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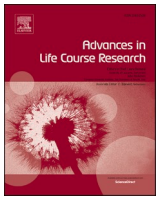

Long-term changes in sense of coherence and mortality among middle-aged men: A population -based follow-up study

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ABSTRACT

Sense of coherence (SOC) scale measures one's orientation to life. SOC is the core construct in Antonovsky's salutogenic model of health. It has been shown that weak SOC correlates with poor perceived health, low quality of life, and increased mortality. Some studies have indicated that SOC is not stable across life, but there are no previous studies on how a change of SOC is reflected in mortality. However, there is some evidence that a change in perceived quality of life is associated with mortality. The study explores the association between the change in SOC and mortality using longitudinal data from a cohort of middle-aged Finnish men recruited between 1986 and 1989. Approximately 11 years after the baseline examinations, between 1998 and 2001, 854 men returned the SOC questionnaire a second time. The baseline SOC was adjusted for the regression to the mean phenomenon between the two measurements. The hazard ratios of the SOC difference scores were adjusted for initial SOC age and 12 somatic risk factors of mortality (alcohol consumption, blood pressure, body mass index, cholesterol concentration, physical activity, education, smoking, marital status, employment status, history of cancer, history of cardiovascular disease and diabetes). SOC was not stable among middle-aged Finnish men and a decline in SOC was associated with an increased hazard of all-cause mortality. In the fully adjusted model, a decrease of one standard deviation (*SD*) of the SOC mean difference increased the mortality hazard by about 35 %, two *SD*s decrease about 70 %, and 2.5 *SD*s about 100 %. Strengthening SOC showed a limited association with decreasing mortality hazards in the age-adjusted model. Policies, strategies, or plans, supporting SOC in the middle-age may help to decrease mortality and increase quality of life in later years.

1. Introduction

Sense of coherence (SOC) is the core construct in Antonovsky's salutogenic model of health, which focuses on the origins of health instead of the origins of disease (Mittelmark & Bauer, 2022). SOC refers to an orientation to life, which is conceptualized as three sub-components called comprehensibility, manageability, and meaningfulness (Antonovsky, 1987). Comprehensibility describes how well one can make cognitive sense of surrounding events, whereas manageability refers to the readiness to manage with these same events. Meaningfulness refers to the perceived deeper meaning beyond the routines of everyday life.

Researchers have developed several versions of the original SOC scale, also called the 'Orientation to Life Questionnaire' (Antonovsky, 1987), and applied these scales as psychometric tools in nearly 50 countries during the past decades (Eriksson & Mittelmark, 2017). The SOC scale has been shown to be reliable, valid, feasible, and cross-culturally applicable (Antonovsky, 1993; Eriksson & Lindström, 2005).

SOC is related to perceived health and (Eriksson & Lindström, 2006) mortality. A recent meta-analysis showed that a weak SOC is associated

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with an increased risk of all-cause mortality in the general adult population even after adjustment for other mortality risk factors (Piiroinen et al., 2020). SOC has been shown to have a positive correlation with quality of life (Eriksson & Lindström, 2007); and declining health-related quality of life (HRQL) has increased whereas improving HRQL has decreased the mortality risk (Fan et al., 2004; Kroenke et al., 2008; Otero-Rodríguez et al., 2010). To the best of our knowledge, there are no previous studies of the association between the change in SOC and mortality.

Although the original theoretical model postulated that SOC would be stabilized by the end of young adulthood (Antonovsky, 1993), later studies have shown that SOC can change during a lifetime. By large, SOC scores tend to increase with age although a study of nearly 19,000 Finns, found that SOC was more stable among persons over 30 years than younger adults (Feldt et al., 2007). Other studies have shown that SOC weakens in the oldest age groups, and the decrease is associated with an accumulation of negative life events (Lövheim et al., 2013; Nilsson et al., 2003; Silverstein & Heap, 2015).

In addition to age, studies have explored the potential instability of SOC in combination with its initial level. In a study of 532 Finnish employees, it was found that SOC was less stable among individuals with an initially weak SOC (Hakanen et al., 2007). Correspondingly, a study of 1254 Swedes found that the SOC scores decreased over time among people with an initially weak SOC (Nilsson et al., 2003). However, negative life events tend to decrease SOC, independent of its initial level (Volanen, Suominen, Lahelma, Koskenvuo, & Silventoinen, 2007).

In this study, we aimed to investigate the relationship between long-term changes in SOC and all-cause mortality in men representative of the general male population in a prospective follow-up setting. We believe that this relationship is worth to be studied for three reasons: 1) based on several prospective cohort studies, SOC appears to vary more than originally theoretically assumed, 2) weak SOC is related to increased mortality risk, and 3) decreasing SOC may increase the mortality risk.

2. Methods

2.1. Sample

Data are from the Kuopio Ischaemic Heart Disease Risk Factor (KIHD) study, which started in 1984 and aimed at discovering explanatory factors for the high prevalence of coronary heart disease among Eastern Finnish men (Salonen, 1988). This population had the highest national recorded incidence of ischaemic heart diseases in the 1980 s (Salonen et al., 1991). Since then, the KIHD study has widened its aims to other chronic conditions and all aspects of health. The KIHD study population consisted of an age-stratified random sample of middle-aged men who lived in the city of Kuopio and its surroundings (Salonen et al., 1992). The recruitment and baseline measurements (Time 1) were carried out in two cohorts, first between 1984 and 1986 and the second one between 1986 and 1989. The first cohort included 1166 men and the second one consisted of 1516 men. All living participants of the second cohort were invited to the 11-year follow-up measurements (Time 2), and 854 men participated and thus, responded to the SOC questionnaire twice. The participants comprised of the following age groups: 42 years ($n = 218$), 48 years ($n = 215$), 54 years ($n = 223$) and 60 years ($n = 198$). All study participants gave written informed consent both at the Time 1 and Time 2. The ethical committee of Kuopio university and Kuopio university hospital approved the KIHD study on December 1, 1983, and again on October 27, 1997 (Approval number 143/97).

2.2. Measurement of SOC

The KIHD study applied the originally 13-item SOC scale, which has relatively high structural validity and high temporal stability (Feldt et al., 2007). However, the final version for the KIHD study did not include the 10th question ("Many people, even those with a strong character, sometimes feel like sad sacks in certain situations. How often you felt this way in the past?") due to difficulties in translating it from English to Finnish (Lynch et al., 1997). Each question was answered on a 7-point semantic scale so that the lowest possible sum score in the total scale was 7 and the highest 84.

Consequently, participants of this study answered the SOC questionnaire twice; at Time 1 between August 1986 and December 1989 and at Time 2 approximately 11 years after Time 1 between October 1998 and February 2001. The mean (range) follow-up time between the measurements was 11.1 (9.7–14.4) years. We used item-specific mean imputation to replace missing values. At Time 1, 92 study participants skipped at least one SOC item, whereas, at Time 2, 58 skipped at least one item. In general, mean imputation is the preferred method when imputing characteristics of study participants (Sullivan et al., 2018).

2.3. Covariates

In addition to age, we included leading somatic risk factors of mortality as covariates (Mathers et al., 2009). We also considered covariates applied in other cohort studies investigating the relationship between SOC and mortality (Piiroinen et al., 2020). These covariates were measured at the baseline and were alcohol consumption, blood pressure, Body Mass Index (BMI), cholesterol concentration, physical activity, education, smoking, marital status, employment status, history of cancer, history of cardiovascular disease (CVD), and diabetes. To avoid over adjustment (Schisterman et al., 2009) we did not control for other psychometrically determining factors, which could be associated with SOC.

The first seven covariates are based on study participants' self-reports i.e., answers to questionnaire items. Physical activity is based on interviews regarding study participants' physical activity during the past 365 days. Blood pressure and BMI are based on direct measurements. Blood pressure refers to the mean value of six systolic blood pressure measurements. Cholesterol concentration is based on blood samples taken at the follow-up visit and refers to the total serum cholesterol level. Diagnosis of diabetes is based on blood samples taken at the follow-up visit together with study participants' self-reports regarding whether they have diabetes or not. Diabetes blood samples refer to the fasting blood glucose level (FBG), and we considered FBG $>$ 6.9 mmol/l as an indicator of diabetes (Mayo Clinic, 2019).

Study participants gave blood samples between 8 and 10 a.m. after abstaining from alcohol for three days and from smoking and eating for 12 h. After a supine rest of 30 min, a research nurse drew blood with Terumo Venoject VT-100PZ vacuum tubes (Terumo Corp., Tokyo, Japan) using no tourniquet. The laboratory of The University of Kuopio, Finland, used an enzymatic method to measure STC concentrations (CHOD-PAP, Boehringer Mannheim, Mannheim, West Germany) and a glucose dehydrogenase method (Merck, Darmstadt, West Germany) after protein precipitation with TCA using a clinical chemistry analyzer (Kone Specific, KONE Instruments Oy, Espoo, Finland) to measure FBG concentrations. Salonen et al. (1991) describe the lipid analysis in detail.

2.4. Outcome variable

All-cause mortality was our outcome of interest. The follow-up started from the Time 2 measurements and lasted to death or to December 31, 2018, whichever came first. Statistics Finland provided causes of death as international classification codes for diseases (ICD) together with dates (Data permission number TK-53–1770–16).

2.5. Statistical analyses

As an index of change in SOC scores, we used difference scores (Human et al., 2013), which were calculated for each participant by subtracting the Time 1 score from the Time 2 score. Thus, a positive difference score indicates a strengthened SOC over the follow-up time.

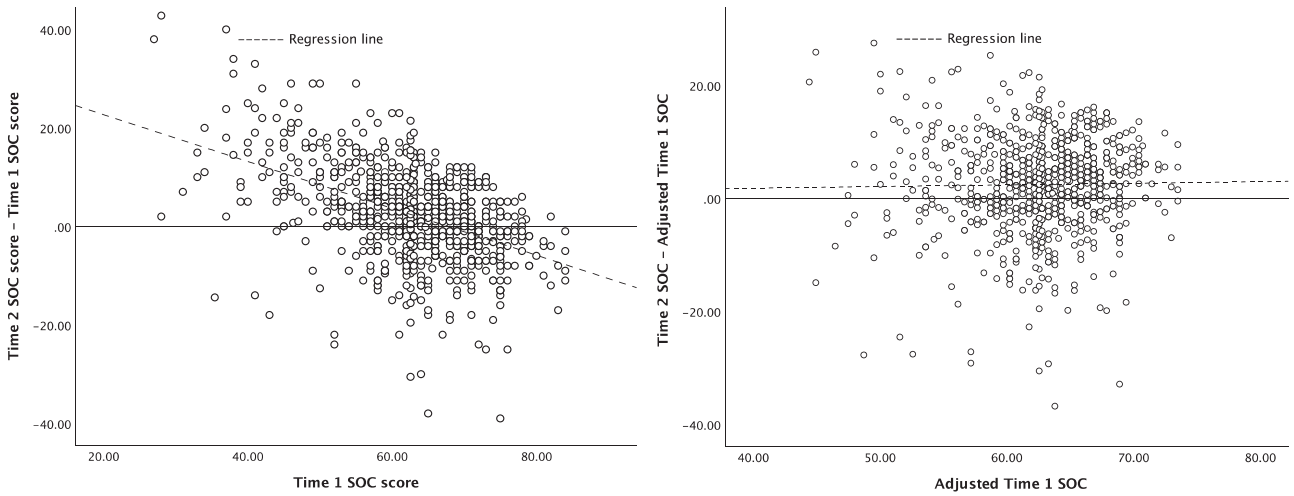

Fig. 1. Scatterplot of Sense of Coherence (SOC) measurements ($n = 854$) showing difference score (Time 2 SOC score minus Time 1 SOC score) against Time 1 SOC score from the Kuopio Ischaemic Heart Disease Risk Factor Study. The solid line represents perfect agreement (no change), and the dotted line is the regression line. The left figure presents the scatterplot using the original baseline scores. In the right figure, the baseline scores are adjusted for the regression to the mean. Time 1 = baseline, Time 2 = 11-year follow-up.

Table 1
Time 1 (baseline) characteristics of the Kuopio Ischaemic Heart Disease Risk Factor study participants by vital status at the end of the year 2018.

Variable	Total	Alive	Deceased	*p*-value
n (%)	854	485 (56.8)	369 (43.2)	–
Time 2 in years (*SD*)	16.1 (5.0)	19.4 (0.8)	11.8 (5.0)	–
Age (*SD*)	51.4 (6.7)	49.1 (6.0)	54.5 (6.3)	< 0.001 *
Alcohol (g/week)	71.3 (108.3)	65.5 (97.4)	78.9 (120.9)	.080
SBP (mmHg)	131.0 (15.4)	130.0 (14.3)	132.2 (16.7)	.043 *
BMI (kg/m^2)	26.6 (3.2)	26.4 (3.2)	26.9 (3.3)	.017 *
STC (mmol/l)	5.7 (1.0)	5.7 (0.9)	5.8 (1.1)	.252
LPA (kcal/day)	143.9 (153.8)	136.4 (135.4)	153.9 (174.8)	.112
Education (years)	9.5 (3.6)	10.0 (3.6)	8.7 (3.5)	< 0.001 *
Smoking *n* (%)	238 (27.9)	107 (22.1)	131 (35.5)	< 0.001 *
Not married *n* (%)	91 (10.7)	47 (9.7)	44 (11.9)	.289
Unknown	1	0	1	–
Retired or no work *n* (%)	236 (27.6)	82 (16.9)	154 (41.7)	< 0.001 *
History of cancer *n* (%)	14 (1.6)	5 (1.0)	9 (2.4)	.108
History of CVD *n* (%)	276 (32.3)	116 (23.9)	160 (43.4)	< 0.001 *
Diabetes *n* (%)	29 (3.4)	10 (2.1)	19 (5.1)	.014 *

Values indicate mean. SD: standard deviation. p-values refer to independent samples t-test or Chi-square test. Time 2 (11-year follow-up), SBP: Systolic blood pressure, BMI: Body mass index, STC: Serum total cholesterol, LPA: Leisure-time physical activity, CVD: Cardiovascular disease, excluding hypertension. * = $p < .05$.

Furthermore, we acknowledged that repeated measurements on two timepoints could be influenced by the regression to the mean (RTM) phenomenon, which is caused by random measurement error and seen as a correlation in a scatterplot of change (Barnett et al., 2005). In our data, we observed a clear negative correlation in the scatterplot of change, which indicates that those whose Time 1 scores were unusually high or low tended to regress towards the mean on the Time 2 measurement (Fig. 1). Therefore, we adjusted the original Time 1 SOC scores for RTM using the formula $x_{adj} = \overline{x} + r(x - \overline{x})$, where $\overline{x}$ is the Time 1 mean, r is the Time 1 correlation to Time 2, and x is the individual's Time 1 score (Linden, 2013). This adjustment removed the negative correlation (Fig. 1).

We applied the Cox proportional-hazards model to compute hazards of all-cause mortality and performed separate models for each point in SOC change. Thus, were compared two groups with each other: decreasing and increasing SOC, the difference score being a cut-off separator between these two. We conducted the analyses in two steps: Model 1, we adjusted for age, and for Model 2 we added the 12 covariates. Of these covariates, six were continuous (alcohol consumption, blood pressure, BMI, cholesterol concentration, physical activity, education in years) and six were binomial (smoking, marital status, employment status, history of cancer, history of CVD, diabetes). In addition to hazard ratios (*HRs*), we report *z*-scores for the difference scores to compare the magnitude of the change with other SOC scales. We evaluated the proportional hazards assumption for the Cox models by means of Schoenfeld residuals and, when necessary, we considered variable-specific time-dependence corrections.

To compare covariate differences between those who were alive and those who deceased by the end of 2018 we used independent samples *t*-test for continuous data and a Chi-square test for nominal data (Table 1). To compare Time 1 and Time 2 differences inside age groups and SOC baseline tertiles (weak, medium, strong) we used paired samples *t*-test (Table 2). Furthermore, we used a one-way analysis of variance to compare the effect of the four age groups (42, 48, 54, and 60) on the difference scores. The significance level was set at $p < .05$. IBM® SPSS® Statistics Version 27 was used to carry out the analyses.

3. Results

3.1. Characteristics of study participants

Of the 854 study participants, 369 (43 %) died during the follow-up by the end of 2018. In each age group, 42, 48, 54, and 60, the respective number of deaths was 45 (21 %), 65 (30 %), 110 (49 %) and 149 (75 %). A cardiovascular disease was the most common cause of death ($n = 170$, 46 % of 369 deaths) followed by cancer ($n = 89$, 24 %). Those who died by the end of 2018 were older, had higher blood pressure and BMI, had fewer years of education, were more often smokers, were more often not employed, and had more often cardiovascular diseases and diabetes compared to those who stayed alive (Table 1).

Table 2
Time 1 (baseline) and Time 2 (11-year follow-up) difference in sense of coherence (SOC) scores for the Kuopio Ischaemic Heart Disease Risk Factor study participants ($n = 854$) for different baseline age groups and SOC baseline tertiles (weak, medium, and strong).

Baseline group	Time 1 not adjusted for RTM (*SD*)	Time 1 adjusted for RTM (*SD*)	Time 2 (*SD*)	*p*-value for not adjusted Time 1 - Time 2	*p*-value adjusted Time 1 - Time 2
Age group 42	62.86 (8.80)	62.71 (4.49)	63.07 (11.05)	.746	.574
Age group 48	62.42 (9.96)	62.49 (5.08)	66.37 (8.98)	< 0.001 *	< 0.001 *
Age group 54	62.12 (9.62)	62.34 (4.90)	65.17 (9.62)	< 0.001 *	< 0.001 *
Age group 60	62.86 (8.45)	62.71 (4.31)	65.43 (8.94)	< 0.001 *	< 0.001 *
SOC unadj weak	52.36 (7.13)	–	59.86 (9.74)	< 0.001 *	–
SOC unadj medium	63.27 (1.50)	–	64.39 (8.26)	.021 *	–
SOC unadj strong	71.85 (3.88)	–	70.67 (3.88)	.007 *	–
SOC adj weak	–	57.77 (3.69)	60.30 (9.74)	–	< 0.001 *
SOC adj medium	–	63.21 (0.84)	64.77 (8.15)	–	.001 *
SOC adj strong	–	67.54 (1.91)	70.85 (7.42)	–	< 0.001 *

Values indicate mean. SD: standard deviation. RTM: regression to the mean. unadj: SOC 1 unadjusted. adj: SOC 1 adjusted for RTM. p-values refer to paired samples t-test. * = p < .05.

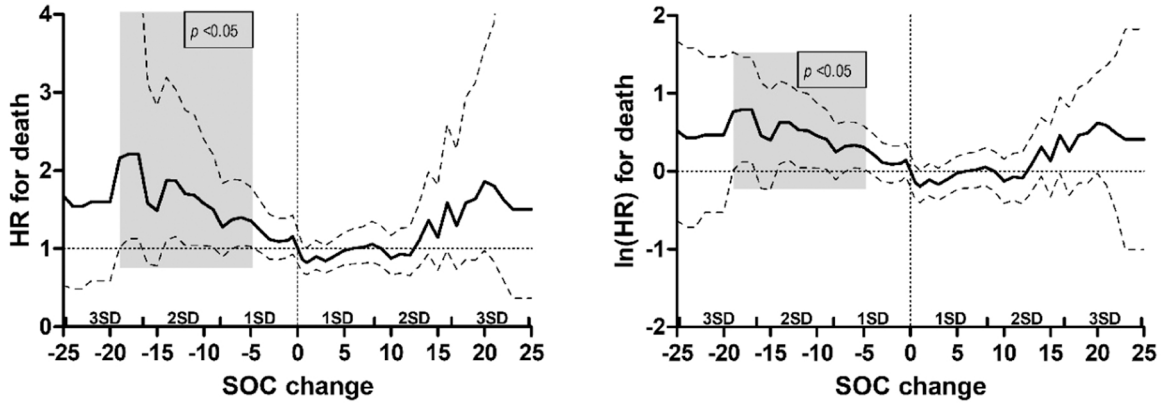

Fig. 2. Relationships of sense of coherence (SOC) change with the hazard ratio (*HR*) for death. The solid lines represent mean *HR*, and the dotted lines its 95 % confidence intervals. On the left figure, the $HR = 1$, and on the right picture $\ln(HR) = 0$ indicates no difference. ln: natural logarithm, *SD*: standard deviation. When the change was < 0 the reference group included all larger values, and when the change was > 0 the reference group included all smaller values.

3.2. Change in sense of coherence scores over 11 years of follow-up

At Time 1, the mean SOC score was 62.6 (*SD* 9.2, range = 27.0–84.0 for unadjusted; and *SD* 4.7, range = 44.4–73.5 for RTM adjusted scores). At Time 2, the mean SOC score was 65.0 (*SD* 9.6, range = 21.0–84.0). The mean difference in SOC score (Time 2 - Time 1) was 2.4 (*SD* 9.3, $p < .001$, for unadjusted Time 1; and *SD* 8.2, $p < .001$, for RTM adjusted Time 1). Compared to the unadjusted Time 1 SOC score, the mean decrease in Time 2 was − 6.7 (*SD* 6.0) points, and the mean increase was 8.0 (*SD* 6.6) points, and the range was − 39.0–42.8 (*SD* 9.3). Compared to the RTM adjusted Time 1 SOC score, the mean decrease in Time 2 was − 6.1 (*SD* 6.3) points, whereas the mean increase was 6.9 (*SD* 5.0) points, and the range was − 36.8–27.5 (*SD* 8.2). The Cronbach's α for the SOC scale was 0.82 at Time 1 and 0.85 at Time 2.

For both unadjusted and RTM adjusted Time 1 SOC, the three oldest age groups (48, 54, and 60) showed a significant increase in Time 2 SOC, whereas the youngest baseline age group (42) remained at the same level during the 11-year follow-up (Table 2). One way analysis of variance showed that the effect of age was significant on the change in SOC ($F(3850) = 6.44$, $p < .001$). For these analyses, we used only the difference scores with original Time 1 values since, with the RTM adjusted values, there was heterogeneity of variances (Levene's test, $p = .014$). Post hoc analyses, using the Bonferroni criterion for significance, indicated that the SOC difference score was lower in the youngest age group of 42 ($M = 0.22$, $SD = 9.82$) than in the age groups of 48 ($M = 3.95$, $SD = 9.30$, $p < .001$) or 52 ($M = 3.05$, $SD = 9.10$, $p = .008$). The two age groups in the middle did not differ from each other, and the oldest age group of 60 ($M = 2.57$, $SD = 9.10$) did not differ from any of the other groups.

Both unadjusted and RTM adjusted Time 1 SOC tertiles (weak, medium, strong) showed significant changes in Time 2. For unadjusted values, there was a mean 7.5 points increase in the weak and 1.1 points increase in the medium tertiles, and 1.2 points decrease in the strong tertile. When Time 1 was adjusted for RTM, all tertiles, weak, medium, and strong showed a significant increase: 2.5, 1.6, and 3.3 points, respectively (Table 2). Between tertiles, a comparison could not be performed, since there was heterogeneity of variances for both unadjusted (Levene's test, $p < .001$) and adjusted (Levene's test, $p = .004$) Time 1 SOC. The *SD*s for the weak, medium and strong SOC tertiles were 7.13, 1.50, and 3.88, respectively unadjusted for Time 1; and 3.69, 0,84, and 1.91 for adjusted for Time 1.

3.3. Change in sense of coherence and all-cause mortality

A decrease of SOC was associated with an increased risk of all-cause mortality, and the association reached statistically significant levels at certain point intervals (Fig. 2). In the age-adjusted Model 1 (see Table A1 Appendix), when Time 1 was adjusted for RTM, those whose SOC had decreased by 4–7 or more points ($z \leq -0.78$ to -1.14) showed 35–55 % ($HR = 1.35$–1.55) higher hazard of dying than those whose SOC decreased less than 4 points. Correspondingly, a decrease by 9–14 or more points ($z \leq -1.39$ to -1.99) increased the hazard 60–85 % ($HR = 1.60$–1.85), and a decrease by 17–19 or more points ($z \leq -2.35$ to -2.60) increased the hazard 121–124 % ($HR = 2.21$–2.24). In Model 1, when Time 1 was unadjusted, a SOC decrease of 1–3 or more points ($z \leq -0.37$ to -0.58) increased the hazard 27–31 % ($HR = 1.27$–1.31), a

Table A1
Age adjusted Model 1 Hazard Rates associated with all-cause mortality among Kuopio Ischaemic Heart Disease Risk Factor (KIHD) study participants for those whose Sense of Coherence (SOC) was measured twice (n = 854) and was decreasing. Reference group was 'SOC increased, remained, or decreased less'.

Decrease in SOC	Time 1 not adjusted for RTM				Time 1 adjusted for RTM			
Points	*HR* (95% *CI*)	*p*	*n* (%)	*HR*[1]	*HR* (95% *CI*)	*p*	*n* (%)	*HR*[1]
< 0	1.26 (1.03–1.56)	.029 *	303 (35.5)	1.27	1.22 (0.98–1.51)	.065	293 (34.3)	1.23
≤ −1	1.27 (1.03–1.57)	.026 *	298 (34.8)	1.28	1.17 (0.94–1.46)	.157	251 (29.4)	1.18
≤ −2	1.27 (1.02–1.58)	.034 *	257 (30.1)	1.27	1.15 (0.91–1.46)	.237	204 (23.9)	1.16
≤ −3	1.31 (1.05–1.64)	.019 *	225 (26.4)	1.32	1.22 (0.95–1.57)	.122	174 (20.4)	1.22
≤ −4	1.25 (0.98–1.59)	.073	193 (22.6)	1.25	1.35 (1.04–1.75)	.026 *	151 (17.7)	1.36
≤ −5	1.35 (1.05–1.74)	.019 *	164 (19.2)	1.36	1.42 (1.08–1.88)	.012 *	127 (15.9)	1.43
≤ −6	1.35 (1.04–1.76)	.027 *	139 (16.3)	1.36	1.49 (1.11–1.99)	.007 *	109 (12.8)	1.50
≤ −7	1.26 (0.95–1.68)	.111	121 (14.2)	1.27	1.55 (1.13–2.12)	.006 *	90 (10.5)	1.58
≤ −8	1.27 (0.93–1.73)	.139	98 (11.5)	1.28	1.42 (0.99–2.02)	.055	74 (8.7)	1.44
≤ −9	1.29 (0.91–1.83)	.149	80 (9.4)	1.30	1.60 (1.20–2.33)	.014 *	63 (7.4)	1.63
≤ −10	1.35 (0.93–1.96)	.119	65 (7.6)	1.36	1.70 (1.13–2.58)	.011 *	54 (6.3)	1.74
≤ −11	1.31 (0.89–1.93)	.168	61 (7.1)	1.32	1.70 (1.06–2.72)	.027 *	44 (5.2)	1.75
≤ −12	1.35 (0.86–2.12)	.195	48 (5.6)	1.36	1.77 (1.10–2.86)	.019 *	39 (4.6)	1.83
≤ −13	1.32 (0.78–2.21)	.301	38 (4.5)	1.33	1.89 (1.17–3.04)	.009 *	37 (4.3)	1.95
≤ −14	1.65 (0.96–2.82)	.070	31 (3.6)	1.68	1.85 (1.10–3.12)	.021 *	32 (3.8)	1.91
≤ −15	1.35 (0.71–2.53)	.359	25 (2.9)	1.36	1.59 (0.84–3.00)	.152	25 (2.9)	1.63
≤ −16	1.22 (0.57–2.60)	.580	22 (2.6)	1.24	1.86 (0.96–3.63)	.068	20 (2.3)	1.92
≤ −17	1.22 (0.58–2.60)	.599	20 (2.3)	1.24	2.24 (1.15–4.36)	.017 *	16 (1.9)	2.33
≤ −18	1.47 (0.69–3.11)	.320	18 (2.1)	1.50	2.24 (1.15–4.36)	.017 *	16 (1.9)	2.33
≤ −19	2.09 (0.98–4.43)	.055	14 (1.6)	2.14	2.21 (1.04–4.70)	.039 *	14 (1.6)	2.29
≤ −20	2.36 (1.05–5.31)	.038 *	11 (1.3)	2.45	1.73 (0.64–4.66)	.278	10 (1.2)	1.79
≤ −21	2.36 (1.05–5.31)	.038 *	11 (1.3)	2.45	1.73 (0.64–4.66)	.278	10 (1.2)	1.79
≤ −22	2.36 (1.05–5.31)	.038 *	11 (1.3)	2.45	1.73 (0.64–4.66)	.278	10 (1.2)	1.79
≤ −23	2.77 (1.14–6.71)	.024 *	8 (0.9)	2.89	1.49 (0.48–4.67)	.493	9 (1.2)	1.55
≤ −24	2.77 (1.14–6.71)	.024 *	8 (0.9)	2.89	1.49 (0.48–4.67)	.493	9 (1.2)	1.55
≤ −25	2.08 (0.67–6.48)	.208	6 (0.7)	2.15	1.65 (0.53–5.18)	.387	8 (0.9)	1.72
≤ −26	1.16 (0.16–8.32)	.882	4 (0.5)	1.18	1.65 (0.53–5.18)	.387	8 (0.9)	1.72
≤ −27	1.16 (0.16–8.32)	.882	4 (0.5)	1.18	1.65 (0.53–5.18)	.387	8 (0.9)	1.72
≤ −28	1.16 (0.16–8.32)	.882	4 (0.5)	1.18	2.28 (0.56–9.25)	.247	5 (0.6)	2.30
≤ −29	1.16 (0.16–8.32)	.882	4 (0.5)	1.18	2.28 (0.56–9.25)	.247	5 (0.6)	2.30
≤ −30	1.16 (0.16–8.32)	.882	4 (0.5)	1.18	1.56 (0.22–11.17)	.658	3 (0.4)	1.58
≤ −31	0.00 (0.00–E)	.923	2 (0.2)	0.00	0.00 (0.00–E)	.923	2 (0.2)	0.00
≤ −32	0.00 (0.00–E)	.923	2 (0.2)	0.00	0.00 (0.00–E)	.923	2 (0.2)	0.00
≤ −33	0.00 (0.00–E)	.923	2 (0.2)	0.00	0.00 (0.00–E)	.95	1 (0.1)	0.00
≤ −34	0.00 (0.00–E)	.923	2 (0.2)	0.00	0.00 (0.00–E)	.95	1 (0.1)	0.00
≤ −35	0.00 (0.00–E)	.923	2 (0.2)	0.00	0.00 (0.00–E)	.95	1 (0.1)	0.00
≤ −36	0.00 (0.00–E)	.923	2 (0.2)	0.00	0.00 (0.00–E)	.95	1 (0.1)	0.00
≤ −37	0.00 (0.00–E)	.923	2 (0.2)	0.00	–	–	0 (0.0)	–
≤ −38	0.00 (0.00–E)	.923	2 (0.2)	0.00	–	–	0 (0.0)	–
≤ −39	0.00 (0.00–E)	.945	1 (0.1)	0.00	–	–	0 (0.0)	–
≤ −40	–	–	0 (0.0)	–	–	–	0 (0.0)	–

Notes. Decrease from Time 1 = baseline to Time 2 = 11-year follow-up; *HR* = hazard rate; CI = confidence interval; * = $p < .05$; RTM = Regression to the Mean; E = very wide. [1]Age as a time-dependent covariate.

decrease of 5–6 or more points ($z \leq -0.80$ to -0.90) increased the hazard 35 % (*HR* = 1.35), and a decrease of 20–24 or more points ($z \leq -2.81$ to -3.20) increased the hazard 136–177 % (*HR* = 2.36–2.77). All other point intervals failed to reach a statistically significant level.

In the fully adjusted Model 2 (see Table A2 Appendix), decreasing SOC was associated with an increased hazard of all-cause mortality only if Time 1 was adjusted for RTM. In this model, a SOC decrease of 5–6 or more points ($z \leq -0.90$ to -1.02) increased the hazard 32–36 % (*HR* = 1.32–1.36), a decrease of 13–14 or more points ($z \leq -1.87$ to -1.99) increased the hazard 73–74 % (*HR* = 1.73–1.74) and a decrease of 17–18 or more points ($z \leq -2.36$ to -2.48) increased the hazard 103 % (*HR* = 2.03).

An increase of SOC showed a statistically significant association with mortality only in the age adjusted Model 1 (see Table A3 Appendix): When Time 1 was not adjusted for RTM, those whose difference scores increased 3 or more points ($z \geq 0.06$) had 20 % (*HR* = 0.80) lower mortality hazard than those with lower values. When Time 1 values were adjusted for RTM, those who increased their SOC by one or more points ($z \geq -0.17$) had 22 % (*HR* = 0.78) lower mortality hazard than those with lower values, and in the group of an increase of 3 or more points ($z \geq 0.07$), the hazard was reduced 20 % (*HR* = 0.80). In one cut-off score group, a higher SOC score increase was associated with a higher hazard: over 20 points ($z \geq 2.13$) increase increased the hazard by 97 % (*HR* = 1.97).

Schoenfeld residuals revealed effects of age, STC, and years of education on the hazard of dying being dependent on the follow-up time. Consequently, we considered these time-dependencies in the additional Cox models and report the *HR*s in Appendix. All in all, the time-dependency did not affect statistical significance.

4. Discussion

The current study showed that SOC is not stable among middle-aged Finnish men and a decline in SOC is associated with an increased hazard of all-cause mortality both in the age-adjusted model and in the model adjusted for several health-related covariates. The latter association was apparent only if the baseline SOC was adjusted for RTM. We believe that the RTM adjustment was a strength of our study and represents a more reliable indication of the true difference and should be used if a correlation can be seen in the scatterplot of change (Barnett et al., 2005), as it was in this data. Therefore, we concentrate the discussion on the RTM-adjusted values.

Similarly, as with the previous findings of the association between changes in HRQL and mortality (Fan et al., 2004; Kroenke et al., 2008; Otero-Rodríguez et al., 2010), the greater the reduction in SOC score the

Table A2
Fully adjusted Model 2 Hazard Rates associated with all-cause mortality among Kuopio Ischaemic Heart Disease Risk Factor (KIHD) study participants for those whose Sense of Coherence (SOC) was measured twice (n = 854) and was decreasing. Reference group was 'SOC increased, remained, or decreased less'.

Decrease in SOC	Time 1 not adjusted for RTM				Time 1adjusted for RTM			
Points	*HR* (95% *CI*)	*p*	*n* (%)	*HR*[1]	HR (95% *CI*)	*p*	*n* (%)	*HR*[1]
< 0	1.16 (0.94–1.43)	.172	303 (35.5)	1.19	1.13 (0.91–1.40)	.272	293 (34.3)	1.16
≤ −1	1.17 (0.95–1.45)	.146	298 (34.8)	1.20	1.08 (0.87–1.35)	.477	251 (29.4)	1.11
≤ −2	1.19 (0.95–1.48)	.126	257 (30.1)	1.21	1.07 (0.84–1.36)	.600	204 (23.9)	1.09
≤ −3	1.24 (0.99–1.55)	.068	225 (26.4)	1.26	1.09 (0.85–1.41)	.489	174 (20.4)	1.12
≤ −4	1.18 (0.93–1.51)	.174	193 (22.6)	1.20	1.21 (0.93–1.58)	.150	151 (17.7)	1.24
≤ −5	1.25 (0.97–1.62)	.081	164 (19.2)	1.29	1.32 (1.00–1.75)	.048 *	127 (15.9)	1.36
≤ −6	1.25 (0.95–1.63)	.107	139 (16.3)	1.28	1.36 (1.02–1.82)	.039 *	109 (12.8)	1.40
≤ −7	1.19 (0.89–1.59)	.246	121 (14.2)	1.22	1.33 (0.97–1.82)	.081	90 (10.5)	1.37
≤ −8	1.15 (0.84–1.58)	.382	98 (11.5)	1.18	1.23 (0.86–1.75)	.268	74 (8.7)	1.28
≤ −9	1.17 (0.82–1.67)	.375	80 (9.4)	1.20	1.43 (0.98–2.09)	.067	63 (7.4)	1.50
≤ −10	1.16 (0.80–1.70)	.437	65 (7.6)	1.19	1.46 (0.97–2.22)	.073	54 (6.3)	1.58
≤ −11	1.13 (0.76–1.67)	.543	61 (7.1)	1.16	1.57 (0.98–2.52)	.063	44 (5.2)	1.68
≤ −12	1.17 (0.74–1.86)	.493	48 (5.6)	1.21	1.58 (0.97–2.57)	.066	39 (4.6)	1.70
≤ −13	1.25 (0.74–2.10)	.413	38 (4.5)	1.30	1.73 (1.07–2.80)	.026 *	37 (4.3)	1.87
≤ −14	1.43 (0.83–2.46)	.198	31 (3.6)	1.51	1.74 (1.02–2.96)	.041 *	32 (3.8)	1.88
≤ −15	1.16 (0.61–2.19)	.648	25 (2.9)	1.20	1.39 (0.73–2.64)	.312	25 (2.9)	1.49
≤ −16	1.12 (0.56–2.28)	.745	22 (2.6)	1.15	1.47 (0.74–2.88)	.269	20 (2.3)	1.58
≤ −17	1.13 (0.53–2.39)	.760	20 (2.3)	1.17	2.03 (1.04–3.97)	.038 *	16 (1.9)	2.21
≤ −18	1.25 (0.59–2.65)	.568	18 (2.1)	1.31	2.03 (1.04–3.97)	.038 *	16 (1.9)	2.21
≤ −19	1.77 (0.83–3.79)	.140	14 (1.6)	1.85	1.98 (0.93–4.23)	.077	14 (1.6)	2.16
≤ −20	2.03 (0.90–4.60)	.089	11 (1.3)	2.14	1.46 (0.54–3.97)	.455	10 (1.2)	1.60
≤ −21	2.03 (0.90–4.60)	.089	11 (1.3)	2.14	1.46 (0.54–3.97)	.455	10 (1.2)	1.60
≤ −22	2.03 (0.90–4.60)	.089	11 (1.3)	2.14	1.46 (0.54–3.97)	.455	10 (1.2)	1.60
≤ −23	2.35 (0.96–5.74)	.060	8 (0.9)	2.49	1.39 (0.44–4.38)	.579	9 (1.2)	1.54
≤ −24	2.35 (0.96–5.74)	.060	8 (0.9)	2.49	1.39 (0.44–4.38)	.579	9 (1.2)	1.54
≤ −25	1.75 (0.56–5.52)	.337	6 (0.7)	1.82	1.50 (0.47–4.74)	.491	8 (0.9)	1.68
≤ −26	0.97 (0.13–7.01)	.976	4 (0.5)	0.99	1.50 (0.47–4.74)	.491	8 (0.9)	1.68
≤ −27	0.97 (0.13–7.01)	.976	4 (0.5)	0.99	1.50 (0.47–4.74)	.491	8 (0.9)	1.68
≤ −28	0.97 (0.13–7.01)	.976	4 (0.5)	0.99	1.91 (0.47–7.84)	.367	5 (0.6)	1.93
≤ −29	0.97 (0.13–7.01)	.976	4 (0.5)	0.99	1.91 (0.47–7.84)	.367	5 (0.6)	1.93
≤ −30	0.97 (0.13–7.01)	.976	4 (0.5)	0.99	1.36 (0.19–9.82)	.759	3 (0.4)	1.38
≤ −31	0.00 (0.00–E)	.927	2 (0.2)	0.00	0.00 (0.00–E)	.927	2 (0.2)	0.00
≤ −32	0.00 (0.00–E)	.927	2 (0.2)	0.00	0.00 (0.00–E)	.927	2 (0.2)	0.00
≤ −33	0.00 (0.00–E)	.927	2 (0.2)	0.00	0.00 (0.00–E)	.949	1 (0.1)	0.00
≤ −34	0.00 (0.00–E)	.927	2 (0.2)	0.00	0.00 (0.00–E)	.949	1 (0.1)	0.00
≤ −35	0.00 (0.00–E)	.927	2 (0.2)	0.00	0.00 (0.00–E)	.949	1 (0.1)	0.00
≤ −36	0.00 (0.00–E)	.927	2 (0.2)	0.00	0.00 (0.00–E)	.949	1 (0.1)	0.00
≤ −37	0.00 (0.00–E)	.927	2 (0.2)	0.00	–	–	0 (0.0)	–
≤ −38	0.00 (0.00–E)	.927	2 (0.2)	0.00	–	–	0 (0.0)	–
≤ −39	0.00 (0.00–E)	.948	1 (0.1)	0.00	–	–	0 (0.0)	–
≤ −40	–	–	0 (0.0)	–	–	–	0 (0.0)	–

Notes. Decrease from Time 1 = baseline to Time 2 = 11-year follow-up; *HR* = hazard rate; *CI* = confidence interval; * = $p < .05$; RTM = Regression to the Mean; E = very wide. [1]Age, total cholesterol concentration, and years of education as time-dependent covariates

higher was the increase in mortality hazard. For example, in the fully adjusted model, those whose SOC difference score was minus one SD or more away from the mean of difference scores showed an about 35 % higher hazard of dying than others. Respectively, if the difference score was minus two *SD*s or more below the mean, the hazard was about 70 % higher, and if the difference score was minus 2.5 *SD*s or more away from the mean, the hazard was about 100 % higher. So, the current study showed a clearly higher mortality hazard with decreasing SOC than the previous meta-analysis with a 17 % increase in the mortality risk in the lowest SOC tertile, when SOC was measured only at the baseline (Piiroinen et al., 2020). However, we acknowledged the difference score partly, and inevitably, reflects SOC at Time 2 as such and, consequently, measuring SOC only once in the time continuum could be enough for detecting people at the greatest risk. On the other hand, in the KIHD cohort, SOC at Time 1 and at Time 2 are poorer predictors of mortality than the difference score, also in models with the maximum adjustment (Piiroinen et al., 2020; unpublished data available upon request).

Contrary to previous studies of the association between changes in HRQL and mortality (Fan et al., 2004; Kroenke et al., 2008; Otero-Rodríguez et al., 2010), our data could not show an equally evident association between an increasing SOC and mortality as with a decreasing SOC. The age-adjusted model indicated that those whose SOC difference score was close to the mean of difference scores or higher had a 20 % smaller mortality hazard during the observation period. Surprisingly, in this model, those few ($n = 13$) whose SOC difference score was over two *SD*s higher than the mean had a nearly 100 % higher risk of dying than others. A similar trend, i.e., higher mortality risk along with the very high difference scores, could be seen in the fully adjusted model as well, but it did not reach statistical significance.

Our study was in line with those earlier findings, in which SOC, on average, tended to increase with age (Feldt et al., 2007; Lövheim et al., 2013; Silverstein & Heap, 2015). In contrast to previous studies (Lövheim et al., 2013; Nilsson et al., 2003; Silverstein & Heap, 2015), our data showed an increase in mean SOC even in the oldest age group (60 at the baseline), and, on the other hand, participants in the youngest age group (42) did not show any increase in the SOC during the 11-year follow-up. This could be interpreted as the oldest participants in our sample were still being relatively young and not having experienced negative life event accumulation, which often are associated with age over 70 (Silverstein & Heap, 2015). On the other hand, the youngest age group could have experienced the deep financial crisis in the 1990 s in Finland more severely than the older age groups. In addition to age, the level of baseline SOC could have influenced the change, but this was unclear in our results. Contrary to another study (Nilsson et al., 2003), in our sample, SOC increased also in the lowest SOC baseline. Still, as in a previous study (Hakanen et al., 2007), our sample showed the highest

Table A3
Age adjusted Model 1 Hazard Rates associated with all-cause mortality among Kuopio Ischaemic Heart Disease Risk Factor (KIHD) study participants for those whose Sense of Coherence (SOC) was measured twice (n = 854) and was increasing. Reference group was 'SOC decreased, remained, or increased less'.

Increase in SOC	Time 1 not adjusted for RTM				Time 1 adjusted for RTM			
Points	*HR* (95% *CI*)	*p*	*n* (%)	*HR*[1]	HR (95% *CI*)	*p*	*n* (%)	*HR*[1]
> 0	0.84 (0.68–1.03)	.099	515 (60.3)	0.85	0.82 (0.66–1.01)	.065	561 (65.7)	0.82
≥ 1	0.83 (0.68–1.03)	.085	505 (59.1)	0.83	0.78 (0.64–0.96)	.019 *	512 (60.0)	0.78
≥ 2	0.83 (0.68–1.02)	.079	460 (53.9)	0.83	0.85 (0.69–1.04)	.107	479 (65.1)	0.84
≥ 3	0.80 (0.66–0.99)	.038 *	409 (47.9)	0.80	0.80 (0.65–0.99)	.035 *	415 (48.6)	0.80
≥ 4	0.83 (0.67–1.02)	.078	369 (43.2)	0.83	0.84 (0.68–1.03)	.090	378 (44.3)	0.83
≥ 5	0.91 (0.73–1.12)	.369	322 (37.7)	0.91	0.92 (0.74–1.13)	.412	322 (37.7)	0.91
≥ 6	0.94 (0.75–1.16)	.550	282 (33.0)	0.93	0.94 (0.76–1.17)	.585	275 (32.2)	0.94
≥ 7	0.92 (0.73–1.15)	.445	252 (29.5)	0.92	0.94 (0.75–1.18)	.612	244 (28.6)	0.94
≥ 8	1.00 (0.79–1.26)	.985	223 (26.1)	1.00	0.99 (0.78–1.26)	.951	199 (23.3)	0.99
≥ 9	1.09 (0.86–1.38)	.488	188 (22.0)	1.09	0.95 (0.74–1.22)	.693	172 (20.1)	0.95
≥ 10	1.13 (0.88–1.45)	.342	161 (18.9)	1.13	0.86 (0.65–1.14)	.296	138 (16.2)	0.86
≥ 11	1.08 (0.82–1.41)	.598	133 (15.6)	1.08	0.92 (0.69–1.24)	.591	112 (13.1)	0.92
≥ 12	1.04 (0.78–1.40)	.772	115 (13.5)	1.05	0.87 (0.63–1.21)	.401	91 (10.7)	0.87
≥ 13	1.04 (0.76–1.42)	.808	97 (11.4)	1.04	1.05 (0.74–1.49)	.766	69 (8.1)	1.05
≥ 14	1.02 (0.74–1.42)	.897	86 (10.1)	1.02	1.32 (0.91–1.90)	.143	53 (6.2)	1.32
≥ 15	1.03 (0.73–1.45)	.882	75 (8.8)	1.03	1.06 (0.68–1.67)	.791	39 (4.6)	1.07
≥ 16	1.02 (0.69–1.50)	.928	59 (6.9)	1.02	1.48 (0.92–2.38)	.110	25 (2.9)	1.49
≥ 17	1.01 (0.66–1.54)	.959	48 (5.6)	1.01	1.24 (0.71–2.16)	.448	20 (2.3)	1.24
≥ 18	0.99 (0.62–1.57)	.954	39 (4.6)	0.98	1.44 (0.79–2.62)	.238	17 (2.0)	1.45
≥ 19	0.96 (0.60–1.54)	.851	36 (4.2)	0.95	1.57 (0.84–2.94)	.162	14 (1.6)	1.58
≥ 20	1.13 (0.69–1.84)	.626	28 (3.3)	1.13	1.96 (1.04–3.67)	.037 *	13 (1.5)	1.99
≥ 21	1.17 (0.71–1.93)	.547	26 (3.0)	1.17	1.77 (0.84–3.75)	.134	10 (1.2)	1.81
≥ 22	1.33 (0.78–2.27)	.295	21 (2.5)	1.35	1.57 (0.59–4.21)	.368	7 (0.8)	1.63
≥ 23	1.56 (0.91–2.67)	.104	19 (2.2)	1.59	1.43 (0.36–5.75)	.614	3 (0.4)	1.47
≥ 24	1.66 (0.91–3.03)	.099	15 (1.8)	1.68	1.43 (0.36–5.75)	.614	3 (0.4)	1.47
≥ 25	1.72 (0.89–3.34)	.108	13 (1.5)	1.75	1.43 (0.36–5.75)	.614	3 (0.4)	1.47
≥ 26	1.66 (0.82–3.34)	.159	11 (1.3)	1.68	0.00 (0.00–E)	.945	1 (0.1)	0.00
≥ 27	1.66 (0.82–3.34)	.159	11 (1.3)	1.68	0.00 (0.00–E)	.945	1 (0.1)	0.00
≥ 28	1.66 (0.82–3.34)	.159	11 (1.3)	1.68	–	–	0 (0.0)	–
≥ 29	1.91 (0.90–4.03)	.091	10 (1.2)	1.96	–	–	0 (0.0)	–
≥ 30	1.96 (0.73–5.27)	.180	6 (0.7)	2.01	–	–	0 (0.0)	–
≥ 31	1.96 (0.73–5.27)	.180	6 (0.7)	2.01	–	–	0 (0.0)	–
≥ 32	2.22 (0.71–6.94)	.170	5 (0.6)	2.24	–	–	0 (0.0)	–
≥ 33	2.22 (0.71–6.94)	.170	5 (0.6)	2.24	–	–	0 (0.0)	–
≥ 34	2.64 (0.84–8.23)	.095	4 (0.5)	2.65	–	–	0 (0.0)	–
≥ 35	2.41 (0.60–9.69)	.215	3 (0.4)	2.44	–	–	0 (0.0)	–
≥ 36	2.41 (0.60–9.69)	.215	3 (0.4)	2.44	–	–	0 (0.0)	–
≥ 37	2.41 (0.60–9.69)	.215	3 (0.4)	2.44	–	–	0 (0.0)	–
≥ 38	2.41 (0.60–9.69)	.215	3 (0.4)	2.44	–	–	0 (0.0)	–
≥ 39	1.80 (0.25–12.80)	.559	2 (0.2)	1.83	–	–	0 (0.0)	–
≥ 40	1.80 (0.25–12.80)	.559	2 (0.2)	1.83	–	–	0 (0.0)	–
≥ 41	6.51 (0.91–46.67)	.062	1 (0.1)	6.54	–	–	0 (0.0)	–
≥ 42	6.51 (0.91–46.67)	.062	1 (0.1)	6.54	–	–	0 (0.0)	–
≥ 43	–	–	0 (0.0)		–	–	0 (0.0)	–

Notes. Increase from Time 1 = baseline to Time 2 = 11-year follow-up; HR = hazard rate; *CI* = confidence interval; * = $p < .05$; RTM = Regression to the Mean; E = very wide. [1]Age as a time-dependent covariate.

variance in SOC scores among those whose SOC was initially weak.

As in a previous study of psychosocial stress and all-cause mortality (Rodgers et al., 2021), the reasons for the association between decreasing SOC and increasing mortality could be biological, psychological, and behavioral. It has been shown that prolonged stress may negatively affect our biological health (e.g., endocrine system, sympathetic-adrenal-medullary system, immune activity, and cognitive perturbations) (Rodgers et al., 2021). A strong SOC may shield us from these effects and help to recover from stressful situations (Eriksson & Lindström, 2006). Weakened SOC may also negatively affect our psychological health (or vice versa) since SOC correlates strongly with depression, and people with weak SOC seem to associate with lesser learned resourcefulness, self-efficacy, locus of control, self-esteem, social skills, and acceptance of disability (Eriksson & Lindström, 2006). Lastly, weakened SOC may associate with behavioral changes, which in turn affect health and mortality. These behavioral changes could include, for example, increasing substance use, consuming more unhealthy foods, or becoming less active (Rodgers et al., 2021). However, our observational study cannot state the causal effects of SOC: decreasing SOC could negatively affect our health or be rather a marker of worsening health.

5. Limitations and future research direction

There are a few limitations in the present study. Firstly, the generalizability of the results is limited: we included only a relatively homogeneous sample of middle-aged Finnish men. Therefore, we recommend that future studies should confirm our findings in a more heterogeneous sample including women, other nationalities, and cultural backgrounds. Secondly, we analyzed only two possible mediators or moderators between the change in SOC and mortality, age, and the baseline SOC level. To clarify, which variables affect the association, more studies are needed. For example, because there is evidence that SOC, especially initially weak SOC, is responsive to negative life events (Volanen et al., 2007), we recommend that future studies should investigate how events during a lifetime affect SOC and predict mortality in different SOC baseline groups.

Table A4
Fully adjusted Model 2 Hazard Rates associated with all-cause mortality among Kuopio Ischaemic Heart Disease Risk Factor (KIHD) study participants for those whose Sense of Coherence (SOC) was measured twice (n = 854) and was increasing. Reference group was 'SOC decreased, remained, or increased less'.

Increase in SOC	Time 1 not adjusted for RTM				Time 1 adjusted for RTM			
Points	*HR* (95% *CI*)	*p*	*n* (%)	*HR*[1]	HR (95% *CI*)	*p*	*n* (%)	*HR*[1]
> 0	0.91 (0.74–1.12)	.358	515 (60.3)	0.89	0.89 (0.72–1.10)	.272	561 (60.0)	0.87
≥ 1	0.90 (0.73–1.11)	.343	505 (59.1)	0.88	0.85 (0.69–1.04)	.118	512 (60.0)	0.82
≥ 2	0.89 (0.72–1.09)	.243	460 (53.9)	0.87	0.92 (0.75–1.13)	.414	479 (56.1)	0.90
≥ 3	0.82 (0.67–1.01)	.060	409 (47.9)	0.81	0.86 (0.70–1.05)	.140	415 (48.6)	0.85
≥ 4	0.85 (0.69–1.05)	.124	369 (43.2)	0.84	0.91 (0.74–1.12)	.385	378 (44.3)	0.91
≥ 5	0.90 (0.73–1.12)	.342	322 (37.7)	0.90	0.98 (0.79–1.21)	.856	322 (37.7)	0.98
≥ 6	0.91 (0.73–1.13)	.397	282 (33.0)	0.90	1.01 (0.81–1.25)	.966	257 (30.1)	1.01
≥ 7	0.88 (0.71–1.11)	.286	252 (29.5)	0.89	1.02 (0.81–1.28)	.893	244 (28.6)	1.02
≥ 8	0.97 (0.77–1.22)	.761	223 (26.1)	0.97	1.06 (0.83–1.35)	.659	199 (23.3)	1.06
≥ 9	1.09 (0.86–1.38)	.493	188 (22.0)	1.10	1.00 (0.77–1.29)	.993	172 (20.1)	1.00
≥ 10	1.06 (0.83–1.37)	.634	161 (18.9)	1.07	0.88 (0.66–1.17)	.384	138 (16.2)	0.88
≥ 11	1.01 (0.77–1.33)	.935	133 (15.6)	1.02	0.93 (0.69–1.25)	.617	112 (13.1)	0.93
≥ 12	0.96 (0.71–1.29)	.770	115 (13.5)	0.96	0.91 (0.65–1.28)	.599	91 (10.7)	0.92
≥ 13	0.94 (0.69–1.28)	.694	97 (11.4)	0.94	1.10 (0.77–1.57)	.614	69 (8.1)	1.10
≥ 14	0.90 (0.65–1.26)	.545	86 (10.1)	0.90	1.34 (0.92–1.95)	.128	53 (6.2)	1.36
≥ 15	0.95 (0.67–1.34)	.753	75 (8.8)	0.95	1.12 (0.71–1.78)	.631	39 (4.6)	1.14
≥ 16	1.04 (0.70–1.55)	.831	59 (6.9)	1.05	1.55 (0.95–2.53)	.077	25 (2.9)	1.59
≥ 17	1.06 (0.69–1.63)	.782	48 (5.6)	1.06	1.27 (0.72–2.24)	.403	20 (2.3)	1.29
≥ 18	1.11 (0.69–1.78)	.680	39 (4.6)	1.10	1.55 (0.84–2.86)	.161	17 (2.0)	1.59
≥ 19	1.06 (0.65–1.73)	.803	36 (4.2)	1.06	1.60 (0.84–3.05)	.156	14 (1.6)	1.64
≥ 20	1.25 (0.75–2.06)	.393	28 (3.3)	1.24	1.82 (0.95–3.47)	.070	13 (1.5)	1.86
≥ 21	1.25 (0.75–2.10)	.391	26 (3.0)	1.25	1.72 (0.80–3.71)	.167	10 (1.2)	1.80
≥ 22	1.36 (0.79–2.35)	.270	21 (2.5)	1.36	1.50 (0.55–4.09)	.427	7 (0.8)	1.63
≥ 23	1.53 (0.89–2.64)	.128	19 (2.2)	1.54	1.41 (0.34–5.81)	.633	3 (0.4)	1.51
≥ 24	1.49 (0.81–2.74)	.204	15 (1.8)	1.49	1.41 (0.34–5.81)	.633	3 (0.4)	1.51
≥ 25	1.47 (0.75–2.88)	.263	13 (1.5)	1.48	1.41 (0.34–5.81)	.633	3 (0.4)	1.51
≥ 26	1.42 (0.70–2.89)	.335	11 (1.3)	1.44	0.00 (0.00–E)	.951	1 (0.1)	0.00
≥ 27	1.42 (0.70–2.89)	.335	11 (1.3)	1.44	0.00 (0.00–E)	.951	1 (0.1)	0.00
≥ 28	1.42 (0.70–2.89)	.335	11 (1.3)	1.44	–	–	0 (0.0)	–
≥ 29	1.62 (0.76–3.48)	.214	10 (1.2)	1.69	–	–	0 (0.0)	–
≥ 30	1.41 (0.52–3.84)	.504	6 (0.7)	1.44	–	–	0 (0.0)	–
≥ 31	1.41 (0.52–3.84)	.504	6 (0.7)	1.44	–	–	0 (0.0)	–
≥ 32	1.21 (0.38–3.86)	.752	5 (0.6)	1.23	–	–	0 (0.0)	–
≥ 33	1.21 (0.38–3.86)	.752	5 (0.6)	1.23	–	–	0 (0.0)	–
≥ 34	1.45 (0.45–4.62)	.534	4 (0.5)	1.47	–	–	0 (0.0)	–
≥ 35	1.34 (0.33–5.50)	.681	3 (0.4)	1.39	–	–	0 (0.0)	–
≥ 36	1.34 (0.33–5.50)	.681	3 (0.4)	1.39	–	–	0 (0.0)	–
≥ 37	1.34 (0.33–5.50)	.681	3 (0.4)	1.39	–	–	0 (0.0)	–
≥ 38	1.34 (0.33–5.50)	.681	3 (0.4)	1.39	–	–	0 (0.0)	–
≥ 39	1.22 (0.17–8.94)	.847	2 (0.2)	1.34	–	–	0 (0.0)	–
≥ 40	1.22 (0.17–8.94)	.847	2 (0.2)	1.34	–	–	0 (0.0)	–
≥ 41	1.80 (0.24–13.72)	.573	1 (0.1)	2.02	–	–	0 (0.0)	–
≥ 42	1.80 (0.24–13.72)	.573	1 (0.1)	2.02	–	–	0 (0.0)	–
≥ 43	–	–	0 (0.0)	–	–	–	0 (0.0)	–

Notes. Increase from Time 1 = baseline to Time 2 = 11-year follow-up; HR = hazard rate; *CI* = confidence interval; * = $p < .05$; RTM = Regression to the Mean; E = very wide. [1]Age, total cholesterol concentration, and years of education as time-dependent covariates.

6. Conclusions

The present study demonstrates that decreasing SOC during late midlife is associated with increased mortality hazard among Finnish men, even after several other mortality risk factors together with their time-dependencies and the RTM phenomenon are taken into consideration. Furthermore, the greater the decrease in SOC is, the greater the increase in the mortality hazard. Based on these findings, and due to the positive correlation of SOC with the quality of life, it could be worthwhile to create SOC supporting policies, strategies, or plans, aimed at the aging population. These measures could help to decrease mortality after midlife and increase the quality of life in older age.

Appendix

See Tables A1–A4.

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