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Low Conscientiousness and Risk of All-Cause, Cardiovascular and Cancer Mortality over 17 Years: Whitehall II Cohort Study

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Abstract

Objective—To examine the personality trait conscientiousness as a risk factor for mortality and to identify candidate explanatory mechanisms.

Methods—Participants in the Whitehall II cohort study (N = 6800, aged 34 to 55 at recruitment in 1985) completed two self-reported items measuring conscientiousness in 1991–1993 ('I am overly conscientious' and 'I am overly perfectionistic', Cronbach's $\alpha = .72$), the baseline for this study. Age, socio-economic status (SES), social support, health behaviours, physiological variables and minor psychiatric morbidity were also recorded at baseline. The vital status of participants was then monitored for a mean of 17 years. All-cause and cause-specific mortality was ascertained through linkage to a national mortality register until January 2010.

Results—Each 1 standard deviation decrease in conscientiousness was associated with a 10% increase in all-cause (hazard ratio [HR] = 1.10, 95% CI 1.003, 1.20) mortality. Patterns were similar for cardiovascular (HR = 1.17, 95% CI 0.98, 1.39) and cancer mortality (HR = 1.10, 95% CI 0.96, 1.25), not reaching statistical significance. The association with all-cause mortality was attenuated by 5% after adjustment for SES, 13% for health behaviours, 14% for cardiovascular risk factors, 5% for minor psychiatric morbidity, 29% for all variables. Repeating analyses with each item separately and excluding participants who died within five years of personality assessment did not change the results materially.

Conclusion—Low conscientiousness in midlife is a risk factor for all-cause mortality. This association is only partly explained by health behaviours, SES, cardiovascular disease risk factors and minor psychiatric morbidity in midlife.

Keywords

cohort study; conscientiousness; mortality; perfectionism; personality traits; socio-economic status

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Introduction

There is increasing interest in the public health implications of personality traits and other stable psychological factors as risk factors for disease and mortality (1–3). Conscientiousness is a personality trait describing personal dispositions, such as self-efficacy, orderliness, dutifulness, achievement-striving, self-discipline and cautiousness (4–6). High scorers tend to be organized, follow instructions carefully, adhere to medical regimens, strive to achieve goals, and are more likely to internalise societal norms which guide their behaviour (6, 7). Low scorers are described as being more disorganized, reckless, laid back and prefer not to make long-term plans (8). Although conscientiousness is considered to be a stable personality trait (9), increases in the mean level of conscientiousness have been observed across the adult life course, which is described as a maturity effect (10).

An association between higher conscientiousness and longevity has been shown in several studies (11, 12), including the Terman Life-Cycle Study (1921) of gifted children (13) where the sample was fairly homogenous on cognitive ability and socio-economic status (SES) and therefore less obviously confounded by these variables. The association between low conscientiousness and poor prognosis or adverse health outcomes has also been observed in clinical studies on patients with chronic renal insufficiency (14), coronary heart disease (15) and those receiving Medicare (16). Adjustment for potential confounding and mediating factors does not appear to attenuate the association markedly (17–20). The effect sizes are comparable to those between SES and between IQ and mortality risk (3). A recent review based on 20 studies estimated the effect size to be consistent with a 1.25 times increase in relative risk of mortality per standard deviation decrease in conscientiousness (5). The relative risk tends to be larger in recent rather than pre-1993 studies, among patients vs. healthy adults, and for longer than shorter (<5 years) follow-up periods (5).

Several factors may link low conscientiousness with mortality risk (8, 21). Possible mediators which have been proposed include low SES (22–24), unhealthy behaviours (4, 23, 25), worse cardiovascular risk profiles including higher inflammation (23, 26, 27) and minor psychiatric morbidity (28). To date, however, no study has included all these possible mediators in a single study and considered disease-specific effects.

The purpose of our study was to evaluate the association between conscientiousness and all-cause and cause-specific mortality over a mean follow-up time of 17 years in the Whitehall II study, a large occupational cohort of British civil servants. We adjusted the analyses for a various sets of possible explanatory variables and examined their explanatory role in these associations. We also calculated the direct, indirect and total effects from low conscientiousness, via possible mediators, to mortality risk.

Methods

Study population and procedure

The Whitehall II cohort recruited 10308 participants (6895 male) at baseline (phase 1, 1985–1988) with a response rate of 73% from 20 London based Civil Service departments (29). The University College London Medical School Committee on the Ethics of Human Research provided ethical approval for the study. Phase 3 (1991–1993), the baseline for the present analysis, was composed of both a questionnaire, which included two self-reported items measuring conscientiousness, and a clinical examination. Informed consent was provided by all participants. The analytic sample comprised 6800 participants (age range 39 to 63) with data at phase 3 on conscientiousness, covariates, candidate mediators and vital status.

Measures

Conscientiousness—Two items were administered at phase 3 of the study, which measured two different components of conscientiousness: ‘I am over-perfectionistic’ and ‘I am over-conscientious’. Response options were presented as ordered categories: ‘very much like me’ (1), ‘fairly like me’ (2), ‘not really like me’ (3), ‘very unlike me’ (4). The two items were combined into a summed composite to reduce measurement error. The combined scale achieved satisfactory internal consistency reliability (range 2 to 8; Cronbach’s $\alpha = .72$).

Mortality—Participants were traced for mortality through the national mortality register kept by the National Health Services (NHS) Central Registry using the NHS identification number assigned to each British citizen. In our analysis, mortality follow-up began at the measurement of conscientiousness (1991–1993, Phase 3) and ended on the 31st January 2010.

Covariates and mediators—Covariates were age in 1991–1993 and sex, both considered confounding factors. Mediators were selected on the basis that they had been identified in previous studies or in dominant theories of personality and disease.

Socio-economic status (SES)—Employment grade was used as a marker of socio-economic status, classified as low, intermediate or high. The civil service grades cover a wide range of salaries (>10-fold difference from highest to lowest), and reflect social status and level of responsibility.

Social support—Participants were asked to report whether they visited, or were regularly visited by, friends and relatives. Response scores were averaged for friends and relatives, and range from 0 to 4 per month. Social support was also indexed by marital status, scored 1 if participants were married or co-habiting (vs. 0 if not married or co-habiting), and by divorce status, scored 1 if participants were divorced (vs. 0 if not divorced).

Psychiatric morbidity—Participants completed the General Health Questionnaire (GHQ-30), a previously validated and widely used measure of minor psychiatric morbidity, particularly psychological distress and anxiety (30). Participants are asked ‘Have you recently...’ in relation to 30 self-reported items, such as ‘Felt that life isn’t worth living’ and ‘Found at times you couldn’t do anything because your nerves were too bad’? Total scores range from 0 to 30.

Health behaviours—Cigarette smoking was classified into separate categories for never smoker (the reference category), current regular smoker and ex-smoker. Alcohol units consumed in the previous week were categorized into abstainers, moderate drinkers (representing 1–14 units/week in women and 1–21 units/week in men; the reference category) and heavy drinkers (representing >14 units in women and >21 units in men). Dietary quality was assessed using the frequency of fresh fruits and vegetables consumed, on an eight-point scale ranging from seldom/never, to two or more times a day. Duration of moderate/vigorous physical activity was recorded in hours per week. Missing data on health behaviours was replaced with the last known value.

Cardiovascular disease (CVD) risk factors—Body Mass Index (BMI) was categorized according to the WHO criteria (31): BMI < 18.5 = underweight; BMI 18.5–24.99 = normal weight (the reference category); BMI 25–29.99 = overweight; BMI ≥ 30 = obese. Blood pressure (systolic and diastolic) was measured as part of the clinical examination, measured twice in a sitting position after 5 minutes rest with the Hawksley random-zero

sphygmomanometer. Measured blood pressure was considered the average of these two measures. As part of the clinical examination, fasting serum was collected between 8am and 1pm and stored at -70°C until analysis. A high-sensitivity immunonephelometric assay in a BN ProSpec nephelometer (Dade Behring, Milton Keynes, UK) was used to record CRP values. IL-6 was recorded using a high-sensitivity ELISA assay. Values lower than the detection limit (0.154 mg/L for CRP and 0.08 pg/mL for IL-6) were assigned a value equal to half the detection limit. Intra- and inter-assay coefficients of variation were 4.7% for CRP, and 7.5% for IL-6. C-reactive protein and interleukin-6 values were natural log-transformed prior to analysis, no constant added.

Data analysis

We first assessed the differences in all covariates, using t-tests for continuous variables and chi-square tests for categorical variables, between those who were alive and those who had died at the end of the follow-up period and between three categories of conscientiousness.

Cox regression with follow-up time as the time scale was used to evaluate the association between the conscientiousness scale and mortality risk, using SPSS version 19. The assumption of proportional hazards was tested by creating a new time-varying covariate ($\ln(T) \times \text{conscientiousness}$) where T was the follow-up time since personality assessment. This variable was not significant ($p = .88$) when entered into a model containing age, sex and conscientiousness, suggesting that the proportional hazards assumption was not violated.

Preliminary analyses screened for interaction terms between conscientiousness and sex or SES. The interaction terms suggested that the association was not significantly different in men and women (p for interaction = .96), and for different employment grades (p for interaction = .10). Therefore, we did not separate the sample into groups according to sex or employment grade.

Analyses were conducted for all-cause mortality and major causes of death (CVD and cancer). We first assessed the association between low conscientiousness and all-cause mortality risk in a model adjusted for age and sex (model 1). Subsequently, the extent to which this association was attenuated was examined by adding SES, health behaviours, biological risk factors, and minor psychiatric morbidity, first sequentially and then simultaneously, to the model adjusted for age and sex (model 1). The attenuation from model 1 attributed to the group of possible mediators under consideration was calculated using the formula " $100 \times (\beta_{\text{Model 1}} - \beta_{\text{Model 1+covariates}}) / (\beta_{\text{Model 1}})$ ". This approach is used widely in epidemiology and is broadly equivalent to testing for mediation, since the percentage attenuation estimates the proportion of the association that is explained by the proposed mediator. An alternative approach is to model the variables explicitly in a hypothesized causal chain, using structural equation modelling combined with Cox regression analysis (32). This involves calculating the direct, indirect and total effects using the MODEL INDIRECT feature of Mplus (version 6.1). The indirect effects are calculated as products of the regression coefficients from the path Cox regression coefficients, which are then exponentiated to obtain the hazard ratio (32). The possibility that effects ran in different directions for different items was tested by repeating models for each item separately. Concerns about reverse causality were addressed by repeating the analysis after excluding participants who died within 5 years of personality assessment. Cox regression models were then repeated for CVD and cancer mortality using SPSS.

Results

Sample description

Compared to the 6800 participants included in the present analysis, the 3508 participants excluded from the analytic sample were slightly older (44.7 vs. 44.3 years at recruitment, $p = .002$) and comprised more females (38.9 vs. 30.1% female, $p < .001$). Descriptive statistics for the analytic sample are shown in Table 1. During the follow-up period (mean 17.26 years, $SD = 2.15$) a total of 468 (6.9%) of participants died. The cause of death was not known for five participants. Of the 463 participants with known cause of death, 125 (27.0%) were CVD deaths and 227 (49.0%) were cancer deaths. For the CVD/cancer mortality models, two/five participants were additionally excluded automatically from the models, because they were censored before the event.

Lower conscientiousness was associated with higher blood pressure, being male (vs. female), seeing fewer friends or relatives, and heavy alcohol drinking (Table 1).

Association between conscientiousness and mortality

Each 1 SD decrease in conscientiousness was associated with a 10% increase in all-cause mortality risk ($HR = 1.10$, 95% CI 1.003, 1.20). The association was only slightly attenuated when controlling for each set of possible mediators. Adjustment for SES attenuated the association by 5%, health behaviours by 13%, biological factors by 14%, minor psychiatric morbidity by 5% (Table 2). Similar patterns of associations were observed for CVD mortality ($HR = 1.17$, 95% CI 0.98, 1.39) and cancer mortality ($HR = 1.10$, 95% CI 0.96, 1.25), but they were not statistically significant partly due to a lack of statistical power.

Results from the structural equation model are shown in Table 3. The model includes low conscientiousness, covariates and all proposed mediators. To facilitate convergence, BMI was treated as continuous because the underweight category contained relatively few participants. To reduce multicollinearity between systolic/diastolic blood pressure, the worst (higher) z score for each measure was retained (33). The effects are decomposed into indirect (mediating) effects, which are summed to provide the total indirect effect (32). To illustrate, the sum of all the indirect effects is $\beta = 0.032$ ($HR = 1.03$). The direct effect is $\beta = 0.063$ ($HR = 1.07$). The combined direct and indirect effect is $\beta = 0.095$ ($HR = 1.10$), consistent with results from Cox regression analysis.

Sensitivity analyses showed that the all-cause mortality association was very similar when analyses were repeated using each of the two items separately. Results did not change materially after excluding participants who died within five years of personality assessment, mitigating concerns about reverse causality (available on request).

Discussion

In a large sample of middle-aged British working adults, a 1-SD lower conscientiousness score was associated with a 10% higher risk of all-cause mortality over a mean follow-up period of 17 years. Socio-economic status (SES), social support, health behaviours, biological pathways and minor psychiatric morbidity only partially mediated the association. The effects were similar for CVD and cancer mortality although these analyses had less statistical power and the results were statistically non-significant.

Strengths of the study include the large sample size and follow-up period which are larger and longer than in many previous studies of conscientiousness and survival (12–14, 16–20, 23, 34, 35). Personality trait stability peaks at the third and fourth decades of life (36) and is considered relatively stable from age 30 (37). Comparatively few studies have measured

conscientiousness in adulthood and subsequent mortality risk, particularly in a large cohort such as Whitehall II. We were able to control for a wide range of possible mediators, which have been proposed in previous studies, but not been available for analysis collectively. We were able to compare competing explanatory variables, by calculating the percentage attenuation observed in the association after adjustment for different kinds of candidate mediators. The causal sequence was specified according to dominant models of personality and health (21–23) which assume that personality influences adult SES rather than vice versa. We cannot rule out the possibility that SES might have influenced the expression of conscientiousness later in time, because conscientiousness and SES were both measured cross-sectionally. It is possible that work-related stressors may influence the expression of personality traits, a research question that has been identified as a priority area for future study (38). The apparent trend showing more GHQ cases in the *more* conscientious group could be driven by the larger proportion of participants responding ‘very like me’ to both items (N = 175; 11.8% of GHQ cases) compared to those responding ‘very unlike me’ to both items (N = 19; 1.3% of GHQ cases). The combination of excessive conscientiousness, perfectionism, anxiety and depression may indicate psychopathology or personality disorder (39).

Brief measures of personality traits have many advantages (40), as may single-item measures, given that results were similar for each of the two items separately or when combined. There are clear disadvantages however, since brief measures cannot capture the full construct of conscientiousness (40). The brief scale used in our study does not correspond to longer conscientiousness scales as measured in the big five. It does however, have face validity and what has been termed the ‘ultimate validity’ of a psychological test (41) – it predicts mortality risk. Pathological levels of perfectionism may overlap with neuroticism (e.g. concerns over trivial mistakes, excessive self-criticism), but we were not able to control for other personality traits or personality disorders. This is a further limitation, although we found little evidence that minor psychiatric morbidity explained the association, and GHQ scores share genetic variance with neuroticism (28). Our results using a global measure of conscientiousness differ from those of a recent study, where perfectionism concerning self-expectations was associated with *increased* risk of mortality, but perfectionism concerning others’ expectations and socially prescribed perfectionism showed no association (42). In the same model, conscientiousness was associated with decreased mortality risk. Similar results in a recent study of diabetic patients pointed to self-oriented perfectionism decreasing mortality risk (43). Additional limitations of our study include the unusual term ‘overly’ which preceded both items, which could influence how participants responded. Civil servants may have interpreted these questions in relation to their occupational responsibilities, rather than their natural ‘shoes off’ self. Despite these limitations, the combined measure was associated with mortality, supporting its predictive validity.

Our study is among the first with the specific aim to describe and explain the association between single-item measures of low conscientiousness in midlife and survival in a large sample of British civil servants. Other studies have utilized measures of personality in childhood (11, 13), in late adulthood and old age (35), or among specific patient groups with existing disease (14, 15). We measured conscientiousness by combining two questionnaire items in order to reduce measurement error, but longer inventories would bring other advantages, such as allowing analysis of more specific ‘facets’ of conscientiousness (40). The recent meta-analytic review of conscientiousness and mortality risk found stronger effects for the achievement (persistence, industriousness) and order (organization, discipline) facets (5) which measure different components of the overall trait. Finally, we were not able to screen for interactions with other personality traits in the big five model. For example, data from the Midlife Development Study (44) suggested that

conscientiousness was protective of mortality risk - but only at the higher levels of agreeableness.

We found no evidence of effect modification by SES, in contrast to the Terman cohort where socioeconomic 'success' interacted with conscientiousness to predict mortality [34]. Participants high in conscientiousness but low in success were more likely to die younger in that study, although the cohort had high mean levels of cognitive ability and SES which could influence the meaning of 'success' and its relationship to mortality. In contrast, the Whitehall II cohort covers a wide range of SES, with a greater than 10-fold difference between the highest and lowest salary.

From a life course perspective, conscientiousness has often been treated as a stable trait, which is associated with educational attainment and career success, and with healthier behaviours in early adulthood. There is growing evidence however, that conscientiousness and other personality traits do change over time (45), having both within- and between-person variation. Mean level of conscientiousness, for example, are known to rise over adulthood (45). Our data point to health behaviours as partial mediators of the all-cause mortality association, consistent with several recent studies (22, 23). Conscientious individuals may internalise social norms about healthy behaviour, including public health advice, or find advice from health professionals easier to follow diligently (7, 27). Those who are less conscientious may find it difficult to follow health advice, or to organize activities in a way that promotes health. We emphasise the importance of collecting detailed measures of health behaviour, particularly the four major unhealthy behaviours: smoking, heavy alcohol use, poor diet and physical inactivity (46) in attempting to understand how low conscientiousness might influence mortality risk. Other health behaviours may be relevant (4), such as adherence to medical regimens and attending screening appointments (47). Adults with low conscientiousness may find it more difficult to adopt public health messages about health behaviours. Our results suggest that other mechanisms are involved in the association however, beyond health behaviours. These mechanisms may include immune system functioning (47), genetic factors (8), neurophysiological mechanisms (5) or personality disorders and psychopathology (39).

In summary, a brief measure of conscientiousness administered at midlife was associated with all-cause mortality risk. The association may arise partly because low conscientiousness is a strong risk factor for unhealthy behaviours (25), low SES (22, 23), or through shared common causes. In our cohort, conscientiousness appeared to influence mortality risk through several different pathways, although none of these pathways offered a full explanation.

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Acronyms

BMI	body mass index
CRP	C-reactive protein
CHD	coronary heart disease
CVD	cardiovascular disease
DBP	diastolic blood pressure
ELISA	enzyme-linked immunosorbent assay
GHQ	General Health Questionnaire
HR	hazard ratio
IQ	Intelligence Quotient
IL-6	Interleukin-6
NHS	National Health Service
SD	standard deviation
SES	socio-economic status
SBP	systolic blood pressure
WHO	World Health Organisation

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Table 1

Descriptive statistics as a function of conscientiousness profile and vital status (N=6800)

	Alive (N = 6332)	Died (N = 468)	Least conscientious (N = 1676)	Intermediate conscientious (N = 3742) ^d	Most conscientious (N = 1382)	p ^c
	Age-adjusted mean ^b (95% CI)		M (SD)	M (SD)	M (SD)	
Low conscientiousness ^d (SD)	.00 (-.02, .03)	.10 (.01, .19)	1.35 (0.48)	-0.07 (0.36)	-1.39 (0.36)	<.001
Age at baseline (range 39, 63)	49.3 (49.1, 49.4)	52.7 (52.1, 53.2)	49.3 (6.0)	49.6 (6.1)	49.5 (6.2)	.26
Physical activity (hours/week)	3.5 (3.5, 3.6)	3.3 (2.9, 3.6)	3.4 (3.9)	3.6 (4.0)	3.6 (4.1)	.12
Systolic blood pressure (mmHg)	120.3 (120.0, 120.7)	122.9 (121.6, 124.1)	121.2 (13.9)	120.5 (13.4)	119.7 (13.5)	.003
Diastolic blood pressure (mmHg)	79.7 (79.5, 79.9)	81.1 (80.3, 82.0)	80.4 (9.5)	79.8 (9.3)	79.0 (9.1)	<.001
C-reactive protein ^e (mg/L)	0.89 (0.86, 0.91)	1.20 (1.08, 1.34)	1.82 (2.83)	1.80 (3.48)	2.10 (6.45)	.47 ^e
Interleukin-6 ^e (pg/mL)	1.49 (1.47, 1.52)	1.81 (1.72, 1.91)	1.93 (2.73)	1.90 (1.95)	1.99 (2.64)	.93 ^e
	Age-adjusted % ^b (95% CI)		N (%)	N (%)	N (%)	p ^c
Male	69.7 (68.6, 70.8)	70.0 (65.3, 74.6)	1244 (74.2)	2617 (69.9)	889 (64.3)	<.001
Low socio-economic status (SES) ^f	15.6 (14.7, 16.5)	20.1 (16.1, 24.1)	286 (17.1)	578 (15.4)	212 (15.3)	.28
Married or co-habiting	77.1 (76.1, 78.1)	69.5 (64.7, 74.2)	1318 (78.6)	2874 (76.8)	1024 (74.1)	.01
Divorced	6.8 (6.2, 7.4)	8.2 (5.5, 10.9)	115 (6.9)	258 (6.9)	98 (7.1)	.96
< 2 friends or relatives seen monthly ^f	15.8 (14.9, 16.7)	15.2 (11.5, 18.9)	246 (14.7)	566 (15.1)	259 (18.7)	.003
Current regular smoker	12.6 (11.8, 13.4)	22.5 (18.3, 26.7)	221 (13.2)	514 (13.7)	168 (12.2)	.33
Heavy alcohol drinker	15.4 (14.5, 16.3)	24.0 (19.6, 28.5)	322 (19.2)	586 (15.7)	175 (12.7)	<.001
Poor diet ^f	37.7 (36.5, 38.9)	44.1 (39.1, 49.1)	643 (38.4)	1456 (38.9)	497 (36.0)	.15
Underweight	0.9 (0.7, 1.1)	1.4 (0.2, 2.6)	12 (0.7)	33 (0.9)	19 (1.4)	.17
Overweight	38.0 (36.8, 39.1)	39.8 (34.8, 44.7)	650 (38.8)	1423 (38.0)	507 (36.7)	.49
Obese	9.0 (8.3, 9.7)	15.0 (11.4, 18.7)	164 (9.8)	356 (9.5)	118 (8.5)	.45
GHQ-30 case (>=5 points)	21.9 (20.9, 23.0)	21.0 (16.7, 25.3)	262 (15.6)	801 (21.4)	417 (30.2)	<.001

Note.

^a Tertile cut points are 4 and 5 (range 2 to 8), with a large intermediate group.

^b Adjusted for age, allowing for age differences in the two groups (age at baseline is unadjusted).

^c p values are for linear trends (analysis of variance or covariance for continuous variables, χ^2 test or logistic regression for categorical variables).

^d Standardised low conscientiousness scale.

^e C-reactive protein and Interleukin-6 were natural log transformed, p value refers to log value.

^f In Cox models, SES, number of friends/relatives seen and diet were treated as continuous.

Table 2

Hazard ratios (95% confidence intervals) for all-cause associated with a one unit increase in low conscientiousness

Model description	All-cause mortality (468 deaths/6800 participants)	
	HR (95% CI)	% attenuation ^a
Model 1: adjusted for age and sex ^b	1.10 (1.003, 1.20)	
Model 2 (socio-economic status): Model 1 +employment grade	1.09 (0.998, 1.19)	5.2
Model 3 (social support): Model 1 +social support from friends/relatives, divorce and marital status	1.10 (1.01, 1.21)	-6.0
Model 4 (health behaviours): Model 1 + current regular smoking, physical activity, diet, abstinent and heavy (vs. moderate) alcohol drinking	1.08 (0.99, 1.19)	13.1
Model 5 (biological risk factors): Model 1 +underweight/overweight/obesity (vs. normal), systolic and diastolic blood pressure, C-reactive protein and IL-6.	1.08 (0.99, 1.19)	13.7
Model 6 (minor psychiatric morbidity): Model 1 + GHQ score.	1.09 (0.998, 1.20)	4.6
Fully adjusted model	1.07 (0.98, 1.17)	28.6

Note.

^aThe percent attenuation refers to the beta coefficient, not the hazard ratio.

^bFor model 1, the associations with all-cause mortality for age and sex (male) are HR = 1.10 (95% CI 1.08, 1.11) and HR = 1.11 (95% CI .91, 1.35).

Table 3

Unstandardised model parameters decomposed into direct and indirect effects, using structural equation modelling

Indirect pathway	Indirect Effect	Hazard Ratio
Employment grade	0.002	1.002
Married or cohabiting (vs. not)	-0.002	0.998
Divorced (vs. not divorced)	0.000	1.000
Number of friends or relatives seen monthly	-0.002	0.998
Current regular smoker (vs. non-smoker)	0.003	1.003
Ex-smoker (vs. non-smoker)	0.002	1.002
Heavy drinker (vs. moderate drinker)	0.010	1.010
Abstinent drinker (vs. moderate drinker)	-0.002	0.998
Fruit/vegetable consumption	0.001	1.001
Physical activity	0.002	1.002
Body Mass Index	0.008	1.008
Blood pressure (highest z score)	0.004	1.004
GHQ score	0.007	1.007
Total of indirect effects	0.032	1.033
Direct effect	0.063	1.065

Note. In the structural equation model, BMI was treated as continuous (facilitating convergence) and blood pressure was the most unfavourable z-score for systolic or diastolic (reducing multicollinearity). All other variables were treated in the same way as for Cox Regression models.