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Ageing and mental health: changes in self-reported health due to physical illness and mental health status with consecutive cross-sectional analyses

Geir Fagerjord Lorem,[1] Henrik Schirmer,[2,3] Catharina E A Wang,[4,5] Nina Emaus[1]

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For numbered affiliations see end of article.

Correspondence to
Professor Geir Fagerjord Lorem; geir.lorem@uit.no

ABSTRACT

Objectives: It is known that self-reported health (SRH) declines with increasing age and that comorbidity increases with age. We wished to examine how age transfers its effect to SRH through comorbid disease and mental illness and whether these processes remained stable from 1994 until 2008. The hypothesis is that ageing and/or the increased age-related burden of pathology explains the declining SRH.

Setting: The Tromsø Study (TS) is a cohort study using a survey approach with repeated physical examinations. It was conducted in the municipality of Tromsø, Norway, from 1974 to 2008.

Participants: A total of 21 199 women and 19 229 men participated.

Primary and secondary outcome measures: SRH is the outcome of interest. We calculated and compared the effect sizes of age, comorbidity and mental health symptoms using multimediator analysis based on OLS regression.

Results: Ageing had a negative impact on SRH, but the total effect of age decreased from 1994 to 2007. We assessed the direct effect of age and then the proportion of indirect age-related effects through physical illness and mental health symptoms on the total effect. The direct effect of age represented 79.3% of the total effect in 1994 and decreased to 58.8% in 2007. Physical illness emerged as an increasingly important factor and increased its influence from 15.7% to 41.2% of the total effect. Age alone had a protective effect on mental health symptoms and this increased (2.5% to 17.3%), but we found a stronger association between mental health symptoms and physical disease in the later waves of the study (increasing from 3.7% to 14.8%).

Conclusions: The results suggest that the effect on SRH of mental health symptoms caused by physical illness is an increasing public health problem. Treatment and care for specific medical conditions must therefore focus more strongly on how these conditions affect the patient's mental health and address these concerns accordingly.

INTRODUCTION

Self-reported health (SRH) is a subjective assessment of current health status as seen by

Strengths and limitations of this study

- The sample comprises large, representative samples of a general population with repeated measures at ~7-year intervals.
- Multimediator analysis allows for the interpretation of the joint effect of age, comorbid disease and mental health on self-reported health.
- We used the repeated measures as separate cross-sectional data in the analysis.
- The first three panels (1974–1986) did not include any adequate measurement of mental health symptoms and were excluded, but the CONOR-MHI (1994) was validated against the Hopkins Symptom Checklist and showed good agreement.

the patient or participant. It is well known that a whole range of biological, psychological and socioeconomic factors affect SRH, and also that these factors interact.[1–5] The research literature suggests that SRH is produced in a cognitive process that is inherently subjective and contextual, and also that SRH predicts mortality and other health outcomes; this shows that the basis of self-rated health lies in the biological and physiological state of the individual organism.[6] Well-known crucial biological factors that independently affect SRH are specific medical conditions (eg, cardiovascular diseases, diabetes and asthma) and health risk factors (eg, resting heart rate, blood pressure, cholesterol, BMI and endocrine measures). Although the effect of SRH attenuates when such variables are controlled for, SRH still remains as an independent variable for all-cause death and other future health outcomes.[7–11] Mental health symptoms affect SRH, but mental health is also affected by physical disease. The literature suggests that severity of mental health symptoms correlates with many specific medical conditions, and consequently with impaired well-being. Comorbid strain increases with increasing age, and older

people are particularly at risk of experiencing anxiety and depression.[12–17]

To summarise, it is well documented that SRH declines with increasing age but whether it is ageing alone or the increased age-related burden of pathology that explains this association is still unanswered. The prevalence of coexisting chronic conditions is rising as life expectancy increases in contemporary Western society.[18] The age-specific decline could mean that the increasing level of pathology due to age explains this specific decline of SRH and not ageing by itself.

There are, to the best of our knowledge, no studies that describe the combined effect of ageing, comorbid physical disease and mental health symptoms on general perceived health status. Moreover, since medical treatment has improved over the past three decades, leading to increased life expectancy, it seems timely to ask whether people's experiences of ageing, comorbid disease and mental health problems remain the same. We wished to examine how age transfers its effect on SRH through comorbid physical disease and mental health symptoms. A further aim was to explore how mental health symptoms are affected by physical disease and whether these processes remained stable from 1986 until 2008.

METHOD
Sample and design
The Tromsø Study (TS) was a cohort study which provided data allowing us to estimate the impact of a broad range of factors on a general population, using surveys and physical examinations in a large representative sample.[19] TS consisted of six surveys conducted in Tromsø in Northern Norway from 1974 to 2008. We used consecutive cross-sectional analyses within the TS. The study population was recruited from all inhabitants in specific age groups. The aim was to include large, representative samples of the Tromsø population, with the invitation of whole birth cohorts and random samples. The attendance rate was high (66–75%). A total of 21 199 women and 19 229 men gave informed signed consent and attended up to 6 separate health examinations. Tromsø 1 was a heart study conducted in 1974 and included only men aged 20–49. Tromsø 2 followed-up the first study in 1979–1980 but included men (aged 20–54) and women (aged 20–49). Tromsø 3 was executed in 1986–1987 and included men and women in the 20–56 age range, and a 10% random selection of persons aged 12–19. We excluded Tromsø 1–3. SRH was introduced during the 1980s; Tromsø 1 and 2 thus lack SRH and Tromsø 3 did not include any adequate measurement of mental health symptoms. Our sample starts with Tromsø 4 in 1994. Tromsø 4 is the largest wave, and participants were followed-up in 2001 and 2007/2008. We excluded those with missing data (n=736 in TS4, n=1132 in TS5 and n=767 in TS6). The final analysis therefore comprised 12 408 men and

13 579 women from TS4, 3108 men and 3746 women from TS5 and 5769 men and 6338 women from TS6. The Norwegian Data Protection Authority and the Regional Committees for Medical and Health Research Ethics North Norway approved the TS.

Measurements
The participants completed a self-administrated questionnaire with questions on a broad range of diseases and symptoms, health behaviour, social conditions, education, financial situation and level of physical activity. *Self-reported health (SRH)*: The independent variable SRH was reported by answering the question, 'What is your current state of health?' with answers ranging from very bad (0) to very good (4) in Tromsø 6, and from poor (1) to very good (4) in Tromsø 4 and 5. *Specific medical conditions*: We selected 13 symptomatic medical conditions reported in all panels. These were psoriasis, food allergies, chronic bronchitis, migraine, ulcer, asthma, thyroid disease, arthritis, myocardial infarction, cerebrovascular stroke, diabetes, osteoporosis and angina. The conditions were self-reported by answering questions such as 'Do you have or have you had….?' We used the Health Impact Index (HII) to measure the comorbid conditions. Diseases have a varied impact on SRH. HII classifies patients with comorbid disease according to the impact that each condition has on SRH by assigning a weight for each condition. HII equals the total score of each condition of the participant. HII thus considers the severity and joint effects of the conditions.[4] The range was 0–18 in TS4, 0–17 in TS5 and 0–22 in TS 6. Online supplementary appendix 1 shows the conditions included with their weights and prevalence in the different waves.

Mental health symptoms were based on a well-validated self-report symptom inventory comprising questions representative of the symptom configurations of anxiety and depression commonly observed among outpatients. It includes questions such as 'Have you experienced sudden fear without apparent reason', '…felt tense or upset', '…easily blamed yourself', '…felt depressed or sad', '…felt useless or worthless', '…felt that everything is a struggle' or '…felt hopelessness'. Each answer is scored from 1 to 4. The measurement is the average score. The range was therefore 1–4 in all waves. The mental health index (CONOR-MHI) used at T4 has been compared with the Hopkins Symptom Checklist (HSCL) with reasonably good agreement. In the following surveys T5–6, the HSCL was used. A cut-off level of 2.15 for significant symptoms is equivalent to the 1.85 level in HSCL-10.[20 21]

Analysis
The purpose of the descriptive statistics was to define the distribution of SRH, comorbid disease and mental health across samples, age groups and gender. We used cross tabulation and two-way ANOVA to describe the characteristics of the sample. *Multimediator analysis* was

used for the analysis of the conditional nature of the mechanism by which age transmits its effect on SRH. The advantage of this method is that it allows for the interpretation of multiple confounders that may function as either mediators or moderators and interprets their joint effect on the statistical model derived from the theoretical model.[22] [23] The analytical goal of the multimediation analysis was to determine how age transfers its effect to SRH directly and through physical disease and mental illness. The first step was the conceptual model, which we based on the idea that age represents the timeline of life in which events like disease occur and physical condition changes. Previous analysis, tracking individual subjects, confirms that SRH decreases with increasing age and whenever levels of pathology increase. This implies that age might influence SRH either directly or indirectly through pathology as life events. The second step was to translate the conceptual model into a statistical model. Figure 1 shows the conceptual model and its translation into a statistical model.[24] The statistical model includes SRH as the outcome (Y), age as the main variable (X) with medical condition (M_1) and mental health symptoms (M_2) as mediators. Our statistical model includes three indirect effect lines (Ind 1–3).

▶ Ind 1: Age→HII→SRH ($a_1 \times b_1$)
▶ Ind 2: Age→HII→HSCL→SRH ($a_1 \times d_{21} \times b_2$)
▶ Ind 3: Age→HSCL→SRH ($a_2 \times b_2$)

We used multiple regression to assess the two mediators (M_1=medical conditions and M_2=mental health) and the reaction (Y=SRH). The regression coefficients, 95% CIs and model summary information for the mediated effect of age on SRH have been published as online supplementary appendix 2.

RESULTS

Characteristics and total effect of age

Table 1 shows the characteristics of the four samples indicating increasing comorbidity with a shift in 2001 (T5) when the comorbid levels decreased with a corresponding increase in SRH. Figure 2 shows profile plots for SRH, comorbidity and ratio of persons with subthreshold and significant mental health symptoms across age and gender. Testing for gender and age differences with two-way ANOVA showed that all mean differences were significant ($p < 0.0001$) for SRH. Here, SRH declined significantly with increasing age with a corresponding increase in comorbidity at all three survey points. Although the gender differences were statistically significant for all three factors, the gender difference in SRH was less than a 10-year age difference in SRH in all waves. For comorbidity, the gender difference was as large as a 10-year age difference for the two intermediate survey points, but less so at the first and last survey points where the gender difference was small. For comorbidity, the most striking finding was the increase
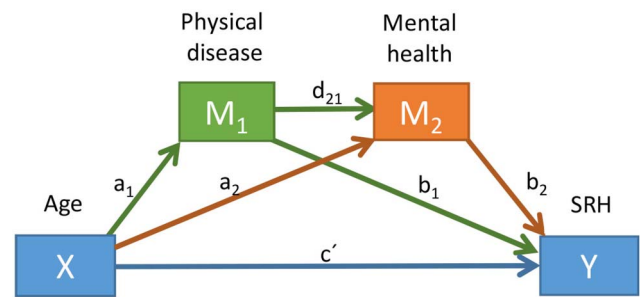
Figure 1 Conceptual and statistical diagram for the mediated effect of age on SRH through comorbid disease and mental health symptoms.

Table 1 Distribution of SRH, physical condition and demographics, specific medical conditions, mental health symptoms and social context by gender in Tromsø 4–6 (1994–2008)

	Tromsø 4	Tromsø 5	Tromsø 6
Self-rated health (mean/SD)	2.82 (0.70)	2.7 (0.67)	2.74 (0.77)
Age (mean/SD)	48.1 (14.8)	60.1 (13.8)	58.7 (12.4)
Health impact index (mean/SD)	0.95 (1.66)	1.72 (2.18)	1.66 (2.21)
Mental health symptoms (mean/SD)	1.25 (0.36)	1.29 (0.38)	1.52 (0.41)

SRH, self-reported health.

by age across all surveys, especially for women, who had an increasing burden of disease as they got older. For mental health symptoms, the greater burden for women was most striking.

The total effect of age

We used an OLS regression model to determine the total effect of age on SRH. Table 2 shows the linear model of the total effect of age on SRH. We see that age had a negative effect on SRH in all samples, and also that the total effect of age attenuated from 1994 to 2008 in parallel with increasing life expectancy in the region. In our model, each year of age represented −0.0175 (CI −0.018 to −0.017) deterioration of SRH in T4 but the effect dropped to −0.013 (CI −0.014 to −0.012) in T6.

The indirect effect of pathology

The M_1 models in online supplementary appendix 2 show that higher comorbidity was associated with increasing age in all waves (Coeff=0.050 in T4; 0.059 in T5; 0.050 in T6). The M_2 models show a significant effect for age on mental health symptoms (Coeff= −0.0002 in T4; −0.0025 in T5; −0.0029 in T6), although
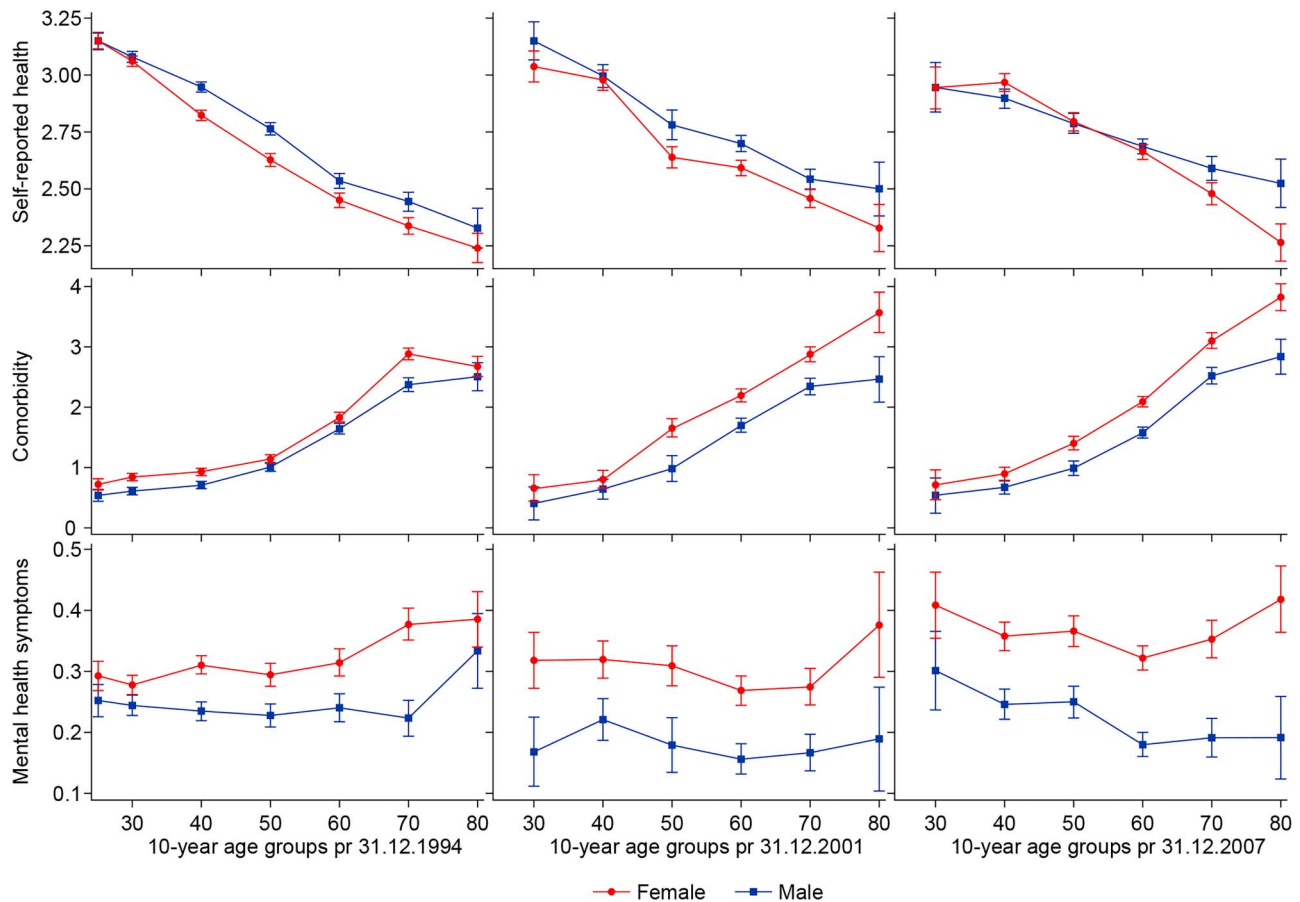
Figure 2 Profile plots for self-reported health for interaction effects between age and gender with 95% CIs. *Self-reported health*: Range from very poor (0) to very good (4) in TS 6, and poor (1) to very good (4) in TS 4 and 5. *Comorbid disease*: Number of diseases grouped into a score with range 0–17 (mean 0.97) in TS 4, range 0–17 (mean 1.59) in TS 5 and range 0–19 (mean 1.53) TS 6. *Mental health symptoms*: CONOR-MHI with range 1–4 (mean 1.52) in TS 4, and HSCL-10 with range 1–4 in TS 5 and 6 (mean 1.25 in TS 5 and mean 1.29 in TS 6). All differences p<0.001. Red lines=women, blue lines=men, CI 95% is SE*1.96.

medical conditions when they occurred affected mental health symptoms more than age (0.030 in T4; 0.032 in T5; 0.041 in T6). All effect lines in the statistical model were estimated by a series of OLS regression models (see online supplementary appendix 2). Table 3 shows the indirect and direct effects of age on SHR. We calculated these from the coefficients in online supplementary appendix 2 according to our statistical model. Adding gender as a moderator on each effect line did not change the overall results.

We found that age had a direct and indirect effect on SRH. The direct effect (c') of age attenuated from 1994 to 2008 (T4: c'=−0.013, T5: c'=−0.011, T6: c'=−0.008). This suggests that age affected SRH independently of pathology even when controlling for the mediators, and also that age itself had a lower impact on SRH at the latest measuring point.

We found that age had an increasing negative indirect effect through comorbid diseases (T4: −0.0034; T5: −0.0035; T6: −0.0042). Since the total effect attenuated in the same period, this implied that the ratio of total to indirect effect of comorbid disease increased

correspondingly more. It was 0.192 in 1994, 0.236 in 2001 and 0.330 in 2007/2008. This trend implied that physical disease was an increasingly important factor relative to age itself to explain why SRH declines with increasing age.

The second indirect effect (Age→HII→HSCL→SRH) included mental health symptoms associated with having a disease. We found a negative effect on SRH T4 of −0.0006, T5 of −0.0013 and T6 of −0.0019. This suggests that having a physical disease was associated with higher levels of mental health symptoms, which in turn affected SRH. The ratio of total to indirect effect of comorbid disease was −0.037 in T4, −0.086 in T5 and −0.148 in T6. Thus, we see a consistent increase in the relative size of the second indirect effect from 1994 to 2007/2008. This implied that the relative significance of mental health issues connected to physical disease increased during this period and at 14.8% of the total effect, it is also clinically significant.

The third indirect effect line (Age→HSCL→SRH) revealed that SRH increased with increasing age, which implies that mental health symptoms are associated with

Table 2 Linear model of the total effect of age on SRH with 95% CIs

	Tromsø 4 Coeff. (95% CI)	Tromsø 5 Coeff. (95% CI)	Tromsø 6 Coeff. (95% CI)
Age	−0.0175 (−0.0181 to −0.0170)	−0.0146 (−0.0157 to −0.0136)	−0.0128 (−0.0139 to −0.0117)
Constant	3.6584 (3.6311 to 3.6856)	3.5742 (3.5111 to 3.6372)	3.4840 (3.4179 to 3.5500)
Note	$F_{(1, 25\ 195)}=4039.67$. p<0.0001. $R^2=0.1382$	$F_{(1, 7764)}=783.57$. p<0.0001. $R^2=0.0917$	$F_{(1, 11\ 962)}=519.36$. p<0.0001. $R^2=0.0416$

CIs and SEs are based on 1000 bootstrap samples.
SRH, self-reported health.

increasing age when controlled for physical disease. The ratio of effect size increased during this period from 0.002 in T4 to −0.0109 in T5 and −0.200 in T6. This implied that when we disregard physical illness and mental health problems associated with physical illness, increasing age had a beneficial effect on SRH.

DISCUSSION

Ageing affected SRH directly and also indirectly through increased levels of pathology. We observed a change in how ageing and physical disease influenced SRH between the different waves. The direct effect of ageing (c′) represented 79.3% of the total effect in 1994, 69.8% in 2001 and 58.8% in 2007/2008. This means that ageing is still the most important factor for SRH, but that ageing itself became relatively less important between the waves. Meanwhile, physical disease became an increasingly important factor for SRH. As shown in table 3, comorbid conditions (HII) represented 15.7% of the total effect in 1994, 26.7% in 2001 and 41.2% in the last wave in 2007/2008. Furthermore, ageing itself had a protective effect on mental health symptoms which increased (2.0–20.0% of the total effect). We found a stronger association between mental health symptoms and physical disease in the later waves (increasing from 3.7% to 14.8%). Mental health symptoms related to physical disease consequently led to lower SRH levels in the later parts of the study.

Physical disease is known to be related to mental health symptoms of anxiety and depression, which the HSCL-10 scale is especially sensitive to measure in a general population.[15] Epidemiological data suggest that severity of mental health symptoms correlates with disease, for example, one-third of stroke survivors develop depression[12] and one-quarter anxiety disorders.[13] Cardiovascular diseases have shown discrete effects for panic disorder and specific phobia.[14 15] Older people with illnesses such as coronary heart disease, arthritis and chronic lung disease show increased levels of depressed mood and impaired well-being.[16] Cumulative stress exposure across different stress domains contributes to depressive symptoms in cancer survivors.[17] Moreover, pessimism, negative cancer-related rumination and physical symptom distress predicted anxiety and depression trajectories.

However, our findings indicate that physical disease in recent decades has become more strongly associated with mental health symptoms, that is, the indirect effect on mental health symptoms via physical disease has increased over time. Accordingly, it seems plausible that physical diseases in terms of SRH affect us more than before, and also that physical disease has a greater impact on our reaction towards illness than before. So how can we explain these findings? Why does physical disease trigger symptoms of anxiety and depression more often than before?

Table 3 Direct and indirect effect size with 95% bias corrected CIs in parentheses, SEs and ratio of indirect to direct effect of age on self-reported health

	Tromsø 4 Effect (95% CI)	Tromsø 5 Effect (95% CI)	Tromsø 6 Effect (95% CI)
Total effect of age on SRH	−0.0175 (−0.0181 to −0.0170)	−0.0146 (−0.0157 to −0.0136)	−0.0128 (−0.0139 to −0.0117)
Indirect effect of age on SRH			
Total	−0.0034 (−0.0032 to −0.0037)	−0.0039 (−0.0034 to −0.0043)	−0.0046 (−0.0040 to −0.0052)
Age→HII→SRH	−0.0027 (−0.0028 to −0.0027)	−0.0043 (−0.0044 to −0.0041)	−0.0053 (−0.0054 to −0.0051)
Age→HII→HSCL→SRH	−0.0006 (−0.0006 to −0.0007)	−0.0013 (−0.0011 to −0.0014)	−0.0019 (−0.0017 to −0.0021)
Age→HSCL→SRH	0.000* (0.0002 to −0.0002)	0.0016 (0.0021 to 0.0011)	0.0026 (0.0032 to 0.0020)
Ratio of indirect to total effect of age on SRH			
Total	0.195 (0.176 to 0.215)	0.267 (0.217 to 0.318)	0.360 (0.284 to 0.442)
Age→HII→SRH	0.157 (0.154 to 0.158)	0.290 (0.281 to 0.299)	0.412 (0.391 to 0.433)
Age→HII→HSCL→SRH	0.037 (0.031 to 0.043)	0.086 (0.071 to 0.102)	0.148 (0.122 to 0.179)
Age→HSCL→SRH	0.002* (−0.010 to 0.013)	−0.109 (−0.135 to −0.083)	−0.200 (−0.229 to −0.170)

CIs and SEs are based on 1000 bootstrap samples.
Indirect effect of X on Y through Mi only=ai×bi, indirect effect of X on Y through M1 and M2 in serial=a1× d21×b2, direct effect of X on Y=c′.
The ratio of indirect effect to direct effect=Mi/c′(figure 1, statistical diagram).
*CIs include zero.
HII, Health Impact Index; HSCL, Hopkins Symptom Checklist; SRH, self-reported health.

One possible explanation may be found in social changes in Norway and the Norwegian healthcare system. Although we today have curative and palliative treatment of many more physical disorders, and more individuals have access to treatment, there is also an increased expectation of 'active ageing' and healthy living.[25][26] This expectation is realistic, as the incidence of especially cardiovascular diseases has been rapidly declining for several decades, but is contrasted by a decreasing case fatality, leaving more of those who still get cancer, coronary heart attacks and stroke with lasting disability as more people survive.[27]

SRH can reflect the states of the human body and mind. People base their health assessments on different types of information and contextual frameworks.[6] It is plausible that people who expect to age actively become unhappy or worried when encountering limitations and disease. It may also be argued that people tend to respond negatively to questions on their health or limitations when comparing their situation with others at similar age. Bodily sensations that are directly available to the individual are another source of information.[6] Accordingly, it seems plausible that people compare current body status with the situation before the disease occurred, and experience fear of relapse or having another disease. We cannot answer this assumption based on three cross-sectional analyses; however, it is a hypothesis that could be answered by tracking individuals in the TS cohort.

Furthermore, current healthcare services are organised to place greater emphasis on efficiency than on care, and society has a faster pace of life so that older people more often live alone and isolated than a few decades ago. From an evolutionary perspective, symptoms of anxiety and depression are understood as normal reactions to life-threatening and uncontrollable situations. For example, fear is an obvious adaptive function as it stimulates the 'fight-or-flight' response when the individual is exposed to a threat or dangerous situation; unless the individual can escape, it will hide or 'freeze' the situation.[28] Furthermore, Gilbert describes anxiety disorders as a maladaptive expression or phenotype of the original functional fear system where the acute stress response is triggered in an inappropriate manner.[29] Similarly, Nettle proposes that depression may represent a maladaptive expression of an original functional control system for positive affect, that is, a functional downregulation of positive affect in certain situations and contexts.[30] Gilbert describes such a downregulation of positive affect as a defensive reaction, a similar fight-or-flight response, in situations where the individual experiences loss of control over aversive events or over significant resources including the social environment.[29] An increased incidence of comorbid physical disorders with consequent reduced access to social participation can thus be a plausible explanation of an increase in mental symptoms related to physical disorders.

Interestingly, we found that age by itself was protective of mental health symptoms when controlled for the mental health symptoms associated with physical illness. Several studies focus on how physical disease is associated with increased risk of mental health symptoms. In our study, this mechanism represented 5% of the total effect in 1994, 6% in 2001 and 12% in 2008. Our findings concur with studies on patient populations showing that mental health is an important aspect of impairment of SRH when physical illness occurs.

Strengths and limitations of the method
HII includes 13 symptomatic medical conditions, but does not include risk factors such as hypertension and dyslipidemia. These could be included as mediators on the age->HII->SRH effect line, but this did not change the overall findings of the model. The TS includes

cancer but it is self-reported and does not distinguish between those with an active illness and those who have had cancer. That was the most likely explanation for why it did not add to the model[4] and was therefore not included.

Although measured on an ordinal scale, the underlying phenomenon of SRH is continuous, and the scales represent similar logical increments. Furthermore, the distribution of SRH, apart from being staggered, resembled the shape of a normal distribution. Hence, an OLS regression model could be used for the analysis of independent associations in the multivariable model.[22] Adding gender as a moderator on each effect line did not change the overall results. Mental health symptoms were measured with different instruments, which may have affected our findings. T5 and T6 used the Hopkins Symptom Checklist (HSCL-10), which is a self-reported symptom inventory comprising 10 items representative of the symptom configurations of anxiety and depression commonly observed among outpatients.[20] T4 used the CONOR Mental Health Index (MHI). This was based on seven questions concerning different symptom configurations of anxiety and depression. It was partly derived from HSCL-10 and the General Health Questionnaire (GHQ). Fortunately, Tromsø 4 is included in the CONOR database that also included HSCL-10. The index has therefore been compared with HSCL-10 with reasonably good agreement. It has been concluded that the scales can be used in epidemiological studies. For comparisons, it is recommended to use the cut-off level of 2.15 for significant symptoms as equivalent to the 1.85 level in HSCL-10.[21 31]

CONCLUSIONS

As medicine advances and life expectancy increases, we have higher expectations for the healthcare system and to remain healthy even in old age. The results suggest that the effect on SRH of mental health symptoms caused by physical illness is an increasing public health problem. It seems that our resilience to diseases is decreasing. Therefore, treatment and care for specific medical conditions must focus more strongly on how these conditions affect the patient's mental health and address these concerns accordingly.

Author affiliations
[1]Department of Health and Care Sciences, Faculty of Health Sciences, The Arctic University of Norway, Tromsø, Norway
[2]Department of Clinical Medicine, Faculty of Health Sciences, The Arctic University of Norway, Tromsø, Norway
[3]Division of Cardiothoracic and Respiratory Medicine, University Hospital of Northern Norway, Tromsø, Norway
[4]Department of Psychology, Faculty of Health Sciences, The Arctic University of Norway, Tromsø, Norway
[5]Division of Child and Adolescent Health, University Hospital of Northern Norway, Tromsø, Norway

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