Secular trends in prevalent mild cognitive impairment: Data from the Swedish population-based study Good Aging in Skåne

Marieclaire Overton1 | Mats Pihlgård2 | Sölve Elmståhl1

1 Division of Geriatric Medicine, Department of Clinical Sciences in Malmö, Lund University, Skåne University Hospital, Malmö, Sweden
2 Perinatal and Cardiovascular Epidemiology, Lund University, Skåne University Hospital, Malmö, Sweden

Correspondence
Marieclaire Overton, Jan Waldenströms gata 35, CRC, Building 28, fl.13, Skåne University Hospital, SE-205 02, Malmö, Sweden. Email: Marie_Claire.Overton@med.lu.se

Funding information
Swedish Ministry of Health and Social Affairs; county Region Skåne; Medical Faculty at Lund University; Swedish Research Council, Grant/Award Numbers: 2017-01613, 2017-00639, 2021-01437

Abstract
Background: Research suggests that incident dementia is decreasing, yet research on secular trends of prodromal dementia such as mild cognitive impairment (MCI) is lacking.

Methods: To determine change of MCI prevalence over time and potential explanatory factors, four baseline samples (years 2001–2020) of Swedish participants (n = 3910) aged 60 and 81 at examination were compared.

Results: An overall drop of 9 to 10 percentage points in MCI prevalence between 2001 and 2020 was observed, with lower odds ratios (OR) for MCI in the latest birth cohorts compared to earliest (e.g., ORs for 60-year-olds in latest born = 0.53; 95% confidence interval [CI] 0.37–0.76). Adjustments for sociodemographic (e.g., education), lifestyle, vascular and metabolic health and depression could not fully explain the observed MCI decline (e.g., 60-year-olds, OR = 0.59; 95% CI 0.40–0.88).

Discussion: Studies like this are imperative as even a slight postponement in the onset of dementia could have a substantial impact on future public health burden.

KEYWORDS
dementia, mild cognitive impairment, prevalence, risk factors, secular trends

1 INTRODUCTION

As the population of older adults increases, prevalence of age-related cognitive deficiencies, such as Alzheimer’s disease, is also expected to rise, causing a severe burden on society.1 Somewhat contradictory to this expectation, age-specific prevalence2–4 and incidence5–8 of dementia have seemingly dropped in several high-income countries. Underlying causes of prevalent dementia decline are suggestive that individuals with dementia have shorter life expectancy or are being cured. As there is no cure for dementia to date and survival after dementia diagnosis is seemingly stable or even increasing,9 an alternative explanation is that onset of dementia is decreasing.10 Plausibly, a consecutive decline in prodromal stages of dementia, such as mild cognitive impairment (MCI) should also be observed. Yet, only a handful of studies examining secular trends of MCI currently exist.10–12 In addition, even small changes in future estimates of dementia could have a substantial impact on forthcoming public health, therefore implementing investigations of temporal changes of MCI is imperative.

Twelve modifiable risk factors, accountable for 40% of dementia cases worldwide, have been proposed.13 Factors include sociodemographic status such as low education; lifestyle factors: smoking, physical inactivity, obesity, excessive alcohol consumption and social isolation; vascular and metabolic disease; and other health-related factors: hypertension, diabetes, traumatic brain injury, hearing impairment, depression, and air pollution. In accordance, most of these factors are also associated with MCI and MCI progression14,15 and their
prevalence have changed simultaneously with declining dementia occurrence. For instance, positive trends in lifestyle choices (e.g., improved exercise habits or rise in smoking cessation), better treatment of vascular conditions (e.g., increased use of antihypertensives), and higher educational attainment are some of the sought-out explanations of lower dementia rates. Yet, to what extent these changes have directly impacted the observed decline in prevalence and incidence of MCI and dementia remains unclear.

Using four population-based cohorts from the south of Sweden, this study aimed first to investigate secular trends in prevalent MCI during the last two decades (2001–2020). Second, guided by the leading modifiable causes of dementia and MCI, we investigated whether demographic and lifestyle features, vascular and metabolic health, and depression can explain potential secular trends in prevalent MCI.

2 | MATERIALS AND METHODS

2.1 | Population sample and data collection

The sample was drawn from the Swedish longitudinal aging study Good Aging in Skåne (GÅS-project). Participants from rural and urban areas of the southern part of Sweden ages 60 (born 1941–1956) and 81 (born 1920–1937) at the time for examination were invited at random from the population registry in 2001: cohort 1 (C1), 2007: cohort 2 (C2), 2012: cohort 3 (C3), and 2018: cohort 4 (C4). Each wave took approximately 3.5 years to complete. This type of study design enables the comparison of multiple birth cohorts the same age at examination. Waves one and two had participant rates of 60%; wave three had 70% and no rate is available for wave four as it is not yet complete. A total of 3111 60-year-olds (mean age = 60.12, standard deviation [SD] = 0.76) and 1394 81-year-olds (mean age = 81.3, SD = 0.50) were extracted from the GÅS-sample. There was a total exclusion of 211/239 (60-/81-year-old) participants due to dementia (13/55), not enough cognitive data to classify MCI (17/55), and both impaired functional disabilities and impaired cognitive test scores but no dementia (17/102); 143/51 participants were used to create norm scores (see Overton et al. for norm-score application). The remaining 2900 60-year-olds and 1010 81-year-olds were used for prevalence calculations. The study was approved by the regional ethics committee of Lund University (LU 744-00) and written consent from all participants was obtained and in case of cognitively impaired individuals, consent was obtained from closest relative or guardian.

The participants were invited for a full day with examinations conducted by a nurse, physician, and a psychological test administrator. The cognitive test battery included tests measuring four cognitive domains: episodic memory, speed of processing, verbal ability, and visuospatial skills and global functioning (Mini-Mental State Examination [MMSE]). A shorthand version of the Comprehensive Psychiatric Rating Scale was used to assess depression. Dementia was diagnosed by the examining physician in accordance with Diagnostic and Statistical Manual of Mental Disorders, 4th Edition and medical records. Stroke (transient ischemic attack, cerebral hemorrhage, or cerebral infarct), myocardial infarction, diabetes (type 1 or 2), and medication for hypertension was also determined by the physician asking the participant or by medical records. Smoking (current or never/former smoker) and exercise habits (exercise regularly/sometimes or never), co-habitant status, education (primary school, upper secondary school, or university degree), and alcohol consumption were self-assessed via questionnaires. Alcohol consumption was divided into three categories: alcohol consumption 2 to 7 days a week, 2 to 4 days a month, and never consume alcohol.

2.2 | Implementation of MCI criteria

An algorithmic approach was applied using the expanded original Mayo Clinic criteria to define MCI cases: subjective and/or informant
cognitive complaint, normal functional ability, no dementia, and objective cognitive impairment in one or more cognitive domains relative to normative data. MCI cases were further divided into the subgroups: single-domain and multidomain MCI (MCIs/MCIm) and amnestic and non-amnestic MCI (aMCI/naMCI).

MCIs was defined as having at least one impaired test score in the cognitive domain, whereas MCIm was defined as having impaired test scores in multiple cognitive domains. Impaired test score was established when a participant had a score below the seventh percentile of test scores in a healthy subpopulation, when taking age, sex, and educational factors into account.1

Subjective and informant cognitive complaint was either confirmed through a complaint from the participant or by a concern from the examining physician. The Katz Index of Independence in Activities of Daily Living (ADL)-index was used to evaluate functional abilities. Participants with impaired personal ADL were excluded from the MCI sample; mild problems of instrumental ADL were permissible.15 Participants were excluded from the entire sample if they had dementia and if they had both impaired cognitive test results and impaired ADL and no dementia. Some participants had insufficient cognitive data to be classified as MCI or non-cognitively impaired (NCI); these participants were classified as healthy if they had a MMSE over 26,17 or else they were excluded.

2.3 Statistical analyses

Cohorts were stratified into two age groups: 60 and 81 for cohort comparison. Chi-square and t-tests were used to explore cohort variations in demographic, lifestyle, and health and cardio-cerebrovascular factors and depression. Proportions with 95% confidence intervals (CI) were calculated for each cohort and \( \chi^2 \) tests were run to detect cohort differences for MCI and linearity was tested using Chi-2 linear-association test. In addition, a series of logistic regression was performed, with MCI prevalence as outcome variable and birth cohort as predictor variable. We used five logistic models to estimate odds ratios (ORs) and 95% CIs of MCI in the different cohorts, controlling for demographics (sex and education); lifestyle, vascular, and metabolic factors; and depression. To further inspect whether differences in MCI prevalence were the same for all types of educational attainment levels, logistic regression analyses stratified by the three educational groups (primary, secondary, and university) were performed. All analyses were performed using IBM-SPSS statistics package 25.

3 RESULTS

3.1 Descriptive statistics

Differences in the characteristics between the various cohorts were detected (Table 1). A successive increase in higher education (secondary/university) with birth years was observed. For the 60-year-olds, body mass index (BMI) increased with birth years and there was a significantly smaller proportion of smokers in the later birth cohorts compared to the earlier birth cohorts (e.g., in C4, 20.1% smoked vs. 28% in C1). The later born 81-year-olds reported exercising more and consuming more alcohol than those born earlier. Proportions of cardio- and cerebrovascular conditions such as stroke, myocardial infarction, and diabetes were similar in all the cohorts. There were, however, significantly more members with diabetes in the later birth cohorts for 81-year-olds (C1: 5.8% vs. C4: 12.6%). Observed in both age groups, systolic blood pressure significantly decreased and uses of antihypertensives increased with birth year. Last, the number of participants with depression significantly decreased with birth year.

3.2 MCI prevalence

For the 60-year-olds, 482/2900 MCI cases were identified, leading to an overall MCI prevalence of 16.5% (95% CI: 15.8–18.0; Table 2). There were significant birth-cohort differences with higher proportions of MCI in the earlier birth cohorts: C1: 22.1% (95% CI 18.7–25.8), C2: 16.9% (95% CI 14.7–19.3), C3: 14.4% (95% CI: 12.2–16.9), and C4: 13.0% (95% CI 9.72–16.8). The earlier birth cohorts had higher proportions of multi-domain MCI (e.g., C1: 33.7% vs. C4: 18%) than the later borns, although this Chi-2 linear association was on the border of significance (P = .06). Of those with MCI, no significant differences (\( \chi^2 = 5.16, P = .16 \)) in the spread of males or females among the different cohorts were observed.

For the 81-year-olds, 327/1010 MCI cases were identified, leading to an overall MCI prevalence of 21.5% (95% CI: 20.9–26.2; Table 2). Significant differences in MCI occurrence between birth cohorts were observed, where the earliest born cohort had the highest MCI prevalence C1: 29.1% (95% CI: 22.5–36.1) compared to the later born cohorts C2: 18% (95% CI: 13.8–24.0), C3: 22% (95% CI: 17.8–26.6), and C4: 19% (95% CI: 14.8–23.8). This decrease in prevalence was consecutively falling, with the exception for C3 in which the number increased slightly from the previous cohort. No significant cohort differences were observed for single/multi-domain or amnestic/non-amnestic MCI, nor were there significant cohort differences between men and women in MCI prevalence (\( \chi^2 = 1.45, P = .692 \)).

3.3 Logistic regression

The ORs for MCI decreased with birth year for the 60-year-olds when adjusting for birth cohort (crude model), ORs: C2: 0.72, C3: 0.60, C4: 0.53 (Table 3). ORs comparable to the crude model were observed in all models. Noticeably, the differences in ORs for C2 (compared to C1) were attenuated when education and sex were included in the models.

For the 81-year-olds, birth cohort was significantly associated with odds of MCI, and lower ORs of MCI were observed for the latest birth cohort compared to the earliest born (ORs: C2: 0.56, C3: 0.69, C4: 0.58, C1 is ref). As indicated in the previous cohort analyses, the likelihood of MCI was similar for cohorts 1 and 3. Including sex and educational
### TABLE 1
Comparison of descriptive characteristics stratified by age of cohorts examined 2001 and 2020

| Cohort       | 60-year-olds, n | 81-year-olds, n | Test statistic and P-value |
|--------------|-----------------|-----------------|----------------------------|
| **60-year-olds, n** | 553             | 1069            |                            |
| **81-year-olds, n** | 191             | 249             |                            |
| **Age, mean (SD)** |                 |                 |                            |
| 60           | 60.3 (0.40)     | 60.8 (0.69)     | 60.3 (0.45)                | 60.5 (0.38) | \(F = 191\), \(P < .000\) |
| 81           | 81.1 (0.34)     | 81.2 (0.54)     | 81.0 (0.43)                | 81.6 (0.40) | \(F = 127.8\), \(P < .000\) |
| **Female, n (%)** |                 |                 |                            |
| 60           | 273 (49.4)      | 579 (54.2)      | 446 (48.6)                 | 180 (47.7)  | \(\chi^2 = 8.48\), \(P < .05\) |
| 81           | 109 (57.1)      | 141 (56.6)      | 210 (55.3)                 | 192 (57.1)  | \(\chi^2 = 0.315\), \(P = .957\) |
| **Education, n (%)** |                 |                 |                            |
| 60           | 211 (38.9)      | 260 (27.0)      | 207 (24.5)                 | 71 (21.3)   | \(\chi^2 = 53.9\), \(P < .000\) |
| 81           | 181 (33.3)      | 314 (32.6)      | 319 (37.8)                 | 128 (38.3)  |                            |
| 60           | 151 (27.8)      | 389 (40.4)      | 319 (37.8)                 | 135 (40.4)  |                            |
| **BMI, M (SD)** |                 |                 |                            |
| 60           | 26.9 (4.51)     | 26.5 (5.43)     | 27.1 (4.70)                | 27.3 (4.46) | \(F = 3.57\), \(P < .05\) |
| 81           | 26.4 (4.08)     | 26.0 (4.00)     | 26.1 (3.93)                | 26.2 (4.53) | \(F = 0.4\), \(P = .75\)  |
| **Smoker, n (%)** |                 |                 |                            |
| Yes, 60      | 153 (28.1)      | 220 (20.9)      | 170 (18.6)                 | 75 (20.1)   | \(\chi^2 = 19.5\), \(P < .001\) |
| Yes, 81      | 18 (9.7)        | 13 (5.5)        | 20 (5.3)                   | 20 (6.2)    | \(\chi^2 = 4.48\), \(P = .21\) |
| **Exercise regularly, n (%)** |                 |                 |                            |
| Yes, 60      | 498 (91.5)      | 964 (92.2)      | 838 (92.0)                 | 342 (91.9)  | \(\chi^2 = 0.19\), \(P = .98\) |
| Yes, 81      | 146 (78.9)      | 205 (86.9)      | 327 (88.1)                 | 284 (89.9)  | \(\chi^2 = 13.2\), \(P < .05\) |
| **Alcohol consumption, n (%)** |                 |                 |                            |
| Never, 60    | 51 (9.4)        | 98 (19.2)       | 99 (11.7)                  | 47 (13.8)   | \(\chi^2 = 7.89\), \(P = .246\) |
| 1–4 times a month, 60 | 339 (62.3)      | 560 (58.2)      | 504 (59.6)                 | 194 (57.1)  |                            |
| 2–7 times a week, 60 | 154 (28.3)      | 304 (31.6)      | 243 (28.7)                 | 99 (29.1)   |                            |
| Never, 81    | 64 (34.4)       | 53 (23.2)       | 82 (24.9)                  | 72 (23.3)   | \(\chi^2 = 28.3\), \(P = .000\) |
| 1–4 times a month, 81 | 106 (57.0)      | 124 (54.4)      | 177 (53.8)                 | 152 (49.2)  |                            |
| 2–7 times a week, 81 | 16 (8.6)        | 51 (22.4)       | 70 (21.3)                  | 85 (27.5)   |                            |
| **Cohabitant, n (%)** |                 |                 |                            |
| 60, yes      | 372 (68.4)      | 761 (72.1)      | 674 (73.8)                 | 269 (71.7)  | \(\chi^2 = 5.03\), \(P = .169\) |
| 81, yes      | 87 (46.8)       | 134 (55.8)      | 204 (54.3)                 | 174 (52.3)  | \(\chi^2 = 3.96\), \(P = .266\) |
| **Stroke, n (%)** |                 |                 |                            |
| Yes, 60      | 13 (2.4)        | 33 (3.1)        | 25 (2.7)                   | 14 (3.7)    | \(\chi^2 = 1.70\), \(P = .636\) |
| Yes, 81      | 28 (14.7)       | 36 (14.5)       | 62 (16.3)                  | 67 (19.9)   | \(\chi^2 = 4.05\), \(P = .256\) |

(Continues)
**TABLE 1** (Continued)

| Cohort | Myocardial infarction, n (%) |
|---------|-------------------------------|
| 160 (1941–1943) | Yes, 60 12 (2.2) 30 (3.8) 10 (3.0)  χ² = 3.68, P = .30 |
| 81 (1920–1922) | Yes, 81 31 (11.1) 52 (16.1) 38 (12.3)  χ² = 6.01, P = .11 |

| Systolic blood pressure, M (SD) |
| 60 | 141 (21.4) 137 (18.4) 140 (19.2) 130 (15.8)  F = 30.3, P < .000 |
| 81, M (SD) | 153 (25.1) 146 (213) 150 (21.2) 140 (21.2)  F = 16.6, P < .000 |

| Use antihypertensives, n (%) |
| 60 | Yes, 109 (19.8) 222 (27.8) 88 (26.3)  χ² = 12.1, P < .05 |
| 81 | Yes, 73 (38.4) 195 (60.2) 189 (61.2)  χ² = 32.3, P < .001 |

| Diabetes, n (%) |
| 60 | Yes, 40 (7.3) 54 (6.8) 21 (6.3)  χ² = 1.55, P = .67 |
| 81 | Yes, 11 (5.8) 43 (13.3) 39 (12.6)  χ² = 7.78, P < .05 |

| Depression, n (%) |
| 60 | No depression 456 (85.2) 692 (89.4) 296 (91.4)  χ² = 20.9, P < .001 |
| Mild, moderate, and severe 79 (14.8) 82 (10.6) 28 (8.6) |
| 81 | No depression 138 (79.3) 279 (92.4) 273 (92.9)  χ² = 27.1, P < .000 |
| Mild, moderate, and severe 36 (20.7) 23 (7.6) 21 (7.1) |

| MMSE, M (SD) |
| 60 | 27.8 (2.11) 27.9 (2.72) 27.9 (3.1)  F = 4.58, P < .005 |
| 81 | 26.2 (2.84) 26.6 (3.40) 26.7 (3.33)  F = 1.50, P = .212 |

Note: Cohort 1 was examined from 2001, cohort 2 from 2006, cohort 3 from 2012, and cohort 4 from 2018.

Abbreviations: BMI, body mass index, weight in kilograms divided by the square of the height in meters; MMSE, Mini-Mental State Examination; SD, standard deviation.

Despite emerging evidence that incident dementia is declining in Western countries, limited investigation on secular trends of prodromal stages of dementia such as MCI has been conducted. This study provides evidence for decline in MCI over the last 19 years in four separate cohort samples of Swedish adults aged 60 and 81. In addition, it reports that a severe form of MCI (i.e., multiple-domain MCI) has also decreased. On inspection of demographic, lifestyle, vascular and metabolic conditions, and depressive features, all evidently contributing to the development of dementia, cohort differences were detected. In addition to having higher education, the later birth cohorts had overall lower systolic blood pressure, used more antihypertensives, exercised more, smoked less, and were less depressed than the earlier birth cohorts. Contrary to this healthier trend, there was an increase in prevalent diabetes, higher BMI, and alcohol consumption. These factors could only in part explain the decrease in the observed MCI prevalence.
TABLE 2  Cohort-differences in prevalence of MCI, amnestic- and non-amnestic, single- and multi-domain

|                | Cohort 1 | Cohort 2 | Cohort 3 | Cohort 4 | \(\chi^2\) test and P-value | \(\chi^2\) linear-association and P-value |
|----------------|----------|----------|----------|----------|-----------------------------|-------------------------------------------|
| **60-year-olds** |          |          |          |          |                             |                                           |
| MCI, n (%)      | 122 (22.1) | 180 (16.9) | 132 (14.4) | 48 (13.0) | \(\chi^2 = 18.5, P < .001\) | \(\chi^2 = 16.9, P < .001\) |
| NCI, n (%)      | 431 (77.9) | 883 (83.1) | 782 (85.6) | 322 (87.0) |                             |                                           |
| Amnestic MCI, n (%) | 42 (34.4) | 60 (33.3) | 40 (30.3) | 19 (39.6) | \(\chi^2 = 1.45, P = .69\) | \(\chi^2 = 0.00, P = .95\) |
| naMCI, n (%)    | 80 (65.6) | 120 (66.7) | 92 (69.7) | 29 (60.4) |                             |                                           |
| Multidomain MCI, n (%) | 69 (66.3) | 113 (68.1) | 90 (72.6) | 35 (81.4) | \(\chi^2 = 4.01, P = .26\) | \(\chi^2 = 3.49, P = .06\) |
| MCI  | 35 (33.7) | 53 (31.9) | 34 (27.4) | 8 (18.6) |                             |                                           |
| MCIm | 35 (33.7) | 53 (31.9) | 34 (27.4) | 8 (18.6) |                             |                                           |
| **81-year-olds** |          |          |          |          |                             |                                           |
| MCI, n (%)      | 53 (29.0) | 44 (18.5) | 80 (22.0) | 60 (19.0) | \(\chi^2 = 8.54, P < .05\) | \(\chi^2 = 3.83, P < .05\) |
| NCI, n (%)      | 130 (71.0) | 194 (81.5) | 284 (78.0) | 256 (81.0) |                             |                                           |
| Amnestic MCI, n (%) | 21 (39.6) | 21 (47.7) | 35 (43.8) | 34 (56.7) | \(\chi^2 = 3.76, P = .29\) | \(\chi^2 = 2.45, P = .12\) |
| naMCI, n (%)    | 32 (60.4) | 23 (52.3) | 45 (56.3) | 26 (43.3) |                             |                                           |
| Multidomain MCI, n (%) | 36 (80.0) | 31 (75.6) | 55 (75.3) | 34 (73.9) | \(\chi^2 = 0.53, P = .91\) | \(\chi^2 = 0.51, P = .51\) |
| MCI  | 9 (20.0) | 10 (24.4) | 18 (24.7) | 12 (26.1) |                             |                                           |
| MCIm | 9 (20.0) | 10 (24.4) | 18 (24.7) | 12 (26.1) |                             |                                           |

Note: Forty-five of 32 (60/81-year-olds) participants did not have enough data to determine multiple or single MCI. Abbreviations: aMCI, amnestic mild cognitive impairment; MCI, mild cognitive impairment; MCIm, mild cognitive impairment multidomain; MCIs, mild cognitive impairment single domain; naMCI, non-amnestic mild cognitive impairment; NCI, no cognitive impairment.

4.1 Secular trends of MCI prevalence

There was a drop of 9.1 and 10 percentage points in MCI prevalence over the course of 19 years for the 60- and 81-year-olds, respectively. Our results that severity of MCI and overall prevalence of MCI are seemingly declining are consistent with studies reporting a secular decrease in dementia incidence. For example, a recent investigation using aggregated data from seven population-based studies in North America and Europe revealed a 13% decrease per decade in dementia incidence over the last 25 years.\(^6\) In addition, a systematic review,\(^21\) including 43 articles, determined mixed results where global dementia prevalence was on the rise, but a decline was observed in data after 2010 in the United States, UK, and Sweden. The same review concluded decrease or stable numbers for dementia incidence.

Recent Swedish data point to a more optimistic picture, in which population-based studies report a temporal decline in dementia prevalence (1986–2010) in rural (age 78+)+2 and urban areas (age 85).\(^22\) Additionally, survival rate after dementia diagnosis is increasing.\(^4,23\) However, a recent report by the Swedish National Study on Aging and Care, with data from four harmonized studies, including the GÅS-project, established stable prevalence between 2001 and 2010.\(^24\) Two studies from Stockholm suggest that incident dementia has decreased during the last 20 to 25 years.\(^23,25\) However, data from another large Swedish city reported stable 5-year incidence comparing 70-year-olds in 1971 to those in 2000.\(^26\) In summary, Sweden is one of the countries to repeatedly report declining numbers in dementia; still, not all Swedish studies propose decline.\(^27,28\) Further research is required to establish whether cognitive decline is occurring in the very recent years in Sweden and other high-income countries.\(^21\)

Studies reporting temporal trends on prodromal dementia are rare. Using data from the population-based study Einstein Aging Study (New York, USA)\(^10\) the authors concluded stability of incident aMCI among men and women aged 70+ examined in 1993 and 2016. Upon inspection of prevalent aMCI in our birth cohorts, for the 60-year-olds, a significant decrease was seen (i.e., C1: 8.9% vs. C4: 5.6%). Equally, there differences did not reach statistical significance. UK data (age 65+) from the Cognitive Function and Ageing Studies confirmed overall MCI prevalence to be stable between 1991 and 2011.\(^11\) Differences in the application of MCI definition (e.g., consensus vs. algorithmic approach), time periods, and geographical regions may explain inconsistencies in results, for example, overall MCI prevalence has been found to vary between 3.2% and 42% due to heterogeneity.\(^29\) At present, no studies have investigated temporal trends in severity of MCI. Last, it is worth mentioning that a Chinese study\(^12\) established increase in MCI prevalence (age 60+, MCI = 22.9% 2010 and 27.8% in 2015), which
TABLE 3  Odds ratios for prevalent MCI in four separate birth cohorts stratified by age groups 60 and 81

|                     | 60-year-olds | 81-year-olds |
|---------------------|--------------|--------------|
|                     | OR           | 95% CI (lower; upper) | P-value | OR           | 95% CI (lower; upper) | P-value |
| **Model 1 crude**   |              |               |         |              |               |         |
| Cohort 1 (reference)| .000         |               | .038    | .000         |               | .038    |
| Cohort 2            | 0.72         | (0.56; 0.93)  | .012    | 0.56         | (0.35; 0.88)  | .012    |
| Cohort 3            | 0.60         | (0.45; 0.78)  | .000    | 0.69         | (0.46; 1.04)  | .073    |
| Cohort 4            | 0.53         | (0.37; 0.76)  | .001    | 0.58         | (0.38; 0.88)  | .011    |
| **Model 2 demographic** |           |               |         |              |               |         |
| Cohort 1 (reference)| .004         |               | .032    | .004         |               | .032    |
| Cohort 2            | 0.80         | (0.62; 1.04)  | .101    | 0.53         | (0.53; 0.33)  | .008    |
| Cohort 3            | 0.64         | (0.49; 0.84)  | .002    | 0.72         | (0.72; 0.48)  | .126    |
| Cohort 4            | 0.59         | (0.41; 0.85)  | .005    | 0.57         | (0.57; 0.37)  | .014    |
| **Model 3 lifestyle** |           |               |         |              |               |         |
| Cohort 1 (reference)| .003         |               | .016    | .003         |               | .016    |
| Cohort 2            | 0.84         | (0.66; 1.14)  | .302    | 0.47         | (0.28; 0.78)  | .004    |
| Cohort 3            | 0.66         | (0.50; 0.88)  | .004    | 0.77         | (0.50; 1.18)  | .230    |
| Cohort 4            | 0.56         | (0.38; 0.81)  | .003    | 0.58         | (0.37; 0.92)  | .020    |
| **Model 4 metabolic, vascular health factors** | | | | | | |
| Cohort 1 (reference)| .006         |               | .004    | .006         |               | .004    |
| Cohort 2            | 0.85         | (0.64; 1.12)  | .240    | 0.43         | (0.24; 0.75)  | .003    |
| Cohort 3            | 0.64         | (0.48; 0.86)  | .003    | 0.69         | (0.43; 1.11)  | .128    |
| Cohort 4            | 0.59         | (0.40; 0.87)  | .008    | 0.47         | (0.27; 0.79)  | .005    |
| **Model 5 depression** | | | | | | |
| Cohort 1 (reference)| .006         |               | .020    | .006         |               | .020    |
| Cohort 2            | 0.87         | (0.66; 1.16)  | .551    | 0.49         | (0.27; 0.87)  | .015    |
| Cohort 3            | 0.64         | (0.48; 0.87)  | .004    | 0.83         | (0.51; 1.36)  | .458    |
| Cohort 4            | 0.59         | (0.40; 0.88)  | .007    | 0.51         | (0.29; 0.89)  | .019    |

Notes: Model 1: adjusted for birth cohort. Model 2: adjusted for sex and education. Model 3: adjusted for sex, education, smoking, exercise, cohabitant, alcohol usage, and BMI. Model 4: adjusted for sex, education, smoking, exercise, cohabitant, alcohol usage, BMI, stroke, myocardial infarction, diabetes, systolic blood pressure, and antihypertensives. Model 5: adjusted for sex, education, smoking, exercise, cohabitant, alcohol usage, BMI, stroke, myocardial infarction, diabetes, antihypertensives, and depression.

Abbreviations: BMI, body mass index; CI, confidence interval; MCI, mild cognitive impairment; OR, odds ratio.

is not surprising as the decline in cognitive impairment is mostly seen in high-income countries.21 Remarkably, although previous results are inconsistent, the reported stability or decline is still detected despite increased survival rates in the cognitively impaired and with an increasing aging population supposedly enhancing prevalence numbers. Perhaps then decline is larger than described in prior research.

Declining rates of dementia incidence together with stable MCI might be suggestive of prolonged stages of MCI.10 Our findings together with previous Swedish research signifying decline in dementia incidence advocates that the time between cognitive health and dementia has not temporally changed, rather it supports the proposition that the entire process of developing dementia has been extended.9,30–32 Determining temporal trends of incident MCI could further provide evidence for this argument.

4.2 Exploratory factors of MCI prevalence

The explanatory factors for MCI trends in this study were chosen due to evidence linking them to dementia13 and MCI14 and that a substantial number of these factors have, concurrently to declining trends of dementia, also changed throughout the last decades. For instance, management of vascular disease such as regulation of hypertension33 and treatment of stroke34 has improved and supposedly results in fewer dementia cases.35 Still, similar to our conclusions, studies adjusting for vascular factors, such as stroke, hypertension, antihypertensives, and myocardial infarction do not detect attenuated cohort trends on dementia.5,7,8,25 The positive health trends seen in the GÅS-samples are consistent with national4,25,36 and global trends from other high-income countries37,38 showing older adults are smoking less, have lower blood pressure, use more antihypertensives, and are more engaged in physical activities. Yet, it remains unclear why these positive trends could not explain decline in MCI prevalence.

On a more detrimental note, our results were consistent with prior Swedish research25,39 and studies from other high-income countries40 reporting that alcohol consumption, especially moderate to high consumption in the older population, is seemingly increasing. Controlling for alcohol consumption did not alter observed cohort trends, although higher ORs for MCI were observed for non-drinkers compared to drinkers. Nevertheless, individuals with MCI and high alcohol
consumption have a higher risk of developing dementia,41 therefore the escalation in alcohol intake is of public health concern. Other risk factors for MCI, MCI progression, and dementia include obesity42,43 and diabetes44–46 and consistent with our results these conditions are increasing worldwide.47 Perhaps the higher diabetes prevalence in later birth cohorts reflects increased survival and improved treatment among the older diabetics in our sample.5 Albeit, increase in obesity and diabetes may adversely affect rates of MCI prevalence and dementia incidence in coming decades.6 This negative inclination together with positive trends in lifestyle and vascular factors makes underlying causes for temporal trends in dementia and MCI difficult to untangle as one beneficial health factor might be eliminated by another non-beneficial factor.

When stratified by sex, regression analyses revealed that prevalence of MCI declined in both sexes (data not shown). Notably, the decrease was only statistically significant among women, indicating that the overall observed decline of MCI was predominantly driven by women, consistent with previous Swedish data on dementia decline.25 Women are more at risk for development of dementia, at least for AD, and a decrease of MCI among women could infer a narrowing of the gap between the sexes and dementia risk; further research on the matter is therefore warranted.

As level of education and overall cognitive functioning has increased globally the last century48 and low education is considered a risk factor for cognitive impairment, it was thought that education could explain some of the observed cohort trends. Indeed, adjusting for education did attenuate differences in MCI prevalence between C1 and C2 in the 60-year-old group; however, no other reductions in significance levels or ORs were observed.

There is evidence that level of education can explain a majority of Swedish cohort differences. For instance, differences in prevalent dementia between 85-year-olds examined in 1986 and 2008 were fully attenuated when adjusted for education.22 Additionally, in a study with US data, education and net worth explained up to 43% of the cohort differences in prevalent cognitive impairment.40 On the contrary, detected decline in dementia incidence for 70-year-olds examined in 1971 and 2000 remained when adjusting for education, despite significant differences in attained education between cohorts. This holds true for several studies trying to explain cohort differences in cognitive impairment with differences in educational level.5,25,31

The additional analyses to inspect whether the observed decline in MCI prevalence was similar in all educational levels provided inconclusive results.

Indeed, for our 60-year-olds, the earliest born cohorts still had higher odds of MCI prevalence compared to the later born cohorts; however, the decrease was not as prominent as when the analyses were run with all educational groups together. For the 81-year-olds, none of the significant differences in odds remained when stratified by education; however, it is likely that the groups were too small to detect differences. Prior research also provides inconclusive results, with dementia decline primarily in either low or in the higher educated groups or equal decline in all educational groups.7,22,25,50 Fewer cases of cognitive impairment in one specific educational group could reflect that quality of education in that specific group has improved more so than in other educational groups. However, the results for the 60-year-olds indicate that age-related cognitive impairment has decreased similarly across all educational levels.

Educational attainment is proposed to represent cognitive functioning and cognitive reserve,49 yet it may not sufficiently represent the observed cognitive gains. Other cognitively stimulating activities throughout life such as work complexity may perhaps be better measurements of cognitive functioning in later life. Further investigation is warranted to determine whether there is an unequal cognitive gain among different sociodemographic groups.

In summary, the explored factors could only partially explain the observed decline, despite adjusting for education-based risk factors for MCI and dementia. Risk factors from a life-course perspective are desirable as certain conditions, for example hypertension or obesity, are suggested to be more detrimental to cognitive health in midlife than having the same condition in later life.53 Cohort differences in childhood nutrition could also play a role in late-life cognition. Supplementary research with life-course perspectives to explain underlying causes of decline in MCI and dementia is therefore warranted.

### 4.3 Strengths and limitations

To the authors’ knowledge, this is the first European study to report secular trends for both amnestic and non-amnestic MCI. Strengths include that MCI diagnosis was based on the same standardized study assessments applied uniformly throughout the study. This is especially important when comparing diagnoses from different time periods as changes in diagnostic criteria (e.g., causing an increase in MCI detection) can possibly disguise a true decline in MCI. Another strength is that the sample includes data from both rural and urban areas, improving generalizability of results.

This study has some limitations. First, baseline data was used to create norm scores for all cohorts, perhaps leading to underdiagnosis of MCI in later birth cohorts due to the potential usage of outdated norms. Noticeably, previous analyses with our data have shown so-called Flynn effects (i.e., generational improvement on cognitive performance) exclusively on speed of processing task, leaving a very small impact on MCI diagnosis. In addition, we applied norm scores corrected for education, probably reducing cohort effects. Second, there were few cases stratified into amnestic/non-amnestic and single/multiple MCI, particularly for 81-year-olds, which limited us to engage in further cohort analyses on subtypes. Third, our study design limits the investigation of improved cognition in age groups between 60 and 80 and 81+ years of age. It would have been desirable to have these age groups to confirm the secular decrease in MCI. Markedly, using 60-year-olds to examine pre-stages of dementia may not be optimal, as cognitive impairment in these age groups could reflect other issues such as stress or work-overload or early signs of non-apparent cardiovascular conditions, all affecting cognition. Even if overall MCI for our 60-year-olds was slightly higher than previously reported (e.g., 16.5% vs. 13.4%), this study still provides valuable evidence on temporal improved
cognition (MCI diagnosis) in younger older adults, and arguably better cognitive reserve protects against developing dementia.

4.4 Concluding remarks

Even if the total numbers of dementia increase due to population aging, a slight postponement in the onset of dementia could have a substantial impact on future public health burden. Thus, the results presented here of declining MCI together with others reporting decline in dementia incidence provide an optimistic outlook.

ACKNOWLEDGMENTS

The authors want to thank the GÅS participants and GÅS team members for collecting the data and a special thanks to Aldana Rosso for her valuable input on the final stages of the manuscript. The SNAC-GÅS study is funded by Swedish Ministry of Health and Social Affairs, the county Region Skåne, the Medical Faculty at Lund University, and the Swedish Research Council (grant numbers 2017-01613, 2017-00639, 2021-01437).

CONFLICTS OF INTEREST

The authors report no conflicts of interest.

REFERENCES

1. Prince M, Bryce R, Albanese E, Wimo A, Ribeiro W, Ferri CP. The global prevalence of dementia: a systematic review and metaanalysis. Alzheimers Dement. 2013;9:63-75.e2.
2. Matthews FE, Arthur A, Barnes LE, et al. A two-decade comparison of prevalence of dementia in individuals aged 65 years and older from three geographical areas of England: results of the Cognitive Function and Ageing Study I and II. Lancet. 2013;382:1405-1412.
3. Langa KM, Larson EB, Crimmins EM, et al. A Comparison of the Prevalence of Dementia in the United States in 2000 and 2012. JAMA Intern Med. 2017;177:51-58.
4. Wimo A, Sjölund BM, Sköldunger A, et al. Cohort Effects in the Prevalence and Survival of People with Dementia in a Rural Area in Northern Sweden. J Alzheimers Dis. 2016;50:387-396.
5. Derby CA, Katz MJ, Lipton RB, Hall CB. Trends in Dementia Incidence in a Birth Cohort Analysis of the Einstein Aging Study. JAMA Neurol. 2017;74:1345-1351.
6. Wolters FJ, Chibnik LB, Waziry R, et al. Twenty-seven-year time trends in dementia incidence in Europe and the United States: The Alzheimer Cohorts Consortium. Neurology. 2020;95:e519-e531.
7. Satizabal CL, Beiser AS, Chouraki V, Chêne G, Dufouil C, Seshadri S. Incidence of Dementia over Three Decades in the Framingham Heart Study. N Engl J Med. 2016;374:523-532.
8. Ahmadi-Abhari S, Guzman-Castillo M, Bandosz P, et al. Temporal trend in dementia incidence since 2002 and projections for prevalence in England and Wales to 2040: modelling study. BMJ. 2017;358:j2856.
9. Prince M, Ali GC, Guerchet M, Prina AM, Albanese E, Wu YT. Recent global trends in the prevalence and incidence of dementia, and survival with dementia. Alzheimers Res Ther. 2016;8:23.
10. Derby CA, Katz MJ, Rozner S, Lipton RB, Hall CB. A Birth Cohort Analysis of Amnestic Mild Cognitive Impairment Incidence in the Einstein Aging Study (EAS) Cohort. J Alzheimers Dis. 2019;70:5271-s281.
11. Richardson C, Stephan BCM, Robinson L, et al. Two-decade change in prevalence of cognitive impairment in the UK. Eur J Epidemiol. 2019;34:1085-1092.

12. Lu H, Wang XD, Shi Z, et al. Comparative analysis of cognitive impairment prevalence and its etiological subtypes in a rural area of northern China between 2010 and 2015. Sci Rep. 2019;9:851.
13. Livingston G, Huntley J, Sommerlad A, et al. Dementia prevention, intervention, and care: 2020 report of the Lancet Commission. Lancet North Am Ed. 2020;396:413-446.
14. Campbell NL, Unverzagt L, LaMantia MA, Khan BA, Boustani MA. Risk factors for the progression of mild cognitive impairment to dementia. Clin Geriatr Med. 2013;29:873-893.
15. Petersen RC, Caracciolo B, Brayne C, Gauthier S, Jelic F, Fratiglioni L. Mild cognitive impairment: a concept in evolution. J Intern Med. 2014;275:214-228.
16. Ekström H, Elmståhl S. Pain and fractures are independently related to lower walking speed and grip strength: results from the population study “Good Ageing in Skåne”. Acta Orthopaedica. 2006;77:902-911.
17. Overton M, Elmståhl S, Pihsolgård M. Prevalence and incidence of Mild Cognitive Impairment across subtypes, age, and sex. Dement Geriatr Cogn Disord. 2019;47:219-232.
18. Overton M, Pihsolgård M, Elmståhl S. Test administrator effects on cognitive performance in a longitudinal study of ageing. Cogent Psychology. 2016;3:1260237.
19. Montgomery SA, Asberg M. A new depression scale designed to be sensitive to change. Br J Psychiatry. 1979;134:382-389.
20. Katz S, Akpm CA. A Measure of Primary Sociobiological Functions. Int J Health Serv. 1976;6:493-508.
21. Stephan BCM, Birdi R, Tang EYH, et al. Secular Trends in Dementia Prevalence and Incidence Worldwide: A Systematic Review. J Alzheimers Dis. 2018;66:653-680.
22. Skoog I, Börjesson-Hanson A, Kern S, et al. Decreasing prevalence of dementia in 85-year olds examined 22 years apart: the influence of education and stroke. Sci Rep. 2017;7:6136.
23. Qiu C, von Strauss E, Bäckman L, Winblad B, Fratiglioni L. Twenty-year changes in dementia occurrence suggest decreasing incidence in central Stockholm, Sweden. Neurology. 2013;80:1888-1894.
24. Fratiglioni L, Ding M, Santoni G, et al. Demensförekomst i Sverige: geografiska och tidsmässiga trender 2001-2013. Resultat från den svenska nationella studien om åldrande, vård och omsorg - SNAC. Stockholm 2017.
25. Ding M, Qiu C, Rizzuto D, Grande G, Fratiglioni L. Tracing temporal trends in dementia incidence over 25 years in central Stockholm, Sweden. Alzheimers Dement. 2020;16:770-778.
26. Sacuiu S, Gustafson D, Jörgensen M, et al. Secular changes in cognitive predictors of dementia and mortality in 70-year-olds. Neurology. 2010;75:779-785.
27. Wiberg P, Waern M, Billstedt E, Ostling S, Skoog I. Secular trends in the prevalence of dementia and depression in Swedish septuagenarians 1976-2006. Psychol Med. 2013;43:2627-2634.
28. Mathillas J, Löveheim H, Gustafson Y. Increasing prevalence of dementia among very old people. Age Ageing. 2011;40:243-249.
29. Ward A, Arrighi HM, Michels S, Cedarbaum JM. Mild cognitive impairment: Disparity of incidence and prevalence estimates. Alzheimers Dement. 2012;8:14-21.
30. Dufouil C, Beiser A, Chêne G, Seshadri S. Are Trends in Dementia Incidence Associated With Compression in Morbidity? Evidence From The Framingham Heart Study. J Gerontol B Psychol Sci Soc Sci. 2018;73:565-572.
31. Jagger C, Matthews FE, Wohland P, et al. A comparison of health expectancies over two decades in England: results of the Cognitive Function and Ageing Study I and II. Lancet. 2016;387:779-786.
32. Langa KM, Larson EB, Karlawish JH, et al. Trends in the prevalence and mortality of cognitive impairment in the United States: is there evidence of a compression of cognitive morbidity? Alzheimers Dement. 2008;4:134-144.
33. Danaei G, Finucane MM, Lin JK, et al. National, regional, and global trends in systolic blood pressure since 1980: systematic analysis of
health examination surveys and epidemiological studies with 786
country-years and 5.4 million participants. *Lancet*. 2011;377:568-577.

34. Feigin VL, Forouzanfar MH, Krishnamurthi R, et al. Global and regional
burden of stroke during 1990–2010: findings from the Global Burden
of Disease Study 2010. *Lancet North Am Ed*. 2014;383:245-255.

35. Winblad B, Amouyel P, Andrieu S, et al. Defeating Alzheimer’s disease
and other dementias: a priority for European science and society.
*Lancet Neurol*. 2016;15:455-532.

36. Johansson S, Wilhelmsen L, Welin C, Eriksson H, Welin L, Rosengren A. Obesity, smoking and secular trends in cardiovascular risk factors in middle-aged women: data from population studies in Göteborg from 1980 to 2003. *J Intern Med*. 2010;268:594-603.

37. Zhou B, Bentham J, Di Cesare M, et al. Worldwide trends in blood pressure from 1975 to 2015: a pooled analysis of 1479 population-based measurement studies with 19.1 million participants. *Lancet North Am Ed*. 2017;389:37-55.

38. Pesce G, Marcon A, Calciano L, et al. Time and age trends in smoking cessation in Europe. *PLoS One*. 2019;14:e0211976-e.

39. Waern M, Bentham J, Di Cesare M, et al. Worldwide trends in blood pressure from 1975 to 2015: a pooled analysis of 1479 population-based measurement studies with 19.1 million participants. *Lancet North Am Ed*. 2017;389:37-55.

40. Koch M, Fitzpatrick AL, Rapp SR, et al. Alcohol Consumption and Risk of Dementia and Cognitive Decline Among Older Adults With or Without Mild Cognitive Impairment. *JAMA Network Open*. 2019;2:e1910319-e.

41. Albanese E, Launer LJ, Egger M, et al. Body Mass Index in midlife and dementia: Systematic review and meta-regression analysis of 589,649 men and women followed in longitudinal studies. *Alzheimers Dement (Anst)*. 2017;8:165-178.

42. Cova I, Clerici F, Maggiore L, et al. Body Mass Index Predicts Progression of Mild Cognitive Impairment to Dementia. *Dement Geriatr Cogn Disord*. 2016;41:172-180.

43. Chatterjee S, Peters SA, Woodward M, et al. Type 2 Diabetes as a Risk Factor for Dementia in Women Compared With Men: A Pooled Analysis of 2.3 Million People Comprising More Than 100,000 Cases of Dementia. *Diabetes Care*. 2016;39:300-307.

44. Luchsinger JA, Reitz C, Patel B, Tang MX, Manly JJ, Mayeux R. Relation of diabetes to mild cognitive impairment. *Arch Neurol*. 2007;64:570-575.

45. Pal K, Mukadam N, Petersen I, Cooper C. Mild cognitive impairment and progression to dementia in people with diabetes, prediabetes and metabolic syndrome: a systematic review and meta-analysis. *Soc Psychiatry Psychiatr Epidemiol*. 2018;53:1149-1160.

46. Friedrich MJ. Global Obesity Epidemic Worsening. *JAMA*. 2017;318:603.

47. Trahan LH, Stuebing KK, Fletcher JM, Hiscock M. The Flynn effect: a meta-analysis. *Psychol Bull*. 2014;140:1332.

48. Fratiglioni L, Hui-Xin W. Brain Reserve Hypothesis in Dementia. *J Alzheimers Dis*. 2007;12:11-22.

49. Sullivan KJ, Dodge HH, Hughes TF, et al. Declining Incident Dementia Rates Across Four Population-Based Birth Cohorts. *J Gerontol A Biol Sci Med Sci*. 2019;74:1439-1445.

50. Overton M, Pihlsgård M, Elmståhl S. Up to speed: Birth cohort effects observed for speed of processing in older adults: Data from the Good Ageing in Skåne population study. *Intelligence*. 2018;67:33-43.

51. Kumar R, Dear KB, Christensen H, et al. Prevalence of mild cognitive impairment in 60- to 64-year-old community-dwelling individuals: The Personality and Total Health through Life 60+ Study. *Dement Geriatr Cogn Disord*. 2005;19:67-74.

**SUPPORTING INFORMATION**

Additional supporting information may be found in the online version of the article at the publisher’s website.

---

**How to cite this article:** Overton M, Pihlsgård M, Elmståhl S. Secular trends in prevalent mild Cognitive impairment: Data from the Swedish population-based study Good Aging in Skåne. *Alzheimer's Dement*. 2022;8:e12260.

https://doi.org/10.1002/trc2.12260