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RESEARCH

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Social integration and mortality across the life course: findings from the Tromsø study

Ainhoa Ugarteche Pérez^{1*}, Nadine El Rashidi¹, Erlend Hoftun Farbu², Raphaële Castagné¹, Sameline Grimsgaard², Tom Wilsgaard², Dragana Vuckovic^{3,4}, David Tang^{3,4}, Marc Chadeau-Hyam^{3,4}, Torkjel M Sandanger², Cyrille Delpierre¹, Michelle Kelly-Irving¹ and Lola Neufcourt¹

Abstract

Context Poor social relationships have been associated with increased mortality risk. Configurations of social relationships vary across the life course, affecting the association with mortality. We investigated the relationship between social integration and mortality in four age groups and to examine three blocks of potential intermediate variables.

Methods Tromsø Study wave 4 and 6 data and mortality events in the Norwegian Population Registry were linked. A three-level (low, medium, high) social integration index (SII) was constructed from scores to three related items: social participation, living with spouse and frequency of contact with friends. Adjusted cox-regression models and including intermediate variables in blocks (1:behavioral, 2:psychosocial resources, 3:biological markers) were conducted in the study population ($N=9879$) and in age-stratified subsamples: [25–52]y. ($N=6785$); [53–59]y. ($N=1679$); [60–65]y. ($N=846$); [66–74]y. ($N=569$). Additional sex-stratified and causes-of-death models (cancer, cardiovascular diseases, neuropsychiatric conditions and other deaths) were constructed.

Results A graded SII and mortality risk association was observed in confounder-adjusted models in the total study population: individuals with medium and low SII had incrementally higher risk of mortality compared to those with high SII ($HR_{\text{mediumSII}}[95\%CI] = 1.15 [1.02; 1.31]$, $HR_{\text{lowSII}}[95\%CI] = 1.42[1.14; 1.76]$). This association was mainly observed in the [53–59]y. group and among men. Health behaviors and the psychosocial resources block partly attenuated the association, but biological markers did not. Low SII was particularly associated with deaths from “other causes”, including respiratory conditions, and to a lesser extent with deaths from cardiovascular disease and cancer.

Conclusion Our study consolidates evidence on the relationship between social integration and mortality risk, especially in mid-adulthood, and suggests some potential underlying pathways. To reduce mortality risk inequalities, we encourage structurally addressing social marginalization.

Keywords Social relations, Life-course, Mortality

*Correspondence:

Ainhoa Ugarteche Pérez
ainhoa.ugarteche-perez@inserm.fr

¹EQUITY research team, Inserm, CERPOP-UMR1295, Université Toulouse III
Paul Sabatier, 37 Allées Jules Guesde, Toulouse 31000, France

²Department of Community Medicine, Faculty of Health Sciences, UiT the Arctic University of Norway, Tromsø, Norway

³Department of Epidemiology and Biostatistics, School of Public Health, Faculty of Medicine, Imperial College London, London, UK

⁴MRC Centre for Environment and Health, School of Public Health, Imperial College London, London, UK



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Introduction

Having poor social relationships has been linked with adverse health outcomes including cardiovascular disease (CVD), cancer [1], depression [2] and cognitive decline [3]. Additionally, social isolation, which can be defined as a lack of social relationships, is described as a main risk factor of all-cause mortality [4] and recent meta-analyses using large population data from high-income countries, mainly in North America, Europe and, Asia, confirmed evidence on this link [5, 6]. Individuals with poorer social relationships or socially isolated had a greater all-cause mortality risk compared to their counterparts with adequate social relationships.

The association between social relationships and mortality has been examined using measures describing functional and structural dimensions of social relationships. Functional measures such as social support, refer to social relationships that provide resources or aid for informational or emotional processing. Structural measures such as social networks or number of social interactions, measure the size of an individual's social sphere. Some unidimensional measures have been related to excess mortality, including low perception of social support [7, 8], from the functional dimension, and living alone or having a low social contact and participation [9], from the structural dimension. However, multidimensional constructs that combine both functional and structural measures of social relationships were found to be most predictive of mortality compared to unidimensional measures in a meta-analysis from [5]. Few validated multidimensional measures of social relationships exist [10, 11] and often researchers are limited by data availability. When possible, multidimensional constructs of social relationships are favored in epidemiological studies of mortality since they are comprehensive and, therefore, more adapted to investigate the pathways and intermediate factors that link social relationships and health.

One of the pathways that may explain the association between social relationships and mortality is the behavioral pathway. Previous studies have shown that socially isolated people were more likely to adopt harmful behaviors such as smoking, more sedentary behaviors, low fruit and vegetable intake and lower diet quality, supporting the hypothesis that the influence of network members (e.g., spouses, family members, friends) can encourage individuals to exhibit health-promoting behaviors and discourage them from health-damaging behaviors [12–15]. Another pathway potentially underlying the relationship between social relationships and mortality is the psychosocial pathway, which may involve various factors like coping resources or self-perception of health. Social relationships and network support can enhance peoples' beliefs in their abilities and result in self-confidence, self

efficacy and a sense of control, which in turn, facilitate effective coping behaviors when facing stressors across a variety of situations [16, 17]. In addition, social support has been consistently positively associated with self-rated health across different cultural, economic and geographic settings [18], suggesting that those with strong social ties are likely to have multiple resources (stress reduction, shared experiences, better access to information) that enable them to maintain better overall health [18, 19]. Another pathway by which social relationships influence mortality is the biological pathway, which involves a direct effect through the physiological response to stress. A systematic review by Wiley *et al.* (2017) [20] reported that, in the majority of studies looking at social relationships and allostatic load – a measure of cumulative physiological wear-and-tear- a greater social support was associated with lower levels of allostatic load.

So far, no studies investigating possible mechanisms that relate social relationship and mortality analyze, together, behavioral, psychosocial resources and biological pathways. Additionally, an important part of the existing literature is conducted among older adult populations.

The configurations of social relationships as well as the health promoting influences of intermediate factors may vary across the life course. The life course stage and overall context may modify the composition and nature of social relationships and their influence on health [15, 21, 22]. For example, as we age through adulthood, selective narrowing of social relationships has been linked with improved mental health and life satisfaction and, at the same time, with increased cardiovascular reactivity, while, in younger adult populations inverse associations, respectively, are observed [23, 24]. Such varying patterns of association between social relationships and health over the life course merit further study.

Analyzing data from a Norwegian population cohort of adults, the goals of this paper are, first, to explore the relationship between a multidimensional measure of social relationships, adapted from the Social Integration Index (SII) proposed by Berkman and Syme [25], and all-cause mortality over 12 years, in the total population and in four derived age-groups; and second, to assess the potential pathways in this association by exploring the influence of three blocks of intermediate factors: (1) health behaviors, (2) self-perception of health and ability to cope with difficulties as one block of variables reflecting psychosocial resources, and (3) a composite measure of biological health as an indicator of physiological response to stress. Additionally, to explore potential variations in the association, we will analyze sex-stratified samples and examine the relationship between the SII and four groups of specific causes of deaths (cancer,

CVD, neuropsychiatric disorders and ‘other causes,’ selected based on their prevalence in Norway) in the total population.

Methods

Data & study population

The data used were part of the Tromsø study, an on-going population-based, prospective, cohort study of participants residing in the Tromsø municipality, in Northern Norway. The study initiated in 1974 and has completed seven data collection waves (wave 7 in 2015–2016). It recorded sociodemographic, health and biomedical data through self-completion questionnaires and medical examinations. Participants were invited by mail using Norwegian Population Registry addresses. Questionnaires translated into English are found on the study webpage (www.tromsostudy.com). Ethics and study protocol approval was provided by the Regional Committee of Medical, Health Research Ethics of Northern Norway and Data Inspectorate of Norway and was assessed by the Norwegian Centre for Research Data. Participants provided informed consent in forms and data were securely stored in an IT system approved by the Norwegian Data Protection Authority. All-cause mortality and specific causes of death data were available through the Norwegian Population Registry and the National Causes of Death Registry. Further details on design and data collection procedures are described elsewhere [26, 27].

The present study analyzes data from participants who attended both waves 4 (1994–1995) and 6 (2007–2008) ($N=10\,322$), where data were collected on the main exposure (social integration) and intermediate factors for pathway analysis: health behaviors, health perception, coping with difficulties and biological health measures. These two specific waves were selected for the presence of variables of interest and close in time to have an early as possible start of follow-up.

Social integration index

The SII was adapted from the one introduced by Berkman and Syme [25] using data from wave 4 to study the relationship between social relationships and mortality. The SII combined structural and functional aspects of participant’s social relationships based on answers to three questions on (1) living with their spouse, (2) frequency of going out to gatherings and, (3) number of close friends they can contact. Each of the three SII components were granted a score between 0 and 2. Those living with their spouse received a score of 2 and, otherwise, a score of 0 was attributed. “Never or rarely,” “monthly” and “weekly” answers to the going out on gatherings question were allocated scores of 0, 1 and 2, respectively. Number of friends that participants could contact were categorized into tertiles: [0:2] friends = 0, [3:4] friends = 1 and, 5 or

more friends = 2. Finally, the three components were summed to create a social integration index on a [0:6] scale. The social integration index was divided into three categories, low [0:1], medium [2:4] and high [5:6], ensuring that both extreme categories (low and high), despite their design specificities, were large enough in size for the analyses. Based on the scoring described above, those in the high SII group were systematically living with their spouse.

Mortality

A binary variable indicated the occurrence of death. Mortality events were included from wave 6 (2007–2008) until the last specific causes of death registry update, December 31st of 2020; for a 12-year follow-up. Time to mortality was scaled on participant’s age. Study entry-points corresponding to participant’s age at wave 6 were staggered and right-censoring was conducted. Scenarios of censoring were emigration or end of follow-up, whichever occurred first.

Additionally, four binary causes of death variables were constructed (1 for death by event of interest, 0 otherwise). Events of interest were the most common causes of death in Norway, namely (1) cancers, (2) cardiovascular diseases, (3) neuropsychiatric conditions and, a last death category, (4) other causes, including all other causes of death not categorized into events of interest 1–3. Cases were coded and identified using the International Classification of Disease- 10 (ICD-10) nomenclature from the World Health Organization (Details are available in S.Tb1). A breakdown by sub-pathology is available in S.Tb2.

Covariates

Covariates were conceptually classified into confounders or intermediate variables meant to assess the possible mediating pathways between SII and mortality. The relationship of covariates to the exposure and outcome are graphically presented in a conceptual diagram (Fig. 1).

Confounders included sex, age, education, presence of psychological problems and health conditions, collected at wave 4. Intermediate variables were collected at wave 6 and divided into three blocks: (1) smoking, physical activity, alcohol consumption (behavioral pathway), (2) perception of health and coping with difficulties (psychosocial pathway) (3) and a biological health score (BHS) (biological pathway). Medical treatments used in wave 6 (for blood pressure, diabetes and lipid control) were added as confounders specific to the BHS intermediate pathway. All variables were self-reported except for the biomarkers composing the BHS which were measured in blood samples collected during a medical examination [27].

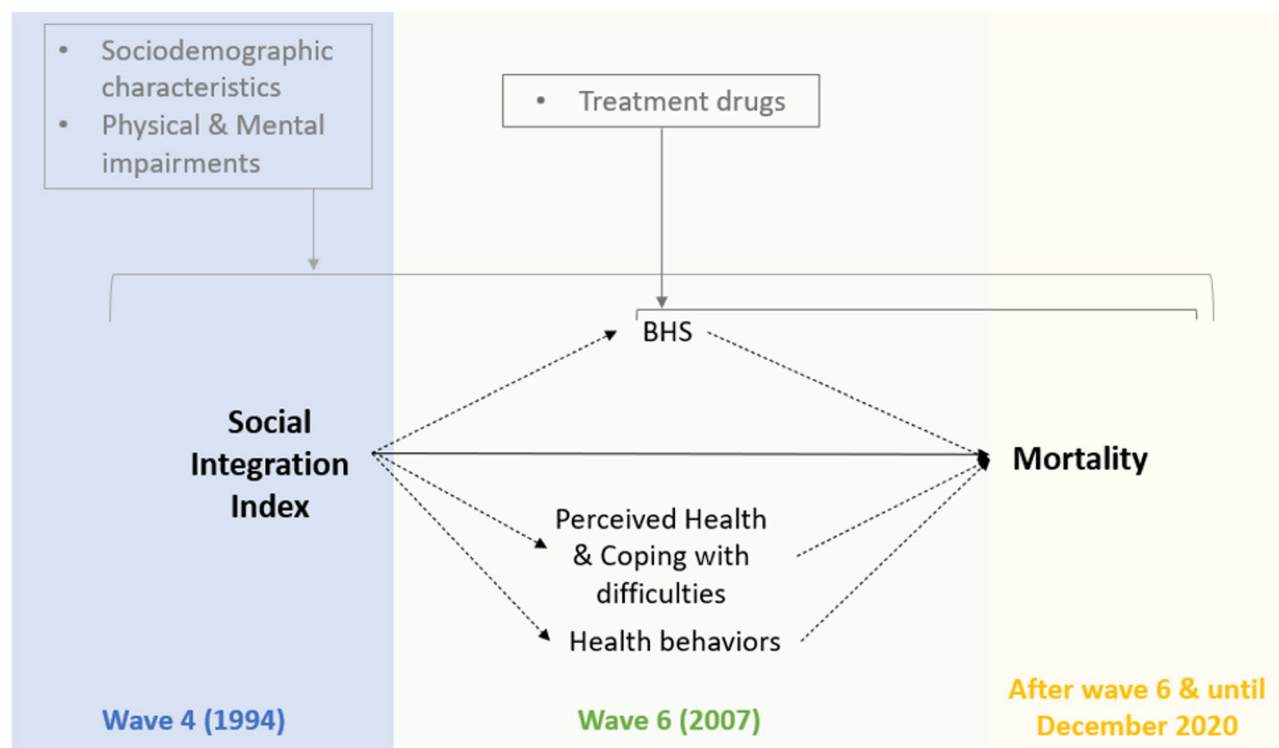


Fig. 1 Conceptual diagram describing relationships between study variables and periods of measurement. Grey arrows: confounding pathways, dotted black lines: intermediate pathways, solid black line: the main investigated relationship

Attained education (five-levels, from primary school to over 4 years of university), psychological problems (No, Yes), smoking (Never, In the Past, Currently), medical treatment (Never used, In the Past, Currently), physical activity (Never, Monthly, Weekly, Approximately every day), alcohol consumption (Monthly, Weekly, Never), perceived health (Very bad, Bad, Neither good nor bad, Good, Excellent) and coping with difficulties (“Have you felt unable to cope with your difficulties during the past couple of weeks?” Not at all, No more than usual, Rather more than usual, Much more than usual) were kept in their raw configuration.

A health conditions score was created from 12 potentially chronic illnesses self-reported at wave 4 (cancer, chronic bronchitis, osteoporosis, fibromyalgia, liver disease, thyroid disease, epilepsy and, surveyed only to participants above 70 years, arthrosis, cataract, glaucoma, Parkinson) and divided into a three-category variable (0, 1 and 2 or more conditions).

The BHS theoretically aims to measure an overall physiological dysregulation, also referred to as “wear-and-tear” [28, 29]. This study follows methodological considerations already developed in wave 6 of the Tromsø study [30]; we identified individuals at risk of poor biological health based on their biomarker concentration distribution by sex and by age groups (below 60 and 60 years or above) likely showing clinically-relevant differences.

According to data availability, 11 biomarkers were used, representing the cardiovascular (systolic, diastolic blood pressure and pulse rate), metabolic (total cholesterol, high-density lipoprotein (HDL), low density lipoprotein (LDL), triglycerides, glucose and glycated haemoglobin (HbA1C)) and inflammatory (C-reactive protein (CRP) and fibrinogen) systems. A score of 1 was attributed if the biomarker’s concentration was in the upper quartile (except for HDL for which it was the lowest quartile) and, otherwise, of 0. All individual biomarker scores by participant were added to create a BHS of range [0:11].

Statistical analysis

Analyses were conducted in the study population ($N=9879$; 1754 deaths) as well as in four age-stratified subsamples. Stratification cut-offs were defined using age at baseline (wave 4) and such that each sample included approximately the same number of deaths to ensure that there was a substantial number of events in each sample. Consequently, subsamples had varying sizes and lengths of intervals: [25–52] years ($N=6785$, 69%; 405 deaths), [53–59] years ($N=1679$, 17%; 412 deaths), [60–65] years ($N=846$, 9%; 454 deaths), [66–74] years ($N=569$, 6%; 483 deaths). Analyses were performed in R using R studio environment version (4.1.2). Packages used include ggplot2, survival, survminer and mice.

Descriptive analysis

Means with standard deviations (SD) and proportions were reported for continuous variables and categorical variables respectively. Survival probabilities by SII groups were visually described with Kaplan Meier (KM) curves using age as time-scale and accounting for age-dependent staggered entries.

Multivariable analyses

Cox regression models with staggered entry, using age as time-scale, were created to estimate the risk of all-cause mortality related to the SII. High SII was used as the reference level. Estimates were hazard ratios (HR) with 95% confidence intervals (CI).

Sequences of models were constructed, starting with a crude model (Model 0) and then, adjusting for all baseline confounders at wave 4 (Model 1). Blocks of intermediate variables were independently added to Model 1 to assess an attenuation of the association between SII and mortality, which was hypothesized to suggest potential mediation. This resulted in Models 1a (+ Health Behaviors), 1b (+ Perception variables & coping with difficulties), 1c (+ BHS) and 1c-bis, accounting for BHS specific confounders (+ BHS & medical treatments at wave 6). Model 2 adjusted for all variables at once. Cox proportional hazard assumptions were verified using graphical diagnostics of the Schoenfeld residuals.

Complementary analyses

Main analyses were reproduced in sex-stratified samples from the total study population and from the age subsamples to uncover any sex-specific differences in the SII-mortality relationship. Similar descriptive and multivariable analyses as in the main analyses were performed with specific causes of mortality in the total sample ($N=9879$) to deepen the examination of the SII-mortality relationship. Censoring additionally included death by another cause than the one of interest.

Missing data

To control for possible selection bias, missing data on exposure, confounders and intermediate variables were imputed using the MICE R package [31]. For composite variables, namely the SII and the BHS, individual components were imputed and the final scores were calculated using imputed data. The percentage of missing values across the 25 incomplete variables and variable components varied between 0.3% and 15.7%. In total 3393 (34%) out of 9879 participants had partial incomplete data.

As an exception, missing values in the individual components of the score of medical conditions were set to 0 due to the inclusion of items specific to individuals aged over 70, which were not suitable for multiple imputation in the total sample. All variables used in the study

were used as predictor variables. Thirty imputations, with thirty iterations, were conducted under the missing-at-random assumption. Rubin's combination rules were used to obtain summary regression estimates [32]. Age-stratified subsamples were then derived from the imputed datasets.

Sensitivity analyses

All main analyses on all-cause mortality were reproduced in the sample of complete cases ($N=6486$). Complete case analysis was not performed in age-stratified samples because, when creating age-intervals based on number of deaths, boundaries were not identical as in main analyses.

Results

Characteristics of the study population and by age-groups

Out of a sample of the 10 322 individuals participating in both wave 4 and wave 6, we excluded 443 individuals with missing data on specific cause of death to create our study population of 9879 participants (Fig. 2).

The study population and its derived age-stratified subsamples are described in Table 1.

During a mean follow-up period of 12 years (SD: 2), a total of 1754 (18%) individuals died. Out of all deaths, 630 (36%) were from cancer, 442 (25%) from CVD, 228 (13%) from neuropsychiatric conditions and 454 (26%) from other causes.

The mean age at inclusion of the study population was 47 years (SD: 12), with 46% of the sample being men. The majority had a medium SII (70%), 6% had a low SII and 25% a high SII. Most of the population had a low level of education (primary/middle school: 62%) and did not have any medical conditions (72%), a small proportion experienced psychological problems (8%) and blood pressure treatment was the most used medication (25%). Regarding health behaviors, the majority exercised weekly (59%), were monthly alcohol consumers (66%) and were previous or current smokers (63%). Most participants had good/excellent perceived health (65%) and reported not finding it hard to cope with difficulties (65%). The average BHS score was 2.9 (SD = 2.1).

In [25–52], [53–59], [60–65], [66–74] age groups, the number of deaths were, respectively: 405 (6%), 412 (25%), 454 (54%) and 483 (85%). Descriptive statistics in age stratified subsamples are similar to the study population description for the two younger age groups and are different for the elderly groups.

Compared to the total study population, in the [60–65] and [66–74] age groups, individuals were more likely to have a low SII and a lower educational level but less likely to smoke; they were fewer to report “not coping with difficulties” and more frequently reported the use of blood pressure and lipid treatments. In particular, those in the [66:74] age group were more often men, had more

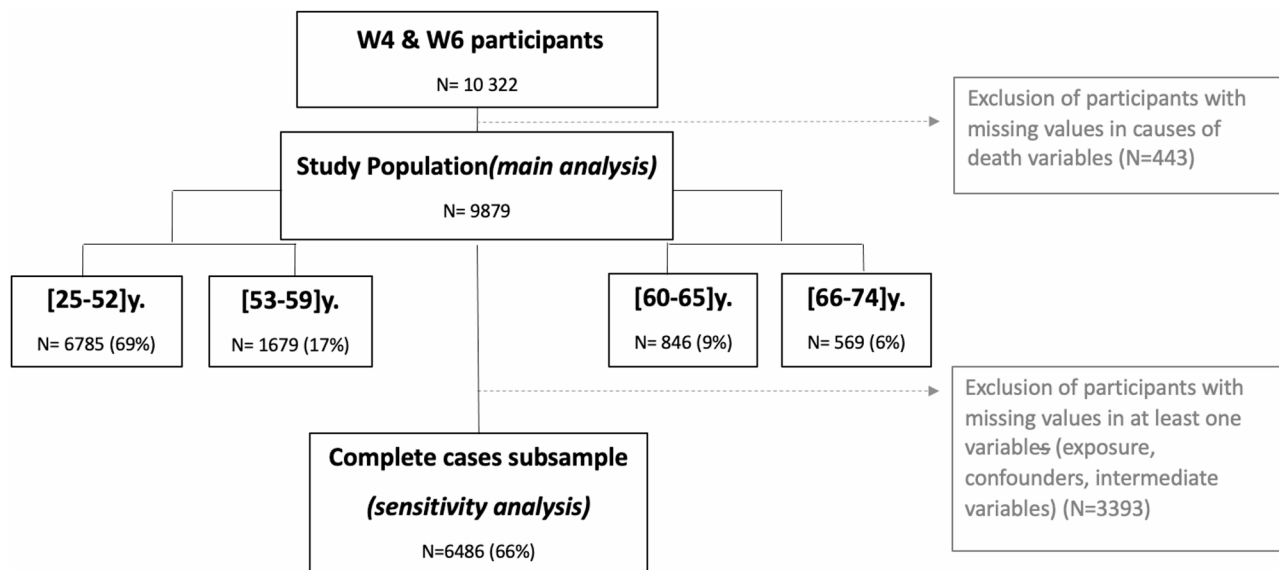


Fig. 2 Flowchart of selection of study population and other samples used in main and supplementary analyses

medical problems, reported more often to “never” exercise and to have “neither good nor bad” perceived health.

Survival probability curves by SII categories in the total study population (S. Figure 1) show a gradient particularly among those who were below 85 years, where those with a low SII have a steeper descending curve compared to those with a medium SII and, even steeper, compared to those with a high SII. Across age subsample survival probability curves, similar trends are found for the [53:59] age group (S. Figure 2).

SII and future risk of death

Figure 3 presents the Cox regression results for the association between SII and mortality risk adjusted for all study variables (first confounders, then intermediate variables by blocks) in the total population and by age groups. Detailed estimates are available in S.Tb3. The proportionality of hazards assumption over time was met in the fully adjusted model across all samples. All estimated hazard ratios describe a mortality risk relative to that of individuals in the reference exposure group: high SII.

In the total study population, analyses showed a gradient in the association between SII and mortality risk after adjustment for confounders (Model 1). Compared to participants in the high SII group, those with a medium SII and a low SII had a higher risk of mortality, with a larger effect size for the low SII group ($HR_{\text{mediumSII}}[95\%CI] = 1.15 [1.02; 1.31]$, $HR_{\text{lowSII}}[95\%CI] = 1.42 [1.14; 1.76]$). Subsequent adjustment for health behaviors ($HR_{\text{lowSII}}[95\%CI] = 1.26 [1.01; 1.57]$, Model 1a) or perception of health and coping with difficulties ($HR_{\text{lowSII}}[95\%CI] = 1.31 [1.05; 1.63]$, Model 1b) reduced the mortality risk while adjustment for the

BHS did not ($HR_{\text{lowSII}}[95\%CI] = 1.38 [1.1; 1.73]$, Model 1c). In the fully adjusted model (Model 2), the risk of mortality tends to remain higher for the low SII group ($HR_{\text{lowSII}}[95\%CI] = 1.19 [0.95; 1.49]$).

In the age-stratified subsamples, after adjusting for confounders (Model 1), a graded relationship between SII and mortality risk was observed in the two youngest age groups, particularly marked in the [53–59]y. group: individuals in the medium SII group presented a higher risk of mortality compared to those in the high SII group ($HR_{[53;59]y}[95\%CI] = 1.46 [1.10; 1.93]$) and this association was even stronger for individuals in the low SII group ($HR_{[53;59]y}[95\%CI] = 2.17 [1.38; 3.43]$). Similar to what was found in the total study population, adjustment for health behaviors or for perceived health and coping attenuated the association but this was not observed when adjusting for the BHS. In the fully adjusted model, the association persisted in the low SII group ($HR_{[53;59]y}[95\%CI] = 1.73 [1.08; 2.77]$).

Complementary analyses on sex-specific trends

Figure 4 and S. Figure 3 show results of cox regression analyses in sex-stratified samples of the total population and age subsamples. In men, a higher mortality risk was found for those with a low SII, compared to those with a high SII, and particularly in the three youngest age groups. Adjustment by confounders and subsequently by blocks of intermediate variables, notably psychosocial variables and health behaviors, attenuated this association. In fully adjusted models, the association with mortality remained marked only for individuals in the [53–59]y. and [60–65]y. groups. No difference was observed among women.

Table 1 Descriptive characteristics in the total study population and by age groups. The Tromsø study 1994–2016

Variables	Statistics: N(%) or mean(sd)				
	Study population (N=9879)	[25–52]y. (N=6785)	[53–59]y. (N=1679)	[60–65]y. (N=846)	[66–74]y. (N=569)
Exposure and Outcome					
Social Integration Index					
High	1954 (24.9)	1432 (25.4)	319 (24.4)	132 (22.4)	71 (21.6)
Medium	5468 (69.6)	3915 (69.5)	916 (70.1)	411 (69.7)	226 (68.9)
Low	439 (5.6)	290 (5.1)	71 (5.4)	47 (8)	31 (9.5)
Missing*	2018	1148	373	256	241
Mortality					
Alive	8125 (82.2)	6380 (94)	1267 (75.5)	392 (46.3)	86 (15.1)
Dead	1754 (17.8)	405 (6)	412 (24.5)	454 (53.7)	483 (84.9)
Cancer	630 (6.4)	229 (3.4)	161 (9.6)	139 (16.4)	101 (17.8)
CVD	442 (4.5)	73 (1.1)	89 (5.3)	120 (14.2)	160 (28.1)
Neuropsychiatric	228 (2.3)	32 (0.5)	63 (3.8)	72 (8.5)	61 (10.7)
Other causes	454 (4.6)	71 (1)	99 (5.9)	123 (14.5)	161 (28.3)
Confounders					
Age (wave 4)	46.6 (11.7)	40.6 (8.4)	55.6 (2)	62.3 (1.7)	68.8 (2.4)
Sex (wave 4)					
Female	5342 (54.1)	3686 (54.3)	863 (51.4)	445 (52.6)	348 (61.2)
Male	4537 (45.9)	3099 (45.7)	816 (48.6)	401 (47.4)	221 (38.8)
Attained education (wave 4)					
University, ≥ 4 years	1370 (13.9)	1109 (16.4)	187 (11.2)	50 (5.9)	24 (4.3)
University, < 4 years	1535 (15.6)	1200 (17.7)	206 (12.3)	89 (10.6)	40 (7.1)
High school diploma	826 (8.4)	725 (10.7)	61 (3.6)	26 (3.1)	14 (2.5)
Middle school	2842 (28.9)	2024 (29.9)	467 (27.9)	207 (24.6)	144 (25.6)
Primary school	3277 (33.3)	1715 (25.3)	751 (44.9)	470 (55.8)	341 (60.6)
Missing	29	12	7	4	6
Psychological problems (wave 4)					
No	8045 (91.5)	5598 (91.9)	1345 (90.6)	682 (91.7)	420 (89.7)
Yes	745 (8.5)	496 (8.1)	139 (9.4)	62 (8.3)	48 (10.3)
Missing	1089	691	195	102	101
Medical conditions (wave 4)					
0	7098 (71.8)	5063 (74.6)	1158 (69)	567 (67)	310 (54.5)
1	2190 (22.2)	1421 (20.9)	401 (23.9)	204 (24.1)	164 (28.8)
2 or more	591 (6)	301 (4.4)	120 (7.1)	75 (8.9)	95 (16.7)
Blood pressure treatment (wave 6)					
Never used	7160 (73.6)	5443 (80.9)	1036 (63.2)	423 (51.5)	258 (47.4)
In the Past	171 (1.8)	113 (1.7)	30 (1.8)	18 (2.2)	10 (1.8)
Currently	2401 (24.7)	1171 (17.4)	574 (35)	380 (46.3)	276 (50.7)
Missing	147	58	39	25	25
Lipid lowering treatment (wave 6)					
Never used	7973 (82.2)	5940 (88.2)	1149 (70.1)	522 (64.8)	362 (69.6)
In the Past	168 (1.7)	85 (1.3)	43 (2.6)	24 (3)	16 (3.1)
Currently	1555 (16)	708 (10.5)	446 (27.2)	259 (32.2)	142 (27.3)
Missing	183	52	41	41	49
Diabetes treatment (wave 6)					
Never used	9276 (96.1)	6508 (97.1)	1513 (93.1)	764 (95.4)	491 (93.7)
In the Past	33 (0.3)	18 (0.3)	6 (0.4)	3 (0.4)	6 (1.1)
Currently	344 (3.6)	177 (2.6)	106 (6.5)	34 (4.2)	27 (5.2)
Missing	226	82	54	45	45
Intermediate variables at wave 6					
Smoking					

Table 1 (continued)

Variables	Statistics: N(%) or mean(sd)				
	Study population (N = 9879)	[25–52]y. (N = 6785)	[53–59]y. (N = 1679)	[60–65]y. (N = 846)	[66–74]y. (N = 569)
Never	3575 (36.8)	2466 (36.7)	587 (35.7)	292 (35.7)	230 (42.5)
In the Past	4255 (43.8)	2800 (41.7)	786 (47.8)	412 (50.4)	257 (47.5)
Currently	1891 (19.5)	1451 (21.6)	273 (16.6)	113 (13.8)	54 (10)
Missing	158	68	33	29	28
Physical activity					
Approx. everyday	1871 (19.5)	1196 (17.9)	376 (23.3)	172 (22.5)	127 (25.3)
Weekly	5664 (59.1)	4091 (61.1)	936 (58)	424 (55.4)	213 (42.5)
Monthly	1476 (15.4)	1128 (16.8)	203 (12.6)	93 (12.1)	52 (10.4)
Never	565 (5.9)	280 (4.2)	99 (6.1)	77 (10.1)	109 (21.8)
Missing	303	90	65	80	68
Alcohol consumption					
Monthly	6412 (65.9)	4617 (68.5)	1038 (62.9)	489 (60.5)	268 (50.5)
Weekly	2145 (22.1)	1646 (24.4)	332 (20.1)	112 (13.9)	55 (10.4)
Never	1170 (12)	476 (7.1)	279 (16.9)	207 (25.6)	208 (39.2)
Missing	152	46	30	38	38
Perceived Health					
Excellent	1378 (14.1)	1137 (16.9)	163 (9.8)	57 (6.9)	21 (3.8)
Good	4971 (50.8)	3496 (51.9)	877 (52.7)	377 (45.5)	221 (39.6)
Neither good nor bad	2904 (29.7)	1781 (26.4)	539 (32.4)	327 (39.5)	257 (46.1)
Bad	494 (5)	304 (4.5)	74 (4.4)	61 (7.4)	55 (9.9)
Very bad	38 (0.4)	18 (0.3)	10 (0.6)	6 (0.7)	4 (0.7)
Missing	94	49	16	18	11
Coping with difficulties					
Not at all	5954 (65.3)	4220 (66.3)	1053 (68.2)	442 (59.2)	239 (51.4)
No more than usual	2866 (31.4)	1931 (30.3)	449 (29.1)	282 (37.8)	204 (43.9)
Rather more than usual	248 (2.7)	181 (2.8)	36 (2.3)	15 (2)	16 (3.4)
Much more than usual	51 (0.6)	33 (0.5)	5 (0.3)	7 (0.9)	6 (1.3)
Missing	760	420	136 (8.1)	100 (11.8)	104 (18.3)
BHScore	2.9 (2.1)	2.9 (2.2)	2.9 (2)	3 (2)	3.4 (2)
Missing**	348	215	50	53	30

*1550 NAs in "Number of friends to talk to", 1085 in NAs in "Living with spouse" & 975 NAs in "Going out on gatherings" components

**Out of 348 NAs, 107 individuals are missing for 5 or more biomarkers of the score

Complementary analyses on specific causes of death

S. Figure 4 and S.Tb4 show results of the cox regression analyses between SII and specific causes of death in the total population.

Overall, after adjusting for confounders, a higher risk of mortality was observed in participants with a low SII for the four specific causes of death and the association was particularly strong in the heterogeneous group "other causes". Associations between SII and specific causes of death were attenuated when accounting for behaviors and to a lesser extent, when adjusting for perceived health and coping with difficulties. Adjustment by the BHS did not produce any risk changes.

Sensitivity analyses

In complete cases, associations between SII and mortality risk were stronger, especially in the low SII group. Similar to what was reported in the imputed total sample,

confounder-adjusted associations were reduced but persisted after adjustment for health behaviors (Model 1a) and perceived health and coping with difficulties (Model 1b) and, adjustment for the BHS did not influence the association (Model 1c) (S.Tb5).

Discussion

In this study, we found an association between a measure of social integration and all-cause mortality risk in a population-based cohort from northern Norway. We observed a graded relationship across categories of social integration with the risk of mortality increasing as social integration decreased. Health behaviors, and factors related to psychosocial resources such as ability to cope with difficulties and perception of health may deserve attention as intermediate pathways that help explain part of this relationship. In age-stratified samples, the relationship between social integration and mortality was

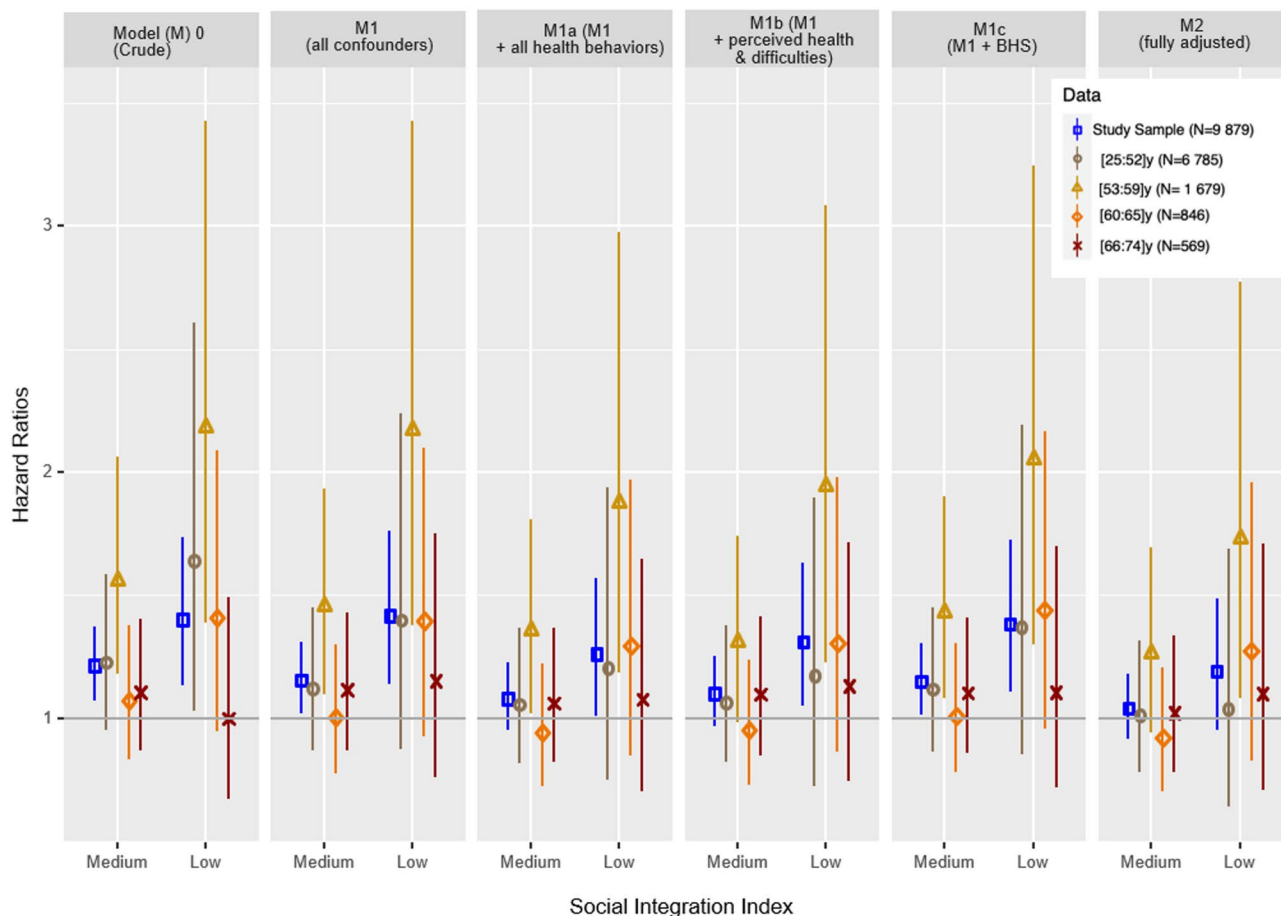


Fig. 3 Hazard ratios for all-cause mortality according to SII groups in the total study population and by age groups. The Tromsø Study 1994–2016

mainly observed in the [53–59]y. group, but less clearly in the three other age groups ([25–52]y.; [60–65]y.; [66–74]y.). In sex-stratified analyses, the association was mostly observed among men but not in women. Additional analysis conducted using specific causes of death showed that SII was most related to mortality from the heterogeneous group of “other” causes of death, including in largest proportions, respiratory infections including Covid-19, unintentional injuries and digestive disease.

In the literature, the relationship pattern describing the link between social integration and mortality risk is not consistent. Tanskanen and Anttila (2016) [33], who used a SII score that is similar to that of Berkman and Syme (1979) [25] ranging between [0:100], and Pantell et al. (2013) [34] and Lee et al. (2023) [35], who both used the four category SII from Berkman and Syme (1979) [25], reported a graded association where the mortality risk increased as level of social integration decreased. However, using similar measures of social integration, Heffner et al. (2011) [36] found a particularly elevated mortality risk among the least socially integrated (at least twice higher) compared to the three other more socially integrated groups. Furthermore, Brummett et al. (2001) [37]

observed an increased risk of mortality only for individuals who had three or fewer people in their social support network. The literature does not support a unique pattern of association however, along with our study, a relationship is consistently highlighted, suggesting that poor social integration is linked with poor health.

Regarding the potential pathways underlying the association between social integration and mortality risk, previous works from Hakulinen et al. (2018) [38], Eng et al. (2002) [39] and Gronewold et al. (2020) [40] conducted in large populations from the United Kingdom ($N = 479\ 054$), the United States ($N = 28\ 369$) and Germany ($N = 4\ 139$), respectively, showed changes in the association after adjusting for health behaviors and biological factors. Consistent with our findings, they each observed that accounting for health behavior factors, namely physical activity, alcohol consumption and smoking, partly reduced the association. However, in contrast with our results, adjusting for biological factors (diastolic and systolic blood pressure being the most commonly used) lowered the risk of mortality. We did not observe such attenuation in our study. However, our use of a composite measure of biological health may have masked

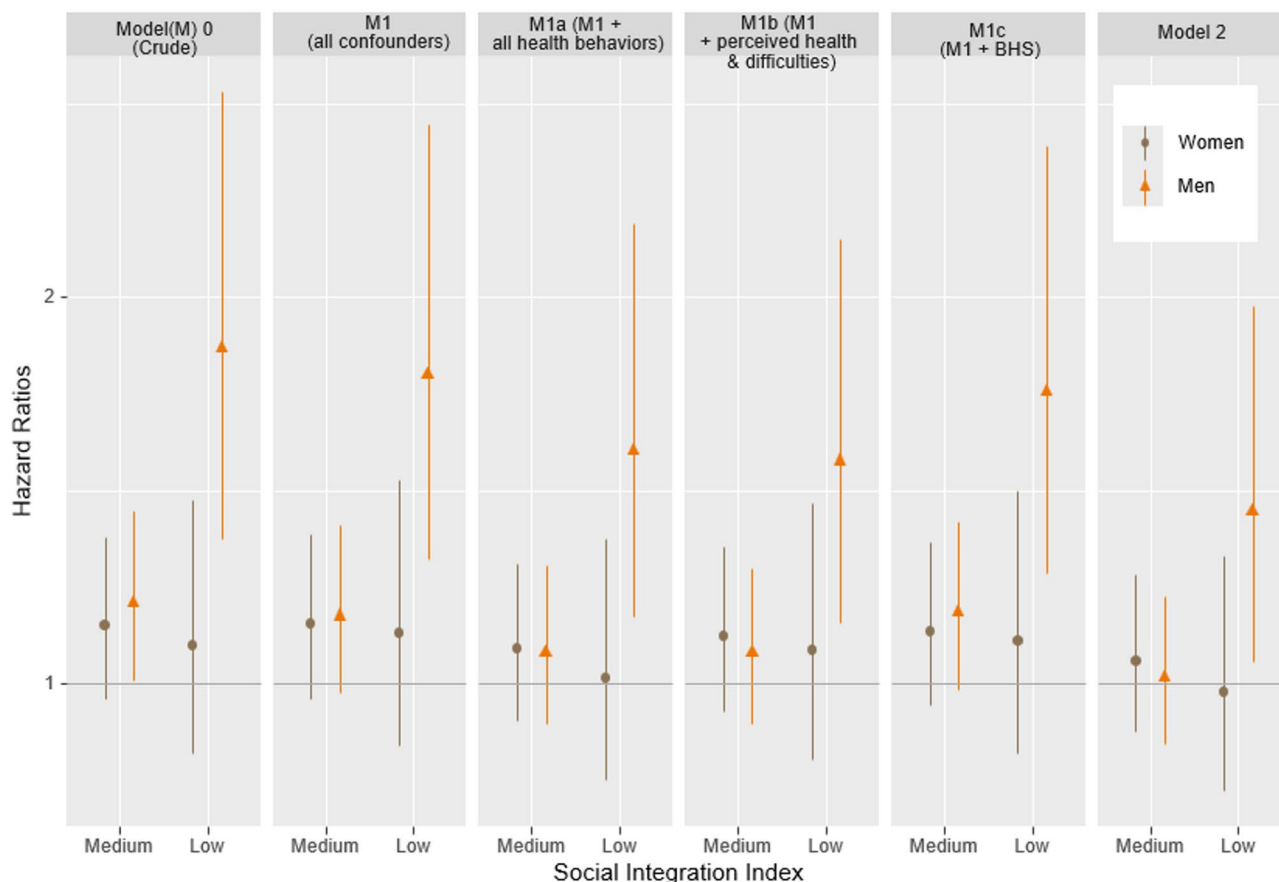


Fig. 4 Hazard ratios for all-cause mortality according to SII groups in total study population and by age groups. The Tromsø Study 1994–2016

the effects of individual biomarkers on the link between social integration and mortality risk. Additionally, we found that adjusting for perceived health and ability to cope with difficulties attenuated the association between SII and mortality, which is consistent with the literature that shows that social isolation measures are related to self-rated health and psychological outcomes [41–43], which are, in turn, related to mortality.

We examined the relationship between social integration and mortality across different life stages and found that it was in the age group of people between 53 and 59 years that the association remained robust and graded, suggesting that social integration is an important determinant of health in mid-life, before entering old age. A meta-analysis by Holt-Lunstad et al. (2015) [44] showed that social integration was more strongly predictive of mortality in studies where the average age of participants was under 65. Additionally, a study on a nationally representative sample of Spanish adults examined the age-specific relationship of loneliness on mortality [45], finding a relationship only among the younger and middle-aged participants, and not the older ones ($\geq 60y$). This absence of association in older adults may be related to various factors, such as, the overriding influence of other

factors on mortality, for example comorbidities; a survivor bias among the elderly driven by other health-favourable characteristics, for example a high socioeconomic status; or the social integration measure not capturing elderly-specific configurations.

A vast literature analysing differences in the association between social relations and health by sex has been conducted in western countries and findings generally show a relationship in both sexes [6, 46, 47], and, often, with higher magnitude in men, as seen in our results. For example, Rico-Uribe et al. (2018)'s [47] meta-analysis showed a higher strength of association between loneliness and mortality for men. The weak association between SII and mortality among the women observed in our study is less expected from the literature. One possible explanation is that being married or living with a spouse, which is a component of our SII index with a cleaving contribution of 0 or 2 (no score in between possible), has been observed as health-beneficial for men while not as much for women [48, 49]. Another hypothesis may relate to the particular context of Norway, being among the countries with the smallest gender gap in labour market and employment participation among OECD countries [50]. An increased involvement

of women in the labour market could palliate the health detriments experienced from a low social integration, as measured by our SII, which captures social components in the personal life rather than in the professional life. Furthermore, we found a pronounced association for men in the [53–59] age group, which may be linked to the relative influence of partnership status over the life-course. Böger & Huxhold (2020) [51] record that partnership status is more predictive of loneliness in middle ages than in advanced ages. Taking this into account, as well as, the general observation that men have health benefits from spousal liaison, can help explain our findings by age groups. With our results, we contribute to illustrating sex-specific differences in the association of social relations and mortality in the context of Northern Norway.

In our cause-specific mortality analyses, social integration tended to be associated with the four studies causes of death, with a stronger association being observed for the heterogeneous group “other”, which is consistent with other research. An extensive literature exists on the relationship between social isolation or similar measures of social relations and both cardiovascular and cancer mortality. A recent systematic review and meta-analysis of 90 cohort studies including 2,205,199 individuals found that social isolation increased both the risk of cancer mortality and CVD mortality [52]. Furthermore, studies from both human population and animals suggest that social isolation may be a risk factor for the development of neuropsychiatric conditions [53]. Studies suggesting a relationship may exist between social relationships and respiratory pathologies [54–56] are in line with the association we observe between SII and “other” causes of death, many of which are respiratory illnesses.

Our results need to be interpreted with caution given our study’s limitations. Firstly, attrition bias related to the longitudinal design of the study may have led to underestimate the link between SII and mortality risk. Secondly, the limited sample size, particularly in subgroup analyses, combined with the unequal distribution of SII categories may have led to imprecise estimates or undetected associations. In addition, this made it difficult to carry out analyses using specific causes of death by age groups. Thirdly, our cut-offs for defining the age-stratified samples were driven by the distribution of deaths across ages to allow for sufficient analytical power, rather than by theoretically defined life-course stages. Age-dependent trends based on life-course transitions such as the average age when individuals become parents or average retirement age could not be clearly unpicked since our age-categories merged together these potential transitional stages of life. Additionally, all variables used in our study, besides date of death, are declared by participants and over year-long survey periods leading to possible measurement errors. Lastly, the models adjusted by

intermediate variables were limited to variable availability. Analysis of other intermediate pathways may deserve attention with other data.

However, our study has a number of strengths, such as the quality and diversity of the data collected in the Tromsø study, which allowed the exploration of behavioral, psychological and biological pathways in the models, and an important sample size which enabled analysis of relationships between SII and mortality by age groups.

Conclusion

Our study explored the association between social integration and mortality risk across different age periods and investigated three potential pathways that may underlie the relationship. Our findings support a growing literature suggesting that social integration is an important determinant of health. The less socially integrated people are, the shorter their life spans. We also showed an important contribution to this association from behaviors and psychosocial-factors, namely perceived health and ability to cope with difficulties. In addition, we reported that this relationship between social integration and mortality risk was especially observed in the middle-age range of our sample, suggesting that interventions to improve social participation, which increases social relations and networks, could have a positive impact on health from mid-adulthood onwards.

Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12889-025-25443-9>.

Supplementary Material 1.

Authors’ contributions

AUP, LN and MKI conceived the study. AUP conducted the data curation, statistical analyses. AUP drafted the original manuscript with considerable additions suggested by LN and MKI. RC, CD, MKI and LN provided insights into the results interpretation and critically revised the manuscript. TW, DV, DT, SG, MCH, NER, EHF, TMS critically revised the manuscript. LN, MKI supervised the work.

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Data availability

Data from the Tromsø Study can be made available to researchers, upon request, after having obtained approval from the Data and Publication Committee (DPC) of the Tromsø Study. Further information can be found at <https://uit.no/research/tromsostudy>.

Declarations

Ethics approval and consent to participate

Ethics and study protocol approval to collect and analyze the present observational data was provided by the Regional Committee of Medical, Health Research Ethics of Northern Norway and Data Inspectorate of Norway.

Participants provided informed consent in forms and data were securely stored in an IT system approved by the Norwegian Data Protection Authority. The present research is conducted with accordance to the guidelines of the Declaration of Helsinki.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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