Impact of Stroke on Cognition in Old Age: Comparison of Two Population-Based Cohorts, Born Up to 30 Years Apart and Followed from Age 70 to 85

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Abstract

Little is known about birth cohort differences in the impact of stroke on cognitive aging. Given improved post-stroke rehabilitation and better treatments for vascular-health risk, we may expect a reduction in the stroke impact in later born cohorts. We tested this prediction using data from two cohorts, born in 1901-07 (n = 1155) and 1930 (n = 919), identified from the same city population at the same age of 70 and subsequently measured on the same cognitive outcomes (i.e., spatial ability, perceptual-motor-speed, and reasoning) at ages 70, 75, 79, and 85. We fitted multiple-group second-order latent growth curve models to the data, regressing the first-order cognitive factor on the time-varying stroke variable, controlling for relevant covariates. Findings revealed moderate to large average cognitive decline (d = -.45) following stroke and the impact was relatively similar across cohorts (1901-07: d = -.52; 1930: d = -.39). However, there was a stroke by age by cohort interaction implying that the stroke impact increased with age in the 1901-07 cohort ($d_{\text{age}\leq75}$ = -.42; $d_{\text{age}\geq79}$ = -.70) but decreased in the 1930 cohort ($d_{\text{age}\leq75}$ = -.53; $d_{\text{age}\geq79}$ = -.17). We found no evidence for lagged effect of stroke beyond impact on measures following the incidence. Our hypothesis was only partially supported, as the impact of stroke was reduced in the later born cohort, but solely at higher ages.

Keywords: aging, cognition, cognitive decline, stroke, birth cohort differences.

Public significance statement: This study suggests that cognitive decline following a stroke is considerable and relatively similar across birth cohorts. However, with age, there is an increase in the impact among members of earlier born cohorts but a decrease among members of later born cohorts. The findings may have implications for predictions of future healthcare challenges.

Post-stroke cognitive impairment is common among affected individuals, frequently with substantial challenges for patients, caregivers, and societies. A number of studies (e.g., Levine et al., 2015; Rajan et al., 2014; Toole et al., 2004; Zheng et al., 2019) have quantified the impairment in cognitive functioning after stroke, although others (e.g., Köhler et al., 2012) have not been able to replicate the findings. Reported effect sizes from these studies also vary considerably (for review see Tang et al., 2018), mostly due to differences in sample composition, study design (i.e., number and length of follow-ups), type of outcomes, and analytical methods. The combined evidence, however, leans toward undesirable effects on subsequent cognitive functioning, although the extent of the decline is largely unknown and most likely varies across populations and birth cohorts.

Notably, little is known about potential birth cohort difference in the post-stroke impact among older adults. Given improved post-stroke rehabilitation, more precise diagnostics, and better medical treatments and follow-ups of vascular health risk factors, such as hypertension, diabetes, and hyperlipidemia, we could expect that the post-stroke cognitive impairment effects should be reduced among individuals in later born cohorts. The age of the patients at the time of the stroke incidence is also known as a recovery prognosis moderator (e.g., Pohjasvaara et al., 1997; Yoo et al., 2020). Advanced ages are associated with higher comorbidity prevalence (e.g., cardio- and cerebrovascular-diseases) that reduce recovery potentials. We may therefore also expect that the beneficial effects of improved rehabilitation, diagnostics, and treatments in the later born cohort, are particularly evident in the higher ages. To our knowledge, no previous study has evaluated birth cohort differences in this respect.

The purpose of our study was twofold. First, to add further evidence of subsequent impact of stroke on cognitive functioning in a population-based sample. Secondly, to provide formal testing and estimate of potential birth cohort differences in the impact of stroke on later cognitive functioning. To accomplish these aims, we analyzed data from two birth cohorts, born up to 30 years apart, identified and drawn from the same city population at the same baseline age of 70, and then measured on the same three cognitive tests at the same ages of 70, 75, 79, and 85. This unique study design allows for a detailed birth cohort comparison of the post-stroke impact.

Analyses of potential birth cohort differences in the post-stroke impact and later trajectories are valuable for several reasons. First, such evaluations provide information concerning generalizability and replication of findings across generations. Each generation of older individuals is exposed to unique living conditions (e.g., in terms of healthcare systems, public health care recommendations, and lifestyle norms) which can moderate the degree of stroke impact on cognition in older adults. Second, besides comparisons of raw format effect sizes, they also allow for quantification of stroke impact as conditioned on the cohort specific level and rate of change. An equal sized stroke impact leads consequently to a higher proportion of the population falling below a healthcare dependency threshold in birth cohorts that generally have lower cognitive ability (ceteris paribus). Also, quantification of the stroke impact as conditioned on cohort specific age-related cognitive decline facilitates an evaluation of the relative importance of stroke in relation to other causes of age-related cognitive decline.

Third, evidence of time trends in the impact of stroke on cognition may assist decision making and predication of future healthcare challenges. Post-stroke cognitive impairment entails substantial financial expenditure for societies (i.e., in terms of healthcare cost, rehabilitation, and long-term care), affected individuals (i.e., loss of productivity and work capacity) and caretakers (i.e., need for assistance in daily living, long-term home care) (Feigin et al., 2022). Documented time trends in the relative importance of stroke for cognitive aging development can improve such predictions by providing information about the expected population proportion falling below healthcare dependency thresholds. Fourth, information about time trends in the cognitive impact of stroke can contribute with cohort specific interventions benchmarks, or yardsticks facilitating the quantification of "true" effectiveness of interventions aimed at reducing cognitive decline after stroke in older adults.

Our main focus in the present study was on birth cohort comparisons in age-related decline as conditioned on stroke incidence. The three cognitive tests that we incorporated into our analyses all aimed to measure various aspects of a latent trait of fluid-like global cognition (i.e., reasoning ability, spatial ability, and perceptual-motor speed). We therefore analyzed the data using a multiple-group second-order latent growth curve model (LGCM; McArdle, 1988; Tisak & Meredith, 1990). The benefit of fitting growth curve models to a latent trait, instead of manifested test variables, is that it improves measurement accuracy, by separating true and error variance, thereby leading to higher estimate precision in the structural part of the model (von Oertzen, et al., 2010), which can be essential when testing higher-order interactions (e.g., stroke by age by cohort). Furthermore, the multiple-group and higher-order aspects of the models allowed for rigid evaluations and modeling of measurement invariances (see e.g., Bontempo & Hofer, 2007; Ferrer et al., 2008) across cohorts, ages, and their interactions, thereby facilitating validity evaluations of the cohort difference comparisons.

Method

Transparency and openness

Data can be made available to researchers meeting access criteria for deidentified data as defined by the H70 study steering board[1]. The analytical code is available from the first author. The study was not formally preregistered.

Participants

All participants were identified and systematically selected in a similar manner from the Swedish Revenue Office Register as part of the Gothenburg H70 studies. Members of the earliest birth cohort (n=1148) were all born between 1st July 1901 to 30th June 1902. The baseline participation rate for this cohort was 85%. Of this sample, 40% (n=460) were randomly selected for participation in the neuropsychological part of the study. Participation rate for this subsample was 84% (n=387). Additional information about demographics for this subsample can be found in Rinder et al., (1975) and Svanborg (1977). In 1986 (at age 85) the remainder of the surviving population identified in 1971 was invited additionally to study participation (n=370 with 60% participation rate; i.e., in addition to those that had participated previously; for more information about this sub-sample see Thorvaldsson et al., 2006). This proportion of the sample only contributed to estimation of individual differences in level of performance at age 85 and not rate of change. Members of the second cohort (n=1281) were born between 1st July 1906 and 30th June 1907 on days ending with 2, 5, and 8. Baseline participation rate for this cohort was 81%. Among these, 40% (n=512) were randomly selected for neuropsychological assessments with participation rate of 79% (n=402). For additional information about this cohort, see Nilsson (1983). Given the purpose of the current analyses we combined the 1901-02 and 1906-07 cohort data to a single cohort (i.e., cohort 1901-07, n = 1159).

Members of the third birth cohort were born in 1930 on days ending with 2, 3, 5, 6, 11, 12, 16, 18, 20, 21, 24, 27, or 30 of each month (n=1250). Participation rate for this subsample was 74% (n=921). Additional information about this subsample can be found in Wiberg et al., (2013). In total, data from 2080 individuals were potentially applicable for the presented data analyses. However, we omitted 6 individuals (4 from cohort 1901-07 and 2 from cohort 1930) as due to severe cognitive impairment already at the baseline age of 70 (defined as zero value on all three cognitive tests) giving us a sample of 2074 individuals to be analyzed. Among these, 898 contributed with cognitive data on one occasion; 448 on two occasions; 590 on three occasions; and 138 on four occasions. In total, we used 10867 data points to define the outcome measure, thereof: 2594 at age 70; 3379 at age 75; 2519 at age 79; and 2375 at age 85. There were 97 individuals who contributed data to the analyses from a single cognitive measurement; 109 from two measurements; 706 from three; 61 from four; 173 from five; 236 from six; 264 from seven; 68 from eight; 236 from nine; 43 from ten; 9 from eleven; and 72 from twelve. The H70 studies were approved by the ethical review board at the University of Gothenburg.

Cognitive measures

Block Design is a measure spatial ability (Dureman et al., 1971). Participants were given colored blocks and asked to construct replicas of prototype model designs presented to the participants in two colors. Seven prototypes were presented with an increasing difficulty. The performance was scored based on how fast the participants correctly replicate the prototypes (incomplete replication was scored zero). Maximum score was 42 and the total time limit was 20 minutes.

Figure Identification is a measure of perceptual-and motor-speed (Dureman et al., 1971). Participants were asked to match, as quickly as possible, a target figure with one identical figure placed in line among four others. The total raw score was calculated as total correct items – (total wrong items / 4), as to penalize for wrong answers and guessing. Maximum score was 60 (1 point an item) and the time limit was 4 minutes.

Figure Logic is a nonverbal measure of inductive reasoning (Dureman et al., 1971). Participants were presented with geometrical figures, organized in rows of five figures per row, and asked to identify the figure that differed in some specific respect from the other four figures. Individual total raw score was calculated as total correct items – (total wrong items/4) to adjust for wrong answers and guessing. The items were not presented in an order of difficulty and the participants were encouraged not to dwell too long on each item if they encountered difficulties. The time limit was 8 minutes, and the maximum score was 30 (i.e., 1 point per item).

At age 70 (i.e., baseline age) half of the cohort born in 1901/02 was randomly selected to take only the Figure Logic test, and at age 70, only 1/3 of the cohort born in 1930 took the cognitive test battery. We can consider this part of the missing data as completely missing at random (MCAR), as traditionally defined (e.g., Graham, 2009), given design-based random causes of missingness. Summary of participation frequency, stratified by birth cohort and age, can be found in Table S1 as part of the supplementary material.

Covariates

Information about the incidence of ischemic and hemorrhagic stroke was symptom-based and gathered in a similar manner across birth cohorts by self-reports during in-person examinations, from close relative informants, or from medical records. Transient ischemic attacks (TIA; i.e., mini-strokes) and silent cerebral infarctions (SCI; i.e., silent strokes) were not included as part of the stroke variable. Given differences in the educational system for our birth cohorts, we simplified the education variable by dichotomizing it, coding 0 = formal education or less, and 1 = more than formal education. Compulsory formal education (called "*folkskola"*) was 6 years for the 1901-07 cohort and 7 years for the 1930 cohort. Marital status was defined at baseline and coded 0 = single and 1 = married or cohabitation. The smoking variable was defined at baseline and coded 0 = non-smoker, 1 = previous smoker; 2 = current smoker. Diabetes and hypertension were defined by self-report during in-person examinations and by medical records.

Statistical analysis

We fitted multiple-group second-order latent growth curve models (McArdle, 1988; Tisak & Meredith, 1990; for review see e.g., Ferrer et al., 2008) to the data within the analytical framework of structural equation modeling (SEM). In all models, we defined the "groups" moderator as birth cohort (i.e., 1901-07 and 1930), and the first-order factor structure of the cognitive latent trait by the three manifested cognitive tests outcomes (i.e., spatial ability, perceptual-motor speed, and reasoning), at each measurement occasion (i.e., at ages 70, 75, 79, and 85). A path diagram of the final model is shown in Figure 1. As conventionally defined: ovals (or circles) refer to latent variables, squares (or boxes) to manifested (or measured) variables, two-headed arrows to covariances and one-headed arrows to regression coefficients (or path regression). In all models, we scaled the cognition factor at each age using the effect coding method suggested by Little et al., (2006). This procedure constrains the set of indicator intercepts to sum to zero and factor loadings to mean value of one within the specific factor. The benefit of this scaling method is that it allows all factor intercepts and loadings to be estimated as free parameters. That is, there is no need to fix selected intercepts and loadings to zero and one, respectively (as is commonly done), in order to scale the factors. We further scaled the age slope second-order factor as implying expected change by one year on the cognitive ability first-order factor. In order to facilitate convergence of the estimation procedure we coded the factor loadings of the age slope as -9, -4, 0, 6, thereby centering the intercept second-order factor at the age 79 measurement occasion. We regressed the time constant covariates (i.e., sex, education, smoker, marital status, diabetes, hypertension, and stroke) on the growth factors (i.e., intercept and age slope) thereby conditioning the time varying impact of stroke on cognition at ages 70, 75, 79, and 85. Prior to fitting the second-order growth curve models, we defined the measurement model and implemented metric and scale invariance testing (see e.g., Widaman et al., 2010) across both cohorts and ages using confirmatory factor analysis models. We estimated all parameters using the robust maximum likelihood alternative implemented to the lavaan R package (version 0. 6. 7; Rosseel, 2012) and for handling missing data we used extrapolation via maximum likelihood under the assumption of missing at random, as conventionally defined (see e.g., Graham, 2009). We standardized relevant effect sizes using the across cohorts combined age 79 variability components as derived from an unconditional second-order growth curve model.

Results

Descriptives for the analyzed sample can be found in Table 1. The sex distribution was similar across cohorts ($\chi^2(1) = 2.05$, $p = .15$). The 1930 cohort had a higher proportion of individuals with more than formal education ($\chi^2(1) = 41.23$, $p < .001$), that were married or cohabiting ($\chi^2(1) = 17.12$, $p < .001$), and were diagnosed with diabetes ($\chi^2(1) = 6.17$, $p < .013$) and hypertension ($\chi^2(1) = 106.65$, $p < .001$). The distribution for the smoker variable differed across cohorts ($\chi^2(2) = 100.39$, $p < .001$) with the 1930 cohort reporting lower proportion of non-smoker ($p < .001$; all post-hoc comparisons were conducted using Bonferroni correction), higher proportion previous smoker ($p < .001$), and lower proportion current smoker ($p = .001$). The 1930 birth cohort also had higher stroke incidence occurrence at all ages, although the relative comparisons were non-significant (i.e., 70 ($\chi^2(1) = 3.68$, $p = .06$); 75 ($\chi^2(1) = 2.64$, $p = .10$); 79 ($\chi^2(1) = 2.73$, $p = .10$); and 85 ($\chi^2(1) = 1.10$, $p = .30$). In total there were 296 individuals with a stroke history, thereof 118 in cohort 1901-07 and 178 in cohort 1930. As expected, the likelihood of stroke occurrence was higher for those participants that had a diagnosis of hypertension (OR = 1.44, 95% CI[1.11, 1.86]) and diabetes (OR = 1.71, 95% CI[1.14, 2.55]), but stroke was only marginally associated with smoking (using non-smoker as references category: $OR_{\text{Previous smoker}}$ = 1.12 ,95% CI[0.82, 1.53]; $OR_{\text{Current smoker}}$ = 1.33 ,95% CI[0.97, 2.01]; see other estimates in supplementary material Table S2). None of the associations of stroke occurrence with the other control variables included in our analyses differed by birth cohorts ($p > .05$).

The correlation matrix for the cognitive tests, including means and standard deviations, as stratified by cohort is shown in Table 2. Comparisons of the mean values (in the third column) demonstrate large cohort differences on all tests and ages in favor of the 1930 birth cohort (for more detailed reporting of cohort differences in the H70 study see Karlsson et al., 2015; Thorvaldsson et al., 2017; factor level mean cohort comparisons by age are shown in supplementary material Table S5). Correlations for the 1901-07 cohort are presented below the matrix diagonal, and for the 1930 cohort above the diagonal. The intra-occasion correlations across the tests (marked in bold) were in the range from .36 to .74. These correlations were generally higher in the 1901-07 cohort (mean r = 63), in comparison with the 1930 cohort (mean r = .50). There are few oddities in the data reported in Table 2. For the reasoning ability measure at age 79, the sample size is relatively small (i.e., n=181). This test was omitted for a randomly selected part of the sample at this occasion (i.e., in 1980), due to time constraints. Also, the distribution in the speed test at age 75 for cohort 1930 is smaller than expected and the distribution in the reasoning test at age 85 in the 1901-07 cohort is larger than expected. Both of these oddities can be explained by the inclusion of slightly different versions of these tests (by mistake) on these occasions. We do not expect that this contributed to difference in the rank-order of these variables (only difference in variances) nor do we see any evidence that this may have influenced the across-tests correlation structure (nor associations with other variables in the data that we examined).

Due to extent, details concerning the specification and evaluation of the measurement model used for the following growth curve modeling, as well as formal testing of measurement invariance across both birth cohorts and ages, are presented as online supplementary material.

Growth curve models

We fitted an unconditional multiple group (i.e., across cohorts) second-order latent growth curve model to the data, using the measurement model (described in online supplementary material) as the first-order factor structure in order to identify a latent factor of cognition at ages 70, 75, 79, and 85. Estimates from this model (see Table 3) demonstrate a substantial cohort difference in average level of performance on the latent trait (as conditioned at age 79) in favor of the 1930 cohort. We can standardize this difference using the combined age 79 variability components (as derived from this model) across the cohorts (i.e., (15.87 – 11.34) / √((21.72 + 17.58)/2)), implying a standardized effect size of d = 1.02 ($\chi^2(1) = 271.61$, $p<.001$). This was in accordance with the Flynn effect (see e.g., Trahan et al., 2013). The variability component estimates from this model also imply larger individual differences in level of performance on the latent trait, at age 79, for the 1901-07 cohort (*SD*: √21.72 = 4.66) in comparison to the 1930 cohort (*SD*: √17.58 = 4.19; $\Delta_{\text{Intercept variance}} = 4.26$: $\chi^2(1) = 4.25$, $p = .039$), thereby implying less heterogeneity in level of performance among members of the later born cohort (see also factor SD by age in supplementary material Table S5).

We can interpret the slope estimates as the expected linear decline on the latent trait for each additional year passing between age 70 to 85. Estimates from the unconditional model (Table 3) indicate that the 1930 cohort showed a somewhat less steep rate of decline (-0.39) in comparison to the cohort born 1901-07 (-0.44;), this difference was however not significant ($\Delta_{\text{Slope mean}} = 0.05$: $\chi^2(1) = 2.73$, $p = .098$). We can standardize these estimates on the combined intercept variability components, implying (-0.44*15) a decline by d =1.49 of a standard deviation over the 15 years study period (i.e., from age 70 to 85) for the 1901-07 cohort, in comparison to (-0.39*15) d = 1.32 in the 1930 cohort. This implies that the difference in performance level on the latent trait increases by .17 of a standard deviation over the 15 years period as a function of cohort differences in rate of change. The estimate from this model also implies a slightly larger individual difference in rate of change for the 1901-07 cohort (*SD*: √0.083 = 0.29) in comparison to the 1930 cohort (*SD*: √0.033 =0.18; $\Delta_{\text{Slope variance}}$ = 0.05; ($\chi^2(1) = 2.54$, $p = .111$). That implies less heterogeneity in cognitive aging trajectories in the later born cohort.

Next, we fitted a conditional multiple group second-order latent growth curve model to the data. A path diagram representing this model is shown in Figure 1 and the estimated parameters can be found under Model 1 in Table 4. Inclusion of the time-constant covariates (sex, education, smoker, marital status, diabetes, hypertension, and stroke) accounted for 10% of the between-person variance at age 79 for cohort 1901-07 and 14% for cohort 1930. These covariates accounted for 17% of the between-person variance in rate of change for cohort 1901-07 and 23% for cohort 1930. Neither sex, smoking, hypertension, or the between-person factor of stroke were significantly associated with the age 79 trait level. However, more than a formal education was associated with higher levels and this effect size was similar across cohorts (1901-07: $d = .66$; 1930: $d = .52$; $\Delta_{\text{Education on intercept}}$ = 0.61; ($\chi^2(1)$ = 1.67, $p = .20$). Being married or cohabiting was also associated with higher cognitive ability in the 1930 cohort ($d = .23$) but not in the 1901-07 cohort ($d = -.00$; $\Delta_{\text{Marital on intercept}}$ = 1.04; ($\chi^2(1) = 4.69$, $p = .030$). Diabetes was associated with a lower level, in a similar manner, in both cohorts (1901-07: $d = -.32$; 1930: $d = -.29$; $\Delta_{\text{Diabetes on intercept}}$ = 0.16; ($\chi^2(1) = 0.02$, p = .89).

Neither sex, smoking, marital status, diabetes, hypertension or the between-person factor of stroke were significantly associated with rate of change. Having more than a formal education was however associated with a steeper rate of decline in a similar manner in both cohorts ($\Delta_{\text{Education on slope}}$ = -0.03; ($\chi^2(1) = 0.27$, $p = .61$). We can standardize these effects using the intercept variances from the unconditional model, implying a difference of .40 of a standard deviation over the 15 years study period as due to having more than a formal education in cohort 1901-07, and .31 in cohort 1930.

The time varying effect of stroke on the cognitive trait was medium to large and significant in both cohorts (1901-07: $d = -.52$; 1930: $d = -.39$). However, the difference between cohorts in this effect was not significant ($\Delta_{\text{Stroke on cognition}} = -0.55$; $\chi^2(1) = 0.30$, $p = .58$). Inserting equality constraint across cohorts revealed a combined estimate of -2.00 (*SE*=0.50), implying a drop by .45 of a standard deviation on the latent trait on measurements subsequent to stroke incidence. Estimating a separate stroke effect at each age per birth cohort did improve the model fit ($\chi^2(6) = 13.50$, $p = .04$). This implied an age difference in the stroke impact that differed across birth cohorts. Further evaluation of these effect sizes revealed that the main difference (in both cohorts) occurred between ages 75 and 79. We therefore constrained the age 70 and 75 stroke effects equal, as well as the age 79 and 85 stroke effects, in both cohorts. This improved the fit ($\chi^2(2) = 7.84$, $p = .02$; see Model 2 in Table 4), in comparison to the model with the age equality constraints. Additionally, realizing the age 70 by 75 and the age 79 by 85 equality constraints did not improve the fit ($\chi^2(4) = 6.25$, $p = .18$). For the earlier born cohort (1901-07), was the stroke impact size at ages 70 and 75 equal to -1.84 (*SE*=0.79; $d = .42$), but substantially higher at ages 79 and 85, or -3.08 (*SE*=1.03; $d = .70$). However, for the later born cohort (1930), was the age 70 and 75 effect equal to -2.34 (*SE*=0.66; $d = .53$), but substantially lower at ages 79 and 85, or -0.74 (*SE*=0.77; $d = .17$). This can be viewed as evidence of birth cohort difference in the age trend of the stroke impact, implying that the effect increases as a function of age in earlier born cohorts but decreases as a function of age in later birth cohorts.

We further added the lagged effects of the stroke impact to the final model (i.e., Model 2 in Table 4), by regressing the first-order cognitive factor at age 75, 79, and 85, on the time-varying stroke variable at ages 70, 75, and 79, respectively. These lagged stroke effects were all allowed to vary across birth cohorts. Inclusion of such lagged effects did however not improve the model fit ($\chi^2(6) = 8.85$, p=.18) with differences in AIC (2.06) and BIC (35.88) favoring the simpler model. Neither did it improve the fit when we constrained the lagged effects equal across ages within cohort ($\chi^2(2) = 0.71$, $p = .70$; ΔAIC = 3.09; ΔBIC = 14.36). We therefore found no evidence of lagged stroke effects.

Estimates as derived from Model 2 in Table 4 are plotted in Figure 2. The plot shows change trajectories for six prototypical individuals belonging to either birth cohort 1901-07 (red lines) or 1930 (black lines). Solid lines refer to non-stroke cases, dashed lines to cases that suffered a stroke at age 72, and long dashed lines refer to cases that suffered a stroke at age 82. The inserted *d* values refer to standardized time-varying (within-person) impact of stroke as conditioned on the birth cohort and age of the incidence. This effect size increased with age in the 1901-07 cohort but decreased with age in the 1930 cohort. The difference between solid red and black lines refers to the birth cohort differences (i.e., the Flynn effect), which was noteworthy substantial ($d \approx .92$), and always relatively larger than the stroke effects.

Discussion

In this study, we compared the impact of stroke on cognitive aging development from age 70 to 85 across two birth cohorts, drawn from the same Swedish city general population and born up to 30 years apart. We first estimated the stroke impact, as unconditioned on birth cohort and chronological age of the patient at the time of the stroke incidence. As expected, and in accordance with previous studies (e.g., Comijs et al., 2009; Levin et al., 2015; Rajan et al., 2014; Zheng et al., 2019), we found a moderate to large stroke effect (i.e., .45 of a SD). Notably, this effect was quite similar across birth cohorts (i.e., .52 in cohort 1901-07 and .39 in cohort 1930), which was unexpected as we anticipated a substantially smaller effect in the later born cohort. However, when conditioning the stroke effect on the age of the patients at the time of the incidence, we found an increased impact across age in the 1901-07 cohort but a decrease in the 1930 cohort. This was only partly in agreement with expectations, as we anticipated that the impact would increase with age in both cohorts.

Given that the observed patterns of findings were only partly in agreement with our expectations, explanations are somewhat speculative. We note that the unconditioned stroke effect was indeed smaller in the later born cohort, although not significantly so. We anticipated a smaller stroke effect in this cohort due to a variety of favorable contextual factors, such as better post-stroke rehabilitation, improved medication and adherence to public health care recommendations, as well as healthier lifestyle norms, producing an overall better health and greater cognitive reserve in the later born cohort. Interestingly, the hypothesis was only supported in the older ages (≥79), where the differences in the stroke impact were substantially larger, and significantly so, in the 1901-07 cohort as compared with the 1930 cohort. A potential explanation for this interaction (i.e., stroke by age by cohort) may relate to increased prevalence of comorbidities (e.g., hypertension, hyperlipidemia, diabetes, and heart diseases), with increasing age, which was less efficiently treated between 1971 and 1986 (for the 1901-07 cohort) in comparison to between 2000 and 2015 (for the 1930 cohort), thereby negatively influencing the post-stroke recovery potentials for the 1901-07 cohort at the higher ages.

The explanation for a reduction in the stroke effects across age in the 1930 cohort is more uncertain but may relate to cohort and age differences in survival rate potentials for patients suffering extensive major stroke (which, given other things equal, leads to larger stroke impact on cognition). Such a survival rate was certainly higher in the later born cohort (Waziry et al., 2020; due to better medical treatment) and at younger ages (Aked et al., 2021; due to better overall health and a higher degree of brain plasticity). This may also be part of the explanation for a slightly larger (although the cohort difference was non-significant) stroke effect at younger ages in the 1930 cohort. Unfortunately, we lack data on the severity and location of the stroke incidence which prevents us from further analyses related to this explanation. However, the drop in the time-varying (i.e., within-person) stroke effect in the 1930 cohort should also be evaluated in the context of the time-constant (i.e., between-person) stroke effects. As shown in Figure 2, those individuals that suffered a stroke at age 82 in the 1930 cohort (black long dashed line) had a substantially lower estimate (d = -.28) on the cognitive trait at the time point prior to the stroke incidence, that is, in comparison to the non-cases (black solid line) at the same age. This implies a standardized total stroke effect (i.e., between-person + within-person effect) of d = .45 (at age 82) and therefore only a small reduction in the total stroke effect in the 1930 cohort across age. We therefore need to interpret the three-way interaction in the context of the cohort by age difference in the weights of the between- and within-person stroke effects. Stroke patients in the 1930 cohort deviated, in terms of cognition prior to the stroke incidence, from the general population to a larger degree than the 1901-07 cohort stroke patients, and this divergency increased with age.

As is evident by comparing the solid black and red lines in Figure 2, there was a large cohort difference in level of cognitive performance across all ages ($d \approx$ .92; for details see Thorvaldsson et al., 2017). In fact, this cohort difference (i.e., the Flynn effect) was a factor of two times larger than the unconditioned time varying stroke effect (d = .42). Such large cohort effects can have ramifications for the practical consequences of the stroke impact. For example, assuming a constant normal distribution for the between-person variance of the cognitive factor across cohorts, time, and non-stroke by stroke groups, as well as defining a threshold of dependency equal to -1.5 SD (arbitrary value) and using the reported estimates from Model 1 in Table 4. Our prediction would imply that 4% of the non-cases and 10% of the stroke cases in the 1930 cohort would fall below such a threshold, while a comparable prediction for the 1901-07 cohort would be that 20% of the non-cases but 42% of the stroke cases would fall below such a threshold. This underlies the importance of considering the Flynn effect when interpreting the potential consequences of stroke for cognitive aging.

In addition to information about the stroke impact on cognition, there are several other inferences that can be derived from our analysis. As example, the models confirm a large Flynn effect, in which a later born cohort outperformed an earlier born, with effect size (1.02/3=.34 standardized point difference across a decade) which is in line with estimates from meta-analyses (Pietschnig & Voracek, 2015; Trahan et al., 2014). Noteworthy, our findings further demonstrate equal rate of age-related cognitive decline across the two examined birth cohorts (BF_{01} = 11. 60)[2]. This is not in agreement with previous reports from the H70 study (c.f., Karlsson et al., 2015; Thorvaldsson et al., 2017) using data from age 70 to 79, in which we found steeper rate of age-related decline in the later born cohort. Adding data from the age 85 cognitive measurements in fact alters this previous inference, which is now in line with reports from several other studies (e.g., Finkel et al., 2007; Gerstrof et al., 2023; Zeliniski & Kennison, 2007), but not with other findings (e.g., Dodge et al., 2015, Gerstrof et al., 2011). Also, the second-order factor variance components were smaller in the 1930 cohort, implying less between-person heterogeneity in cognitive performance and rate of change for later born cohorts. A speculative explanation for this relates to cohort difference in the onset of age-related brain pathology accumulation, which probably and on average, may occur at higher ages in the later born cohorts.

Having more than a formal education was associated with higher cognitive ability and less steep rate of age-associated decline, in both cohorts. This is only partly in agreement with previous studies (e.g., Foverskov et al., 2018; Wilson et al., 2019), as most report association of more education with elevated level, but not rate of decline, in old age global cognition (for review see Seblova et al., 2020). Being married or cohabiting was also associated with higher performance in the 1930 cohort, but not in the 1901-07 cohort. This difference in the effect sizes was in fact significant and indicates that close social relations may be of more importance for cognitive aging in today's societies than have been previously. This does though need be evaluated in other studies. Diabetes was associated with lower cognition but not rate of change in both cohorts, which is in line with some previous studies (e.g., Papunen et al., 2020) but not others (e.g., Hassing et al., 2004).

The design of the H70 study, with members of different birth cohorts identified and systematically drawn from the same city population, at the same age, with subsequent follow-ups at the same ages, using the same cognitive outcomes, allows for birth cohort comparisons in level of performance, rate of change, as well as in their correlates. Analyses of cohort differences in the effects of explanatory variables on level and rate of cognitive change entail modeling of higher order interactions, which require substantial data to be precise. In that respect, does the SEM framework facilitate the analyses by allowing latent trait modeling with higher precision and statistical power. The analytical approach we used allowed for formal testing and evaluations of measurement invariance across cohort and age, thereby verifying assumptions concerning associations between the latent trait and observed behavioral manifestations. These assumptions generally remain untested in analyses based only on manifested variables.

The limitations of the presented study design include few repeated measurements and relatively extended time periods between measurement occasions (i.e., 4-5 years) rendering it impossible to model and separate short-term fluctuations associated with a stroke event from long-term effects. These are to some degree confounded in the presented analyses. Also, birth cohort differences in diagnostic precision and survival rate must be interpreted as part of the contextual effects evaluated in the presented analyses and not controlled for in this study. Both these factors are likely to be higher in the 1930 cohort. Self-report of stroke incidence and lack of detailed clinical ascertainment was another limitation, although additional information from informant and medical records was used when available.

In conclusion, our study provides strong evidence for a substantial impact of stroke on later cognitive development between the ages of 70 to 85. Our study, however, suggests that the generalizability of such effects may vary across cohort and age. The stroke impact increased with age in an earlier born cohort, while it decreased with age in a later born cohort. Notably, the Flynn effect was large (a factor of two in comparison to that of the stroke effect) emphasizing that the clinical consequences of a stroke need to be evaluated in the context of cohort differences.

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Footnotes

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[2]Bayes factor approximation was derived from the equations presented on p. 796 in Wagenmakers (2007; see also Raftery 1999) using the BIC statistics.

Table 1. Descriptives for the H70 study sample (N=2074)

Variables	Cohort 1901-07 (n=1155)	Cohort 1930 (n=919)
Females (%)	707 (61)	534 (58)
More than formal education (%)	392 (35)	444 (50)
Marital status (% married)	550 (48)	521 (57)
Smoker (%)		
Non-smoker	687 (60)	420 (46)
Previous smoker	239 (21)	377 (41)
Current smoker	215 (19)	117 (13)
Diabetes (%)	72 (6)	85 (9)
Hypertension (%)	378 (34)	516 (56)
Stroke[a] (%)		
Age 70	24 (3)	46 (5)
Age 75	19 (2)	36 (4)
Age 79	28 (4)	49 (5)
Age 85	47 (4)	47 (5)

Notes. There were 71 participants with missing data on the education variable, 19 on the smoker variable, 8 on the marital status variable, 19 on the diabetes variable, and 26 on the hypertension variable. [a]Number of new, and first time, stroke incidences per age of measurement. Percentages were derived from the total sample per cohort, except the age 85 sample addition to cohort 1901/02 was omitted for the computation at ages 70, 75, and 79.

Table 2. Descriptives and correlation matrices for the cognitive outcomes stratified by birth cohort 1901-07\1930

	Variables	M	SD	N	1	2	3	4	5	6	7	8	9	10	11	12
1	Spatial age 70	14.76\19.47	7.18\6.99	575\235	1.00	**.57**	**.58**	.75	.56	.60	.72	.42	.47	.71	.56	.42
2	Speed age 70	18.19\25.62	7.68\7.94	572\233	**.61**	1.00	**.52**	.56	.58	.47	.49	.62	.42	.52	.63	.46
3	Reasoning age 70	13.32\16.72	4.71\4.64	748\231	**.52**	**.48**	1.00	.56	.40	.57	.56	.38	.52	.51	.47	.41
4	Spatial age 75	13.24\16.55	6.98\7.06	555\605	.79	.59	.49	1.00	**.48**	**.50**	.74	.42	.41	.57	.48	.35
5	Speed age 75	16.72\23.18	7.17\5.15	544\707	.60	.70	.46	**.67**	1.00	**.46**	.32	.39	.34	.36	.41	.39
6	Reasoning age 75	12.38\14.14	5.32\5.31	284\684	.54	.43	.37	**.60**	**.56**	1.00	.43	.27	.48	.41	.34	.40
7	Spatial age 79	11.25\15.62	7.51\6.47	416\476	.76	.49	.40	.77	.62	.54	1.00	**.45**	**.49**	.63	.49	.34
8	Speed age 79	14.45\21.32	6.47\7.43	398\561	.57	.54	.38	.61	.72	.58	**.71**	1.00	**.36**	.37	.59	.31
9	Reasoning age 79	12.00\14.10	5.33\5.16	181\487	.40	.35	.20	.51	.59	.61	**.58**	**.66**	1.00	.37	.39	.40
10	Spatial age 85	7.75\15.04	6.98\6.80	474\343	.60	.21	.15	.70	.57	.56	.76	.65	.45	1.00	**.56**	**.50**
11	Speed age 85	9.70\17.19	7.11\7.24	439\371	.36	.29	.09	.55	.56	.43	.53	.66	.41	**.73**	1.00	**.52**
12	Reasoning age 85	10.46\13.22	9.33\5.42	401\347	.23	.19	.19	.51	.62	.58	.55	.68	.49	**.67**	**.74**	1.00

Notes. Input data for cohort 1901-07 is below the diagonal and for cohort 1930 above the diagonal. We used pairwise deletion when deriving these matrices. All models were however fitted to the complete data matrix using robust full information maximum likelihood.

Table 3. Estimated parameters from an unconditional multiple group second-order latent growth curve model fitted to the H70 data (N=2074)

Birth cohort	1901-07 (n=1155)		1930 (n=919)	
Parameters	Est.	SE	Est.	SE
Intercept (age 79)	11.34***	0.17	15.87***	0.18
Age slope	-0.44***	0.02	-0.39***	0.02
Variances				
Intercept (age 79)	21.72		17.58	
Age slope	0.08		0.03	
Covariance I&S (cor.)	0.30(.22)		-0.18 (-.24)	
Fit measures				
χ^2(df)	364.68(93)			
χ^2 by cohort	155.20		209.49	
CFI	0.949			
RMSEA 90% CI	0.053 [0.047, 0.059]			
SRMR	0.095			

Table 4. Estimated parameters from two conditional multiple-group second-order latent growth curve models fitted to the H70 data ($n_{cohort\ 1901\text{-}07}$ = 1155; $n_{cohort\ 1930}$ = 919)

	Model 1				Model 2			
Birth cohort	1901-07		1930		1901-07		1930	
Parameters	Est.	SE	Est.	SE	Est.	SE	Est.	SE
Intercept (age 79)	10.67***	0.48	14.76***	0.47	10.67***	0.48	14.77***	0.47
Sex	0.15	0.41	-0.17	0.36	0.15	0.41	-0.18	0.36
Education	2.90***	0.34	2.29***	0.33	2.91***	0.34	2.30***	0.33
Smoker	0.08	0.24	-0.07	0.25	0.08	0.24	-0.08	0.25
Marital status	-0.01	0.34	1.03**	0.34	-0.02	0.34	1.01**	0.33
Diabetes	-1.43*	0.67	-1.30*	0.60	-1.43*	0.67	-1.34*	0.60
Hypertension	-0.28	0.33	-0.10	0.33	-0.29	0.33	-0.11	0.33
Stroke (time constant)	-0.44	0.79	-0.46	0.65	-0.09	0.87	-0.92	0.65
Age slope	-0.37***	0.05	-0.37***	0.06	-0.37***	0.05	-0.37***	0.06
Sex	0.01	0.04	0.11**	0.04	0.01	0.04	0.11**	0.04
Education	-0.12***	0.04	-0.09*	0.04	-0.12***	0.04	-0.09*	0.04
Smoker	0.02	0.03	0.03	0.03	0.02	0.03	0.03	0.03
Marital status	-0.03	0.04	-0.02	0.04	-0.03	0.04	-0.02	0.04
Diabetes	-0.07	0.08	-0.07	0.06	-0.07	0.08	-0.07	0.06
Hypertension	-0.01	0.04	-0.06	0.04	-0.01	0.04	-0.06	0.04
Stroke (time constant)	-0.10	0.07	0.01	0.06	-0.03	0.07	-0.11	0.07
Stroke (time varying)	-2.30**	0.78	-1.75**	0.64				
Age <79[a]					-1.84*	0.79	-2.38***	0.66
Age ≥79					-3.08**	1.03	-0.74	0.77
Variances								
Intercept (age 79)	19.65		15.10		19.65		15.00	
Age slope	0.07		0.03		0.07		0.03	
Covariance I&S (cor.)	0.29 (.25)		-0.09 (-.14)		0.29 (.25)		-0.08 (-.13)	
Fit measures								

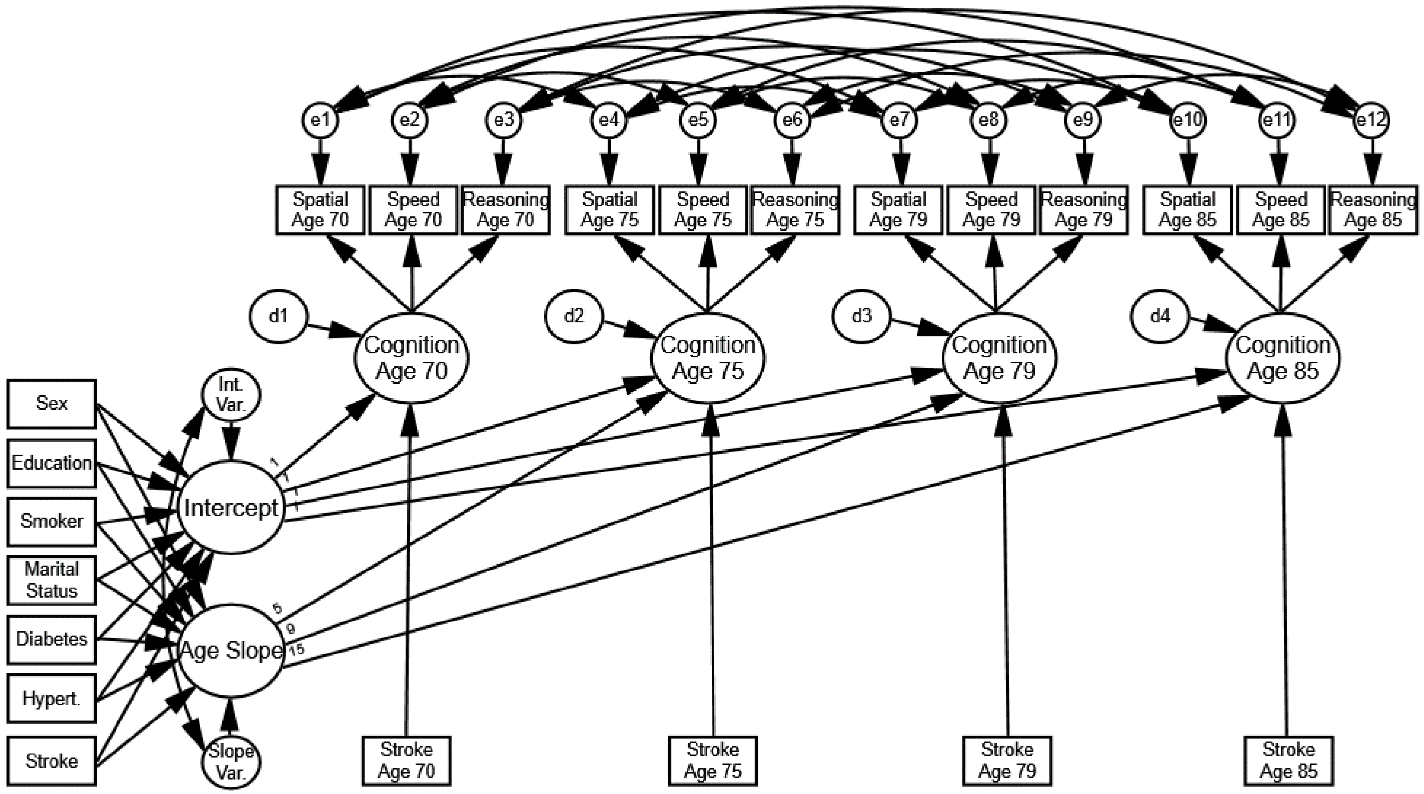
χ^2(df)	769.63(327)		761.29 (325)	
χ^2 by cohort	336.21	433.41	333.21	428.08
CFI	.953		.951	
RMSEA 90% CI	0.034 [0.031, 0.037]		0.036 [0.032, 0.040]	
SRMR	0.060		0.060	

Notes. [a]The time varying stroke effects were constraint equal across ages less than 79 and across ages 79 and more.

Figure Caption

Figure 1. A path diagram of a second-order latent growth curve model fitted to the H70 data. The outcome latent cognitive trait was defined in both cohorts by measures of spatial ability, reasoning ability, and perceptual-motor-speed at ages 70, 75, 79, and 85.

Figure 2. Expected change trajectories, as derived from the final model, demonstrating how the stroke impact differs by birth cohort and age. The *d* values reflect the standardized time varying stroke effects.


e1
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e12
Spatial Age 70
Speed Age 70
Reasoning Age 70
Spatial Age 75
Speed Age 75
Reasoning Age 75
Spatial Age 79
Speed Age 79
Reasoning Age 79
Spatial Age 85
Speed Age 85
Reasoning Age 85
d1
d2
d3
d4
Cognition Age 70
Cognition Age 75
Cognition Age 79
Cognition Age 85
Sex
Education
Smoker
Marital Status
Diabetes
Hypert.
Stroke
Int. Var.
Intercept
Age Slope
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1
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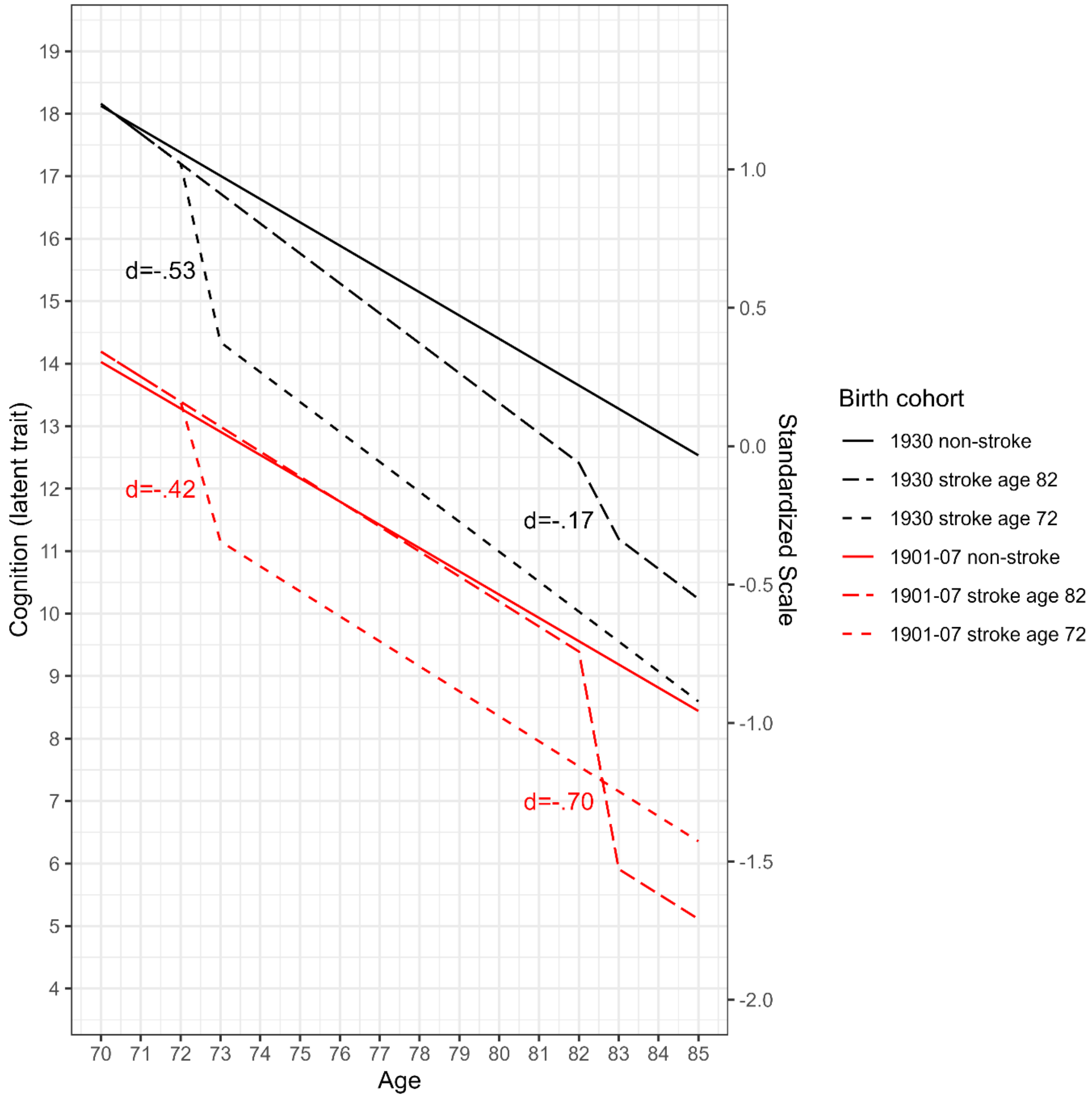
Stroke Age 70
Stroke Age 75
Stroke Age 79
Stroke Age 85

Figure 1



19
18
17
16
15
14
13
12
11
10
9
8
7
6
5
4
Cognition (latent trait)
1.0
0.5
0.0
-0.5
-1.0
-1.5
-2.0
Standardized Scale
70
71
72
73
74
75
76
77
78
79
80
81
82
83
84
85
Age
d=-.53
d=-.42
d=-.17
d=-.70
Birth cohort
1930 non-stroke
1930 stroke age 82
1930 stroke age 72
1901-07 non-stroke
1901-07 stroke age 82
1901-07 stroke age 72

Figure 2.