-shaped relationships were again apparent with total, coronary heart disease, stroke, and respiratory disease mortality, although statistical significance at conventional levels was infrequent. Adjustment for several covariates, including health behaviors and biological risk indicators, did not markedly change this pattern of association.

When we examined the personality–mortality associations in the earlier (20-year) stages of follow-up, there was no suggestion that the strength of the relationship was greater than in the full (42-year) follow-up. This was confirmed statistically using a test of interaction of all personality–mortality associations according to duration of follow-up (all $P \geq 0.08$).

DISCUSSION

In the present study in which we utilized data from a smoking cessation trial in cohort study analyses, there was some evidence that opposing ends of the extraversion spectrum were associated with a modestly elevated risk of coronary heart disease and respiratory disease mortality. That our point estimates did not attain statistical significance in any analyses and were of modest magnitude, however, generally points to a null set of results for both extraversion and neuroticism in relation to a range of mortality outcomes. When we examined the personality–mortality associations in the earlier stages of follow-up, the strength of the relationship was no greater than in the full follow-up, which suggests little support for reverse causation.

Comparison with other studies

Our finding of no clear relationship between total mortality and either personality dimension is supported by a recent meta-analysis on this topic in which the authors aggregated individual-level data from 7 prospective studies (3). In another individual participant meta-analysis of hitherto unpublished studies which featured the 5 major personality types, higher

levels of neuroticism were related to coronary heart disease only, and higher levels of extraversion were associated with stroke only (5). Elsewhere, in accordance with our own findings, investigators in some studies have found little of evidence of a relationship of either extraversion or neuroticism with coronary heart disease (4, 38), stroke (14, 38), or cancer (6).

Study strengths and limitations

Although our study has important strengths—the unusually long duration of follow-up and the high proportion of deaths—it is not without its limitations. We quantified only 2 of the "big 5" personality types, so it is possible that associations with our chronic disease endpoints may have been evident with the remaining types. There was essentially no correlation between our 2 personality domains, which supports the notion that neuroticism and extraversion are separate psychological entities, but this does not rule out the possibility of misclassification of these phenotypes. The fact that this was a study of smokers—at least at trial induction before some members stopped smoking—means that, in principle, there should be no variation in the habit across personality groupings besides possible differences in the numbers of cigarettes smoked per day. Personality was, however, quantified 1 year into the trial after some men had quit (see Web Figure 1), so there were in fact individual differences in the habit. We therefore adjusted our hazard ratios for the number of cigarettes usually smoked at baseline, as well as for smoking status at year 1. We sampled male government workers who, in addition to being smokers, were otherwise classified as being at high risk cardiovascular disease, so the extent to which our findings are generalizable to women, lower risk populations, and a wider social strata is unclear. However, given that we explored risk factor–disease associations rather than simply risk factor or disease prevalence or incidence, we see no strong rationale for such a lack of external validity. This has recently been confirmed in comparative analyses of the original Whitehall II Study relative to general population-based cohort studies (39). Although the most favorable levels of risk factor prevalence and disease incidence were apparent in the Whitehall II Study (the "healthy worker effect"), risk factor–disease associations were essentially the same, and often identical, across studies. This is as expected because the influence of causal factors should not vary by population structure. Similarly, we have no reason to think that we cannot apply the results from the Whitehall Study to other studies in this field. In conclusion, in the present study of men, there was little evidence of a robust association of either neuroticism or extraversion with a range of cause-specific mortality outcomes accumulated over an extended period of follow-up.

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