



# Validity of the Grossarth-Maticek and Eysenck personality-stress model of disease: An empirical prospective cohort study



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## ABSTRACT

Personality-stress subtypes measured via the Short Interpersonal Reactions Inventory (SIRI) have been claimed to predispose to cancer, cardiovascular disease, or overall good health. We examined such associations in a 1993–1996 study on health risk factors in Australians aged over 50 years. 2197 women and 919 men completed the questionnaire, with nine subscores calculated. After a median 23.4 years, protocols were matched against dates/causes of death (1108 out of 3027 respondents with useable SIRI scores had died). Survival analysis tested for associations between subscores and mortality from all causes, mortality from cancers (30% of deaths), cardiovascular disease (23% of deaths), and other known causes (35% of deaths). Type 2 (CHD-prone) and Type 4 (healthy) scores were significantly associated ( $p < 0.05$ ) with all-cause and cardiovascular mortality but not with any-cancer mortality. Despite criticisms of the Grossarth-Maticek and Eysenck data, we found empirical support for some SIRI subtypes. In accord with the Grossarth-Maticek and Eysenck personality-stress model, and consistent with two previous SIRI studies, inverse associations of Type 4 (healthy) scores with all-cause mortality were found and also Type 2 scores predicted CVD mortality. However, no significant relationship was found between Type 1 scores and cancer mortality.

## 1. Background

The *Short Interpersonal Reactions Inventory* (SIRI) – a shortened version of the *Personality-Stress Inventory* (PSI) constructed by Grossarth-Maticek, Eysenck, Vetter, and Schmidt (1988), and further developed by Grossarth-Maticek and Eysenck (1990, 1991) measures a disease-prone personality typology. Beyond introversion-related stress (Saklofske & Eysenck, 2004), the personality-stress model proposes six disease-prone personality subtypes (measured via the SIRI/PSI), each having a characteristic pattern of responding to stressful situations (cf. Eskelinen & Ollonen, 2011; Eysenck, 1985, 1991; Friedman, 1990; Jokela et al., 2014; Kern & Friedman, 2011; Larsson et al., 1995; Lemogne et al., 2013; Orejudo, Froján & Malo, 2004, 2007). Characteristic styles of responding to stressful situations have been claimed to predict future morbidity and mortality as follows: Type 1 (Cancer-prone), Type 2 (Cardiovascular Disease [CVD]-prone), and Type 4 (Healthy/Protective).

According to this typology, Type 1 individuals tend to respond to stressful situations by suppressing emotional expression and denying strong emotional reactions. They are inhibited in expressing their needs and feelings, resulting in helplessness and depression. According to Eysenck (1991b, p. 54), “*The cancer-prone personality has often been described as appeasing, unassertive, overcooperative, overpatient, harmony seeking and conflict avoiding, and compliant and defensive...*” Whereas Type 1 individuals characteristically exhibit underarousal, Type 2 individuals react to stressful situations with heightened arousal, anger and aggression. Both Types 1 and 2 may experience stress as a function of ongoing emotional over-involvement with others and situations that invoke distress, unhappiness and disturbance. Type 3 is associated with ambivalence, whereas Type 4 individuals are purported to exhibit personal autonomy and consequently are better able to deal with stressful situations, associated with good health. Type 5 (rational/anti-emotional) has been classified by Schmitz (1992) as a disposition prone towards endogenous depression. In addition, a self-regulation

*Abbreviations:* BMI, Body Mass Index; CHD, Coronary Heart Disease; CI, Confidence Interval; CVD, Cardiovascular Disease; HR, Hazard Ratio; ICD, International Classification of Disease; NDI, National Death Index; SD, Standard Deviation; SIRI, Short Interpersonal Reactions Inventory

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dimension was proposed by Grossarth-Maticek and Eysenck (1995) to measure personal autonomy or independence (particularly in relation to emotional dependence). They reported that, “large samples of healthy men and women were tested and followed up to demonstrate high predictability of mortality from cancer, coronary heart disease and other causes of death from scores on the questionnaire.”

While these purported associations have been the subject of speculation about their veracity (Pelosi, 2019; see rebuttal by Eysenck, 1991a), three unrelated published studies provide independent empirical evidence as to whether the SIRI subscales can predict cancer and cardiovascular mortality (cf. Kreitler, 2019; Rosengren, Hawken & Ounpuu, 2004). A prospective study in France (Nabi, Kivimaki & Zins, 2008) with  $N = 14,445$  found no associations between SIRI scores and cancer mortality, but significant associations between Type 2 and Type 4 scores and cardiovascular mortality. For all-cause mortality, higher Type 2 (CVD-prone), Type 3 (“Ambivalent”) and Type 6 (“Anti-social”) scores were associated with higher risk. Previously, a case-control study in Japan (Nagano, Sudo, Kubo & Kono, 2001) with  $N = 785$  used SIRI scores to classify patients (with lung cancer or myocardial infarction) and healthy controls into mutually exclusive groups, with patients assigned to the type for which they scored highest. Risk of myocardial infarction was significantly less in those classified as Type 4 than in all other types combined (Odds Ratio 0.584, 95% CI 0.374–0.913) but the association with lung cancer was not significant. In addition, in an Iranian study of business managers with  $N = 94$ , Ghorbani, Watson and Morris (2000), p. 647) reported that, “Among the health types, the coronary-prone scale yielded the strongest pattern of results, displaying hypothesized relationships with three of four stress and four of five psychopathology measures....These data most importantly demonstrated that constructs developed in the West for understanding the role of personality in stress and health apparently have a cross-cultural validity in Iran as well.”

Although we are agnostic as to the validity of the SIRI subscales and their associations with morbidity and mortality, we have relevant data on the SIRI scale scores and mortality from a large prospective study conducted over 27 years, and believe these results should be made available as an independent test of the validity of the Grossarth-Maticek and Eysenck personality-stress model described above.

## 2. Studies and methods

### 2.1. Older Australians’ study

A self-report study into health status among older people was carried out between 1993 and 1996 (Kirk & Martin, 1998; Mosing, Medland & McRae, 2012). Participants gave informed consent to the data collection and storage. This project was approved in accordance with the National Health and Medical Research Council’s National Statement on Ethical Conduct in Human Research (<https://www.nhmrc.gov.au/about-us/publications/national-statement-ethical-conduct-human-research-2007-updated-2018>) by both the QIMR Berghofer Medical Research Institute Human Research Ethics Committee and (for the National Death Index search) the Australian Institute of Health and Welfare (AIHW) Ethics Committee.

Some 2281 pairs of twins aged over 50 years were invited to

participate (cf. Martin, Eaves, Kearsley & Davies, 1978). The combined questionnaire comprised psychological scales, lifestyle measures assessing smoking (Eysenck, 1991b), alcohol consumption (Grossarth-Maticek, Eysenck & Boyle, 1995a) and physical activity. Altogether, 71% of those approached completed the combined questionnaire. Respondents comprised 2197 women (response rate 75%) and 919 men (63%). The mean age of respondents was  $61.5 \pm 8.7$  years (range: 50–94 years). Cases with more than 10% of responses missing from the total 70 items in the combined scales were excluded, resulting in a final sample of 2844 individuals (1985 women and 859 men).

### 2.2. Short interpersonal reactions inventory

Among the psychological measures administered in the study was a short form of the SIRI. Since the primary aim of the study was to consider health issues affecting older people, items from SIRI Types 3 and 6 (not associated with disease proneness) were omitted. Type 1, Type 2 and Type 4 scales were reduced in length pursuant to the factor analysis by Roberts, Duffy and Martin (1995), with just five items retained for each of Types 1, 2, 4(a) (direct scored items) and 4(b) (reverse scored items). Type 4 was analysed separately as a complete construct (direct- and reverse-scored items combined), and also in terms of its separate subscales.

In addition, Self-regulation and Differentiation Scales were constructed by Grossarth-Maticek and Eysenck (1995) (cf. Grossarth-Maticek, Eysenck & Boyle, 2004; Kirk & Martin, 1998) in response to a reported difficulty in distinguishing Type 1 and Type 2 from each other (but not from Type 4) when responses are not interview-derived. The scales were translated for this study by two bilingual translators (George Landers and Bernd Kalinna) with subsequent back-translation to ensure equivalence (cf. Banville, Desrosiers & Genet-Volet, 2000). Translations aimed at making the items more interpretable to the target population while retaining the original intended meaning of each item. To further minimize repetition and reduce administration time, eight items with similar (but reversed) wording were deleted from the Differentiation Scale (see Table 1).

Scores for Type 1, Type 2, Type 4a, Type 4b, Type 5, Self-regulation, Inhibition, and Agitation, were computed from responses to the individual items by summing scores, coded as 1 = No, 2 = Don’t Know, 3 = Yes, or as 1 = Disagree, 2 = Not sure, 3 = Agree. Also, a combined Type 4 score was calculated [(Type 4a score) + (10 - Type 4b score)].

### 2.3. National Death Index search

Participants’ names and dates of birth were submitted to the Australian National Death Index (NDI) (2020) for matching against their records. NDI records contain information about deaths from 1980 onwards. Deaths outside Australia could not be matched through an NDI search, and in some cases (e.g., with common names or where date of birth was inaccurately estimated by relatives of the decedent), matches were not possible. Identifying information was matched against deaths occurring within Australian States and Territories up to the end of October 2017, for a median time period since completion of the questionnaire of 23.4 (SD = 0.66) years. Matching occurred using an

**Table 1**  
Summary statistics for SIRI scores.

	Type1 score	Type2 score	Type4a score	Type4b score	Self-Regulation score	Inhibition score	Agitation score	Type5 score	Type4 (combined)
N	2852	2846	2795	2767	2131	2713	2734	2772	2638
Mean	4.17	2.25	7.38	2.09	33.57	11.23	4.37	11.60	15.30
Median	4.00	2.00	8.00	2.00	34.00	12.00	3.00	12.00	16.00
Std. Deviation	2.96	2.44	2.11	2.27	6.16	3.57	4.10	3.43	3.31
Minimum	0	0	0	0	6	0	0	0	2
Maximum	10	10	10	10	46	18	16	20	20

algorithm based on date of birth, with family and personal names weighted for frequency of names within the index (i.e., a match for an uncommon name was given greater weight than a match for a common name). On receipt of the search results, they were ranked according to matching score and re-checked for their validity by experienced NDI data personnel.

For most deaths occurring before the end of 2016, an underlying cause of death and up to 12 other risk conditions present were reported. The reason for missing causes after that time was because information on the date of death was received or coded earlier than documentation of the actual causes of death. Causes of death were coded by the NDI using the *International Classification of Diseases*, either ICD-9 (up to 1996) or ICD-10 (1997 onwards). Only the ‘underlying cause of death’ (not ‘other conditions present’), was used in the cause-specific analysis. Causes were divided for the current analysis into three broad categories; cancers (malignant neoplasms: ICD-9 codes 140–208, ICD-10 codes C00–C97); cardiovascular diseases (CVD; diseases of the circulatory system: ICD-9 codes 390–459, ICD-10 codes I00–I99); and other known causes. Where no cause of death was available, the date of death was used in analysis of all-cause mortality and the case was censored at the date of death for the cause-specific analyses.

Survival analysis was based on scores and the date of death or censoring (recoded to age at death or at 21st October 2017). Initial analyses were conducted via SPSS (Version 22). This was used for data management, estimation of means and correlations and for survival analysis. However, because our recruitment emphasised twin-pairs, there was genetic overlap between many participants. Thus, to the extent that participants were genetically similar to each other (which varies according to heritability of characteristics under consideration), the effective number of independent observations was less than number of participants and standard errors for calculated statistics would have been underestimated. To overcome this problem, associations between scores and all-cause, any-cancer, any-cardiovascular, and other-cause mortality were tested using Cox regression in STATA (StataCorp LLC) with clustering by family to generate robust standard errors for the regression coefficients and confidence intervals for Hazard Ratios (HRs). Several specific and testable hypotheses can be assessed against our results:

**H1:** that individuals with higher Type 1 scores are more likely to be cancer prone.

**H2:** that individuals with higher Type 2 scores are more likely to be CVD-prone.

**H3:** that Type 4 is indicative of an overall “healthy” disposition.

### 3. Results and discussion

The distributions of scores are shown in Table 1 and Fig. 1, with all subtype pairs being significantly correlated (Table 2).

Results of survival analysis, adjusted for sex, are in shown in Table 3, and results after additional adjustment for body mass index (BMI), alcohol intake and smoking status in Table 4. Some 99% of deaths during the follow-up period took place more than one year, and 97% more than five years, after the initial questionnaire survey.

The survival analysis reveals that SIRI scores for Type 1, Type 2, Type 4b and combined Type 4 were significantly associated with all-cause mortality ( $p < 0.05$  before adjustment for multiple testing). The same scores showed significant associations with cardiovascular mortality, while both Type 4b and combined Type 4 remained significant after Bonferroni correction, ( $p_{\text{CORRECTED}} = 0.019$  and  $p = 0.0024$ , respectively). Higher scores were associated with HRs  $> 1.00$  (increased risk) except for combined Type 4 (as predicted), where the calculation had reversed the direction of its Type 4b component.

Our results do not provide support for H1 (that individuals with higher Type 1 scores are more likely to be cancer prone) with estimated HR = 1.006 (95% CI 0.968 to 1.046,  $p = 0.767$ ). However, there is

reasonable evidence for H2 (that individuals with higher Type 2 scores are more likely to be CVD-prone). When sex is the only additional predictor, the HR (for CVD) = 1.079 (per scale unit, 95% CI 1.019 to 1.143,  $p = 0.009$ ). Adding BMI, alcohol and smoking as additional predictors gives a similar HR (1.077, 95% CI 1.014 to 1.143,  $p = 0.016$ ), suggesting that the effects of Type 2 scores are not mediated by obesity, drinking or smoking.

There is also evidence for H3 (that Type 4 is indicative of an overall “healthy” disposition) as our results show that higher combined Type 4 scores are associated with significantly decreased all-cause mortality (HR = 0.974 per scale unit, 95% CI 0.955 to 0.994,  $p = 0.010$ , with sex as the only other predictor). Adding BMI, alcohol and smoking gives a similar HR = 0.975 (95% CI 0.954 to 0.996,  $p = 0.022$ ), again suggesting that the effects of personality-stress type are not due to obesity, alcohol consumption, or smoking. The effect of the ‘healthy’ Type 4 is most evident for cardiovascular disease; with approximately a 7.5% decrease in HR per unit (combined Type 4 scale), and the most significant associations out of all the tests ( $p = 2.62 \times 10^{-4}$ , and  $p = 6.26 \times 10^{-4}$  with sex, BMI, alcohol, and smoking included). The observed difference between extremes was substantial. The HR for cardiovascular mortality (from comparing the 19.5% of participants at or below 12 on the combined Type 4 scale against the 15.3% at or above 18 on that scale) was approximately two-fold (HR 0.496, 95% CI 0.318–0.774). In order to translate the hazard ratios into something more readily understood (e.g., specific mortality rates of high and low-scoring groups over time), a Kaplan-Meier survival plot (Fig. 2) compares individuals with combined Type 4 scores of 12 or less with those obtaining scores of 18 or more, thereby showing CVD survival against age.

Our findings for Type 4 are similar to those of the French study (Nabi et al., 2008), which showed a reduced risk for cardiovascular disease in individuals with higher Type 4 scores and an increased risk in those with higher Type 2 (CVD-prone) scores, and also to the Japanese study (Nagano et al., 2001). Evidently, the SIRI Type 4 scale measures a “healthy” personality-stress disposition which is negatively related to the long-term risk of cardiovascular disease. Our results are also congruent with those of Ghorbani et al. (2000) who demonstrated in an Iranian sample that the coronary-prone scale yielded the strongest predictive validity of stress/health-related variables. Their findings indirectly support the ability of the SIRI/PSI to predict CVD.

One caveat however is that administration of the SIRI scales is intended for use in face-to-face interviews, whereas our data were gathered via a questionnaire mailed out to participants. It is possible that this variation from the standard administration procedure may account for our failure to find the claimed association between the Type 1 personality-stress score and cancer mortality, although the corresponding expected associations were found for both Type 2 and Type 4 (see Grossarth-Maticek, Eysenck & Barrett, 1993; Grossarth-Maticek, Eysenck & Boyle, 1995b). In addition, it is possible that the reduction of number of items down to just five items per scale, may have reduced predictive validities which may account for the lack of an observed association between Type 1 and cancer mortality. From a psychometric perspective, and in accord with the Spearman-Brown prophecy formula, it is possible that short 5-item scales may have inadequate test-retest reliability and predictive validity (see Boyle, Saklofske & Matthews, 2015). Even so, some of the five-item scales did exhibit significant associations with cardiovascular (CVD) mortality.

In the present study, Type 1 scores were associated with all-cause, cardiovascular, and other-cause mortality, but not (in contrast to the predicted association) with cancer mortality. For Type 2 scores, associated with cardiovascular disease (CVD), an association was observed, although the more generalised “healthy” Type 4 scores exhibited an even stronger association. Type 4b, where higher scores were associated with greater risk of CVD mortality (and probably also with other-cause mortality), was based on reverse-worded items (see Table 1). Assent to these items (“Yes” or “Agree”) was scored more highly than

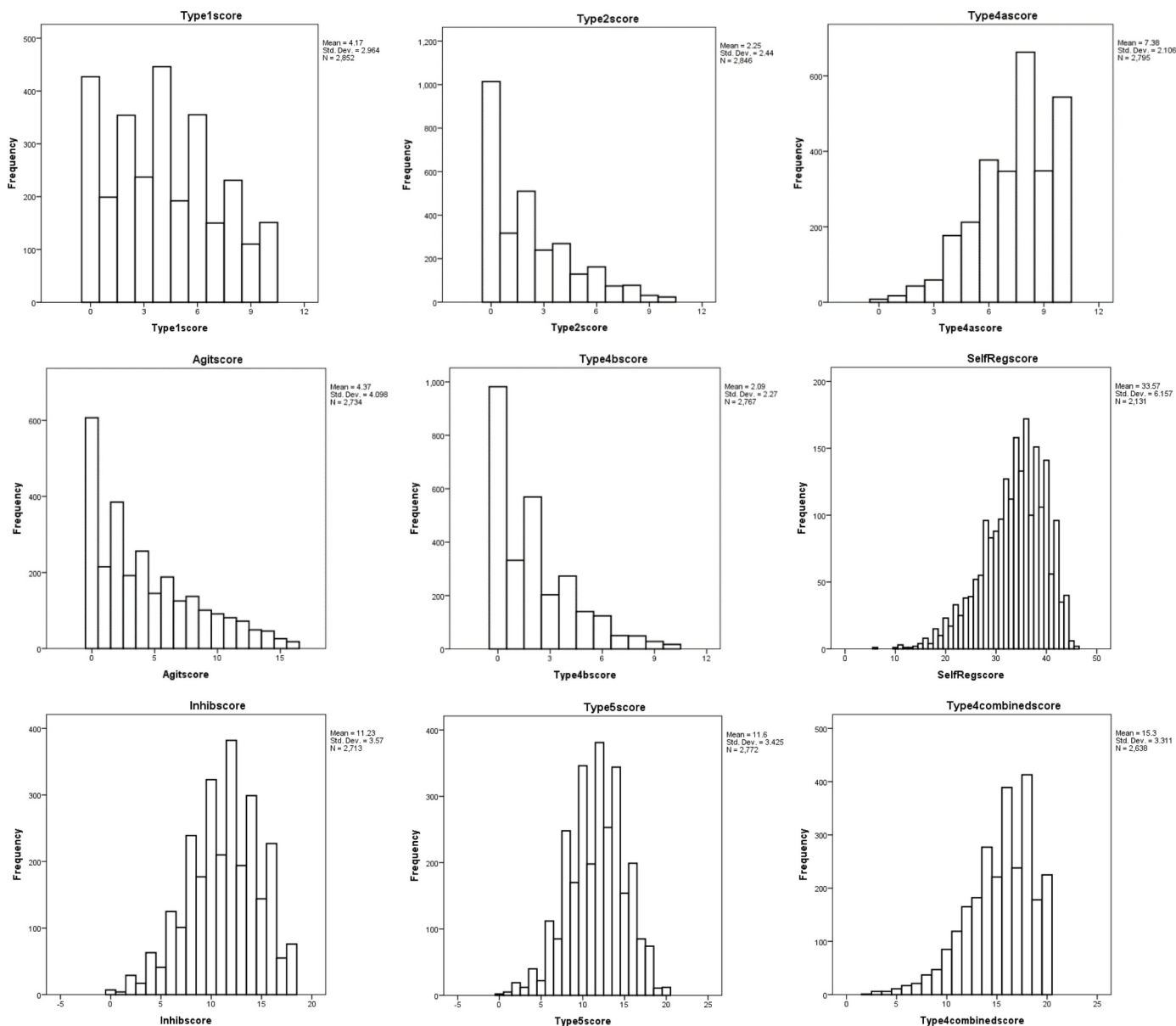


Fig. 1. Distributions of scores from SIRI scales.

rejection (“No” or “Disagree”). The other SIRI scores (Type 5, Self-regulation, Inhibition, Agitation) showed no significant associations with all-cause mortality or with any main causes of mortality.

In order to compare our results with the initial claims of disease associations, we have calculated the relative risk of dying during follow-up, and the risks of dying from cancer and from coronary heart disease (CHD), based on Tables 5, 6 and 7 in Ch. 6 of Eysenck (1991) – (Table 5).

The reported differences in risk are large for all-cause mortality (about 30 to 55-fold for Type 1 compared with Type 4 and about 24 to 55-fold for Type 2 against Type 4). Admittedly the assignment of people to the Type for which they scored highest may make some difference, but if we take our two extremes of the combined Type 4 score ( $\leq 12$  versus  $\geq 18$ ) as contrasting groups for CVD risk (the most favourable comparison in our data), the difference is only two-fold. Based on this, even for our most significant association, the effect on risk is far below that implied by the reported results from the three studies which form the basis of Grossarth-Matecek & Eysenck’s hypothesis (see Eysenck, 1991b, Ch. 6, Tables 5, 6 & 7).

Although we only report associations with mortality (and not

morbidity), and our results do not directly address causation, quite likely these psychological characteristics contributed as risks of disease, rather than *vice-versa* (see Grossarth-Matecek, Eysenck & Boyle, 2000). Firstly, the great majority of deaths in our cohort occurred more than five years after administration of the baseline questionnaire. Secondly, the associations were strongest for cardiovascular deaths whereas we would expect similar effects from each of these disease groups if poor health was causing variation in the personality-stress scores.

Even excluding part-whole correlations (Type 4 with its subtypes 4a and 4b, respectively), scores on the various SIRI scales exhibit some measurement overlap, ranging up to a maximum of 34% shared variance (Table 2). Type 1, Type 2 and Type 4b scores, which were moderately correlated with each other (rank correlations  $> 0.4$ ) each exhibited similar effects on mortality whereas the Agitation score (associated with Type 1, Type 2 and Type 4b scores) did not impact on mortality.

It is difficult to assess what the SIRI scales (especially those that gave significant results) are really measuring, but for at least the “healthiness” Type 4b scale (and to some extent for the Type 1 and Type 2 scales) the statements imply a perceived inability to change things for

**Table 2**  
Rank correlations between SIRI scores.

	Type 1 score	Type 2 score	Type 4a score	Type 4b score	Self-Regulation score	Inhibition score	Agitation score	Type 5 score	Type 4 combined
Type 1 score	rho 1.000 N 2852								
Type 2 score	rho 0.421 N 2757	1.000 2846							
Type 4a score	rho -0.119 N 2723	-0.134 2715	1.000 2795						
Type 4b score	rho <b>0.404</b> N 2684	<b>0.583</b> 2698	-0.137 2638	1.000 2767					
Self-Regulation score	rho -0.138 N 2092	-0.185 2093	<b>0.435</b> 2085	-0.232 2064	1.000 2131				
Inhibition score	rho <b>0.309</b> N 2653	0.097 2634	0.096 2611	0.074 2570	0.197 2032	1.000 2713			
Agitation score	rho 0.276 N 2668	<b>0.489</b> 2657	-0.116 2627	<b>0.488</b> 2585	-0.179 2052	-0.103 2597	1.000 2734		
Type 5 score	rho 0.178 N 2638	0.182 2640	0.092 2593	0.157 2571	0.227 2025	<b>0.351</b> 2527	0.085 2556	1.000 2772	
Type 4 combined score	rho <b>-0.344</b> N 2587	<b>-0.477</b> 2589	<b>0.742</b> 2638	<b>-0.728</b> 2638	<b>0.444</b> 2023	<b>0.020</b> 2487	<b>-0.398</b> 2501	-0.040 2477	1.000 2638

All correlations are statistically significant at  $p < 0.05$ . Correlations  $> 0.30$  are highlighted in bold.

the better. This may relate to stress associated with, for example, low employment-related control (Bosma, Marmot & Hemingway, 1997) that is not necessarily restricted to the workplace, but to the broader epidemiology of psychosocial risk factors for cardiovascular disease (Dimsdale, 2008; Glozier, Tofler, Colquhoun & Bunker, 2013; Okereke & Manson, 2017; Williams, Barefoot & Schneiderman, 2003), beyond the scope of this empirical report.

#### 4. Ethical statement

The study was granted ethical clearance by the Queensland Institute of Medical Research NHMRC Human Research Ethics Committee. The authors report no conflicts of interest.

**Table 3**

Survival analysis (Cox regression, using STATA with familial clustering), with sex as an additional predictor. HR, Hazard Ratio, per unit increase in score; 95% CI, 95% confidence intervals for Hazard Ratio.

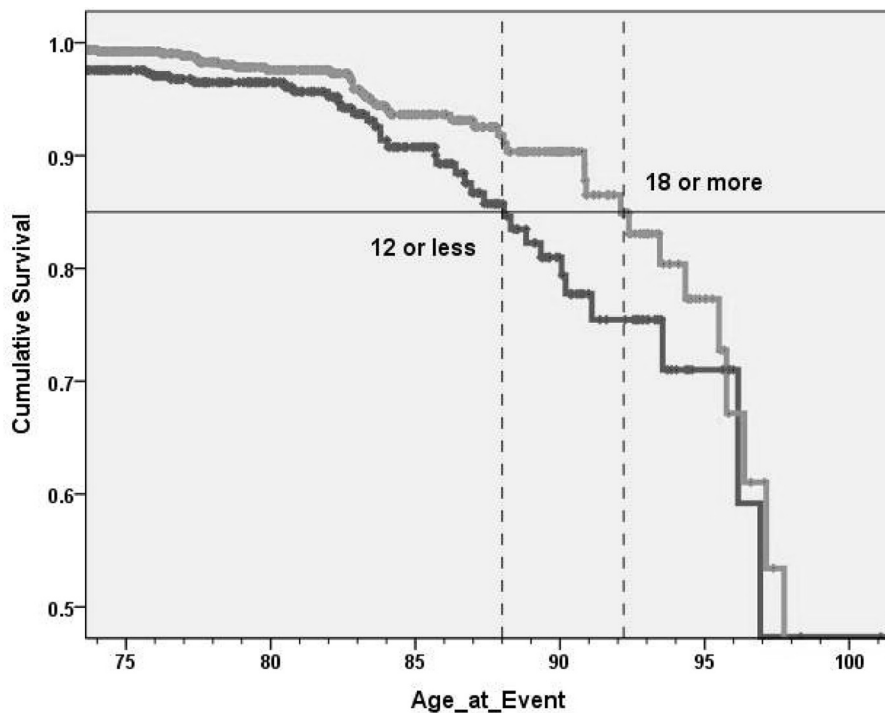
Cause of death	SIRI scale	N Total	N Deaths	HR	95% CI	p
ALL-CAUSE	Type 1	2817	1031	1.0231	1.0016 - 1.0451	0.035
	Type 2	2813	1010	1.0295	1.0026 - 1.0571	0.031
	Type 4a	2762	1005	0.9847	0.9559 - 1.0144	0.309
	Type 4b	2730	961	1.0372	1.0086 - 1.0665	0.010
	Self-regulation	2112	750	1.0046	0.9930 - 1.0162	0.439
	Inhibition	2680	968	0.9969	0.9790 - 1.0151	0.735
	Agitation	2705	982	1.0131	0.9977 - 1.0288	0.096
	Type 5	2735	995	0.9917	0.9731 - 1.0107	0.389
	Combined Type 4	2607	917	0.9739	0.9546 - 0.9937	0.0099
	ANY CANCER	Type 1	2817	311	1.0059	0.9676 - 1.0457
Type 2		2813	308	0.9944	0.9473 - 1.0438	0.820
Type 4a		2762	303	1.0305	0.9760 - 1.0879	0.278
Type 4b		2730	301	0.9553	0.9043 - 1.0091	0.102
Self-regulation		2112	246	1.0208	0.9998 - 1.0422	0.052
Inhibition		2680	296	1.0033	0.9713 - 1.0364	0.842
Agitation		2705	300	1.0011	0.9734 - 1.0296	0.939
Type 5		2735	299	0.9717	0.9405 - 1.0039	0.084
Combined Type 4		2607	287	1.0358	1.0000 - 1.0730	0.050
ANY CVD		Type 1	2817	235	1.0512	1.0053 - 1.0992
	Type 2	2813	228	1.0791	1.0190 - 1.1427	0.0093
	Type 4a	2762	226	0.9522	0.8931 - 1.0154	0.135
	Type 4b	2730	204	1.0984	1.0345 - 1.1663	0.0021
	Self-regulation	2112	151	1.0137	0.9883 - 1.0397	0.294
	Inhibition	2680	215	1.0089	0.9695 - 1.0499	0.663
	Agitation	2705	220	1.0319	0.9992 - 1.0657	0.056
	Type 5	2735	219	1.0407	0.9995 - 1.0836	0.053
	Combined Type 4	2607	197	0.9236	0.8849 - 0.9639	$2.62 \times 10^{-4}$
	OTHER CAUSES	Type 1	2817	354	1.0266	0.9898 - 1.0648
Type 2		2813	348	1.0444	1.0030 - 1.0875	0.035
Type 4a		2762	347	0.9833	0.9357 - 1.0334	0.506
Type 4b		2730	326	1.0652	1.0157 - 1.1172	0.0093
Self-regulation		2112	254	0.9967	0.9773 - 1.0165	0.741
Inhibition		2680	328	0.9903	0.9596 - 1.0220	0.545
Agitation		2705	337	1.0119	0.9863 - 1.0382	0.365
Type 5		2735	351	0.9952	0.9650 - 1.0264	0.762
Combined Type 4		2607	309	0.9619	0.9267 - 0.9984	0.041



**Table 4**

Survival analysis (Cox regression, using STATA with familial clustering), with sex, BMI, alcohol and smoking as additional predictors. HR, Hazard Ratio; 95% CI Low and 95 CI High, 95% confidence intervals for Hazard Ratio. Significant associations ( $p < 0.05$ ) are shown in bold font.

		N Total	N Deaths	HR	CI95 Low	CI95 High	p
ALL-CAUSE	Type 1	2444	873	1.0358	1.0122	1.0599	<b>0.0028</b>
	Type 2	2442	855	1.0238	0.9941	1.0545	0.117
	Type 4a	2410	857	0.9798	0.9484	1.0122	0.218
	Type 4b	2391	819	1.0306	0.9991	1.0631	0.057
	Self-regulation	1881	659	1.0039	0.9914	1.0165	0.542
	Inhibition	2354	827	1.0093	0.9897	1.0294	0.353
	Agitation	2371	844	1.0087	0.9916	1.0261	0.322
	Type 5	2391	851	0.9930	0.9726	1.0139	0.508
	Combined Type 4	2297	787	0.9751	0.9542	0.9963	<b>0.022</b>
	ANY CANCER	Type 1	2444	270	1.0217	0.9801	1.0650
Type 2		2442	265	0.9767	0.9257	1.0306	0.390
Type 4a		2410	264	1.0216	0.9648	1.0819	0.464
Type 4b		2391	262	0.9451	0.8921	1.0013	0.055
Self-regulation		1881	217	1.0189	0.9966	1.0417	0.098
Inhibition		2354	258	1.0203	0.9851	1.0568	0.262
Agitation		2371	263	0.9938	0.9641	1.0244	0.689
Type 5		2391	263	0.9683	0.9353	1.0026	0.070
Combined Type 4		2297	253	1.0351	0.9973	1.0742	0.069
tCVD		Type 1	2444	196	1.0590	1.0055	1.1153
	Type 2	2442	192	1.0765	1.0136	1.1434	<b>0.016</b>
	Type 4a	2410	190	0.9426	0.8800	1.0095	0.091
	Type 4b	2391	172	1.0951	1.0245	1.1705	<b>0.0076</b>
	Self-regulation	1881	135	1.0163	0.9891	1.0441	0.243
	Inhibition	2354	182	1.0231	0.9804	1.0676	0.294
	Agitation	2371	188	1.0303	0.9947	1.0672	0.096
	Type 5	2391	184	1.0372	0.9925	1.0840	0.104
	Combined Type 4	2297	166	0.9243	0.8836	0.9669	<b>6.26E-04</b>
	OTHER CAUSES	Type 1	2444	293	1.0425	1.0007	1.0860
Type 2		2442	287	1.0427	0.9952	1.0924	0.079
Type 4a		2410	289	0.9865	0.9336	1.0424	0.629
Type 4b		2391	271	1.0641	1.0077	1.1237	<b>0.025</b>
Self-regulation		1881	219	0.9938	0.9737	1.0144	0.554
Inhibition		2354	274	1.0009	0.9672	1.0358	0.959
Agitation		2371	282	1.0073	0.9785	1.0370	0.622
Type 5		2391	295	1.0091	0.9760	1.0432	0.595
Combined Type 4		2297	259	0.9644	0.9264	1.0039	0.076



**Fig. 2.** Kaplan-Meier survival curves for death from cardiovascular diseases, contrasting study participants with combined Type 4 scores of 12 or less and those with scores of 18 or more.

The horizontal line represents 85% survival and the vertical interrupted lines show that this occurs at 88.0 and 92.2 years, respectively. Note that because this plot is for cardiovascular conditions only, participants who died from other conditions are censored and the survival curves do not approach zero.

**Table 5**

Estimated Odds Ratios (OR) and 95% confidence intervals for all-cause, cancer and coronary heart disease (CHD) mortality, calculated for studies reported by Eysenck (1991).

All-cause mortality	Type 1 vs Type 4			Type 2 vs Type 4		
	OR	95% CI	p	OR	95% CI	p
Yugoslav sample	31.2	20.8 to 46.7	$1.26 \times 10^{-85}$	24.6	16.7 to 36.2	$4.85 \times 10^{-80}$
Heidelberg sample 1	38.5	13.2 to 112.0	$1.83 \times 10^{-18}$	54.1	19.3 to 152.2	$7.85 \times 10^{-31}$
Heidelberg sample 2	56.8	13.8 to 234.5	$3.31 \times 10^{-24}$	38.6	9.3 to 160.2	$1.94 \times 10^{-15}$
Cancer mortality	Type 1 vs Type 4					
	OR	95% CI	p			
Yugoslav sample	137.1	43.1 to 436.3	$2.38 \times 10^{-64}$			
Heidelberg sample 1	Infinity					
Heidelberg sample 2	Infinity					
CHD mortality	Type 2 vs Type 4					
	OR	95% CI	p			
Yugoslav sample	24.4	11.7 to 51.1	$6.93 \times 10^{-33}$			
Heidelberg sample 1	61.0	8.2 to 455.9	$6.99 \times 10^{-12}$			
Heidelberg sample 2	Infinity					

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## Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.paid.2019.109797.

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