Delayed onset of cognitive terminal decline in later born cohorts: Evidence from a longitudinal study of two cohorts born 29-years apart

Valgeir Thorvaldsson[1,2], Ingmar Skoog[2,3], & Boo Johansson[1,2]

[1]Department of Psychology, University of Gothenburg

[2]Center for Ageing and Health, University of Gothenburg

[3]Department of Psychiatry and Neurochemistry, Institute of Neuroscience of Physiology, Sahlgrenska Academy, University of Gothenburg

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Corresponding author: Valgeir Thorvaldsson, Department of Psychology, Center for Ageing and Health, University of Gothenburg, Box 500, SE-405 30, Gothenburg, Sweden. E-mail: Valgeir.Thorvaldsson@psy.gu.se

Abstract

In this study we evaluated birth cohort (i.e., generational) differences in the onset and rate of acceleration in cognitive decline prior to death (i.e., terminal decline; TD). We obtained data from two cohorts, born in 1901-02 (n=755, 64% females) and 1930 (n=347, 48% females), identified and sampled at age 70 from the same city population and assessed on the same cognitive tests at ages 70, 75, 79, 85, and 88. The 1901-02 cohort was additionally assessed at ages 90, 92, 95, 97, 99, and 100. The outcome was defined at each measurement occasion by a composite score of three cognitive tests assessing spatial ability, perceptual-and-motor-speed, and reasoning. Date of death was obtained from population register, with last update in April 2023, covering over 99% and 38% of the cohort members respectively. We fitted a random TD change point model to the data while accounting for sex and education. Findings revealed expected TD onset (as conditioned on male with formal education) 5.17 (95% HDI [2.54, 8.05]) years prior to death in the 1901-02 cohort, with an acceleration in rate of decline by a factor of 4.43 within the TD phase. This estimate was delayed by 2.53 (95% HDI [5.68, 0.10]) years in the 1930 cohort, with an acceleration by a factor of 5.16 within the TD phase. To the best of our knowledge, this is the first study to present evidence indicating that today´s birth cohort experience on average a shorter TD phase in comparison to earlier born cohorts.

Public significant statement: Older adults typically experience accelerated cognitive decline several years prior to death. This study provides new evidence that the onset of this acceleration is postponed in later born cohorts, indicating a shorter terminal decline period.

Keywords: aging, cognition, terminal decline, birth cohort differences, change point analysis

Terminal decline (TD) refers to acceleration of cognitive decline prior to death (for review see e.g., Bäckman & MacDonald, 2006; Cowl, 2016; Hülür et al., 2015). Such non-linear pattern of aging trajectories has been verified in many large-scale longitudinal cognitive aging studies (e.g., Batterham et al., 2011; Dodge et al., 2011; van den Hout et al., 2011; MacDonald et al., 2011; Muniz-Terrera et al., 2013; Sliwinski et al., 2006; Thorvaldsson et al., 2008; Wilson et al., 2003; 2007). Existing evidence indicates average TD onset between 5 to 10 years prior to death (Karr et al., 2018), but individual differences are substantial (Muniz-Terrera et al., 2013; Thorvaldsson, Skoog, et al., 2017), with some individuals showing onset more than a decade prior to death while others never displaying such TD onset. Higher education (Muniz-Terrera et al., 2014) and ability test scores (Thorvaldsson, Skoog, et al., 2017) have been associated with delayed TD onset, while presence of the ε4 allele of apolipoprotein E gene (Yu et al., 2013) and aggregation of postmortem Alzheimer´s disease pathology (Wilson et al., 2012) have been linked to early TD onset. In the present study we expand knowledge of TD moderators by evaluating potential birth cohort (i.e., generational) differences.

When measured at the same age, later born cohorts tend to outperform earlier born cohorts on most cognitive outcomes (e.g., Brailean et al., 2018; Munukka et al., 2021; Overton et al., 2018). This has been labelled the *Flynn effect* (for review see e.g., Trahan et al., 2014; Pietschnig & Voracek, 2015) and can be explained by reference to overall improvement in environmental circumstances and healthier lifestyles (e.g., better education, nutrition, and healthcare). Studies concerning cohort differences in rate of age-related change, however, show mixed findings, with some showing a shallower decline in later born cohorts (Dodge et al., 2014; Gerstorf et al., 2011; Grasset et al., 2018; Vonk et al., 2019) while others suggesting a steeper (Thorvaldsson, Karlsson, et al., 2017) or no cohort differences in cognitive decline (Finkel et al., 2007; Gerstorf et al., 2023).

Very few studies have hitherto evaluated cohort differences in the context of TD. Gerstorf and colleagues (2011) found a steeper average decline in later born cohorts when analyzing individual trajectories as a function of time to death. Hülür and colleagues (2013) took a different approach by defining *cohort* by date of death, instead of date of birth. Corroborating findings from Gerstorf et al., they found a steeper average decline among those that died at later dates. In the present study, we further evaluate birth cohort differences in TD using data from two age-homogenous population-based cohorts, born in 1901-02 and 1930, identified from the same city population, at the same age, and subsequently measured on the same cognitive outcomes from age 70 until death. Given better health and neuronal intactness, we expect delayed TD onset in the later born cohort. This is in line with the *compression of morbidity* hypothesis (Fries, 1980; 2005), suggesting a shorter period of time between the onset of morbidity-related functional decline and death in later born cohorts.

Method

Transparency and openness

The data used for these analyses can be made available to researchers meeting access criteria for deidentified data as defined by the H70 study steering board. Model code with commentaries can be found as online Supplementary material. The study was not formally preregistered.

Participants

Cohort 1901-02. In 1971, all individuals living in the city of Gothenburg, born between July 1st 1901 and June 30th 1902, on days ending with 2, 5, and 8 were identified from the Swedish Revenue Office Register and invited to participate in the Gerontological and Geriatric Population Study (H70). Baseline participation rate for this sample was 85% (n=1148). About 40% (n=460) of this sample was randomly selected for cognitive assessments. Participation rate for this sub-sample was 84% (n=387). Details concerning the

sample can be found in Rinder et al. (1975) and Svanborg (1977). In 1986, the remaining surviving population, already identified in 1971, was invited for study participation. Response rate for this sub-sample was 60% (n=370). Additional information about this add-on sample can be found in Thorvaldsson et al. (2006). Date of death was obtained from the Swedish Population Register (Statistics Sweden) for more than 99% (n=755) of the sample (see Table 1).

Cohort 1930. In 2000, all individuals, living in the city of Gothenburg, born in 1930 on days ending with 3, 6, 12, 18, 21, 24, and 30 were identified from the Revenue Office Register and invited for participation in the study. Participation rate for this sample was 66% (n=783). About 20% (n=241) of this sample was randomly selected for cognitive testing at age 70, but all survivors of the sample were invited for subsequent cognitive testing. This sample was further extended in 2005, with the inclusion of all individuals in the population born on days ending with 2, 3, 5, 6, 11, 12, 16, 18, 20, 21, 24, 27, and 30. Response rate on this occasion was 63% (n=969) and 925 individuals contributed with cognitive test data. Date of death was obtained for 38% (n=347) of this sample, with last update in April 2023. Further information about this sample can be found in Wiberg et al. (2013). The H70 study data collections were approved by the ethical review board at the University of Gothenburg.

Cognitive measures

We used the following cognitive test measures to derive a global cognition composite score at each measurement occasion. Both cohorts were measured at ages 70, 75, 79, 85, and 88, and the 1901-02 cohort was further measured at ages 90, 92, 95, 97, 99, and 100.

Block Design is a measure of spatial ability. Participants were given colored blocks and asked to construct replicas of prototypes presented in two colors. Seven prototypes were presented with increasing difficulty and performance was scored as conditioned on how fast

the participants correctly replicated the prototypes. Incomplete replication was scored zero. Maximum score was 42 and time limit 20 minutes.

Figure Identification is a measure of perceptual and motor speed. Participants were asked to match, as quickly as possible, a target figure with one identical figure placed in line among four other figures. Total score was calculated as sum of correct items – (sum of wrong items / 4), as to adjust for wrong answers and guessing. Maximum score was 60 and time limit 4 minutes.

Figure Logic is a nonverbal measure of inductive reasoning. Participants were presented with geometrical figures, organized in rows of five figures per row, and asked to identify the target figure that differed from the other four figures. Total raw score was calculated as sum of correct items – (sum of wrong items/4). Items were not presented in order of difficulty, and participants were encouraged not to dwell too long on an item if they encountered difficulties. Maximum score was 30 and time limit 8 minutes.

Details concerning these tests and their applications in the H70 study can be found elsewhere (Berg, 1980; Dureman et al., 1971; Thorvaldsson, et al., 2006; Thorvaldsson, Karlsson et al., 2017). At each measurement occasion, we derived the global cognition composite score by summing over the z-score transformed raw test scores and dividing by the number of contributed test scores for the respective occasion. We used the baseline (i.e., age 70) distributions of the respective test for the transformation. Before transforming, we omitted subsequent zero-values test scores (as due to severe compromised cognition). There were 417 participants who contributed data on 1 occasion; 271 contributed data on 2 occasions; 224 on 3 occasions; 110 on 4 occasions; 39 on 5 occasions; 25 on 6 occasions; 11 on 7 occasions; 4 on 8 occasions; and 1 on 11 occasions.

Data from the Block Design test was available at all measurements occasions, while the Figure Identification test was omitted at age 100 and the Figure Logic test was omitted at

ages 97, 99, and 100. 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 was randomly selected to take the cognitive test battery. The cognitive composite computation was derived from the available data at each measurement occasion.

Covariates

Biological sex was coded 0 for males and 1 for females. Given alternations in the education system across the birth cohorts, we simplified the education variable by dichotomizing it; coding 0 for only formal education or less and 1 for more than a formal education. Compulsory formal education ("*folkskola*") was 6 years in the 1901-02 cohort and 7 years in the 1930 cohort.

Statistical analysis

We fitted a random change point model to the data using Bayesian estimation in the form of Markov Chain Monte Carlo Gibbs sampling, as implemented in JAGS (Plummer, 2003). This is essentially a broken-stick (i.e., a two slope spline) model with random components reflecting individual differences in the change point (i.e., the knot), rate of linear change prior to and after the change point, as well as individual differences in level of performance. We can write the level 1 part of the model as:

$$Cognition_{ij} = \begin{cases} \alpha_j + \beta_{1j}\left(TTD_{ij} - \beta_{3j}\right) + \varepsilon_{ij}, \ if\ TTD_{ij} \leq \beta_{3j} \\ \alpha_j + \beta_{2j}\left(TTD_{ij} - \beta_{3j}\right) + \varepsilon_{ij}, \ if\ TTD_{ij} > \beta_{3j} \end{cases},$$

where cognition is the outcome variable at time $i = 1\ldots, n_j$, within person $j = 1\ldots, N$, and time is structured as *time to death* (i.e., TTD_{ij}) in years, defined as *age at measurement - age at death.* The α_j is the person specific intercept, interpreted as the expected value on the cognition variable at the time of the person specific change point (i.e., β_{3j}). The β_{1j} and β_{2j} refer to person specific estimate of rate of linear change prior to and after the person specific change point, respectively. These parameters then become outcomes at level 2, such that:

$$\alpha_j = \gamma_{00} + \gamma_{01}Cohort_j + \gamma_{02}Sex_j + \gamma_{03}Education_j + U_{0j}$$
$$\beta_{1j} = \gamma_{10} + \gamma_{11}Cohort_j + \gamma_{12}Sex_j + \gamma_{13}Education_j + U_{1j}$$
$$\beta_{2j} = \gamma_{20} + \gamma_{21}Cohort_j + \gamma_{22}Sex_j + \gamma_{23}Education_j + U_{2j}$$
$$\beta_{3j} = \gamma_{30} + \gamma_{31}Cohort_j + \gamma_{32}Sex_j + \gamma_{33}Education_j + U_{3j},$$

where γ_{00}, γ_{10}, γ_{20}, and γ_{30} are the level 2 intercepts, and γ_{01}, γ_{11}, γ_{21}, and γ_{31} are the moderating effects of birth cohort for the respective level 1 parameter. The γ_{02}, γ_{12}, γ_{22}, and γ_{32} refer to the moderating effect of sex and γ_{03}, γ_{13}, γ_{23}, and γ_{33} to the moderating effect of education. The ε_{ij} and $U_{.j}$ are the level 1 and level 2 residuals, respectively. Review of similar models can be found in Muniz-Terrera and Hall (2016), and examples and further details in Dominicus et al. (2008); Hall et al. (2003); Muniz-Terrera et al., (2011); and Thorvaldsson, Skoog, et al. (2017).

We modeled the data using a normal prior with mean derived from linear combination of the level 1 coefficients and precision using a uniform prior ranging from 0 to 100 raised to the power -2. We used normal priors with mean of 0 and a precision of 0.001 for all fixed effects, and a scaled inverse Wishart distribution with 5 degrees of freedom for random effects (Gelman & Hill, 2007). For each model, we simultaneously ran five chains, each with a burn-in of 360,000 iterations and 50,000 for posterior approximation, resulting in 250,000 iterations for inferences. We conducted convergence evaluations using trace plots and marginal posterior density plots, as stratified by chain, as well as Gelman and Rubin (1992) diagnostic criteria with a cut-off of <1.2 (Brooks & Gelman, 1998). For point estimates, we used the means of the marginal posterior density distribution and for precision 95% highest density interval (HDI), defined as 0.025 and .975 quantiles. Missing data were handled via the estimation procedure under the assumption of missing at random as conventionally defined. Given unequal numbers of follow-ups across birth cohorts, we conducted a sensitivity analysis after omitting the age 90, 92, 95, 97, 99, and 100 occasion measurements.

Results

Sample descriptives are shown in Table 1. The cohort born in 1901-02 had higher proportion of females ($BF_{10} > 100$) and lower proportion of individuals with more than a formal education ($BF_{10} = 19.88$). When compared at same ages, the 1930 cohort had a higher mean value on the cognitive composite score, verifying the Flynn effect (for detailed reporting of the Flynn effect in the H70 see Thorvaldsson, Karlsson et al., 2017). The average age of death was 87.24 in the 1901-02 cohort and 85.08 in the 1930 cohort. This value was higher in the 1901-02 cohort given a longer follow-up period and because that a substantial proportion of the 1930 cohort were still alive at the time point of the analyses. Years to death at baseline (i.e., age 70) was 13.38 in cohort 1901-02 and 14.53 in cohort 1930. Number of data points contributing to estimates in the growth curve model are shown in Figure 1 as a function of years to death (with a binwidth of ½ year) by birth cohort, demonstrating substantially larger amount of data, and consequently higher estimate precision, for the 1901-02 cohort.

Estimates from a random TD change point model are shown in Table 2. Average TD onset was 5.17 years prior to death for the 1901-02 cohort (conditioned on male with formal education). This estimate was delayed by 2.53 years in the 1930 cohort. Neither being female nor having more than formal education significantly moderated the TD onset. Average linear decline after TD onset was 1.64 points per year for the 1901-02 cohort, implying an acceleration by a factor of 4.43 compared to linear rate of change prior to TD onset. This estimate was steeper for the 1930 cohort, indicating a decline by 2.53 points per year after TD onset and an acceleration by factor of 5.16 in comparison to decline prior to the TD onset. Being female did not moderate the post TD onset decline. However, those with more than formal education showed a steeper decline in comparison to those with only formal education. As expected, belonging to the 1930 cohort and having more than a formal

education was associated with higher cognitive performance level, but there were no significant sex differences. Raw score trajectories and expected values derived from the model are plotted in Figure 2, demonstrating later TD onset in the 1930 cohort, but also a steeper decline within the TD period.

Sensitivity analyses

Estimates from analyses after omitting data from age 90, 92, 95, 97, 99, and 100 are shown in Table S1, as Supplementary material, indicating similar pattern of findings. The average TD onset from this model was 6.66 (95% HDI [3.10, 10.98]) years prior to death in the 1901-02 cohort, with a delay of 3.67 (95% HDI [-6.87, -0.30]) years in the 1930 cohort. Cognitive decline after TD onset was also steeper in the 1930 cohort.

Discussion

In this study, we evaluated birth cohort differences in onset and rate of TD using data derived from two birth cohorts, born up to 29 years apart, and subsequently measured on the same cognitive outcomes from age 70 until death. Our findings support substantial cohort differences in TD. The onset was postponed by 2.5 years in the cohort born 1930 and there was a substantially larger acceleration in decline after TD onset among those individuals. Findings are in agreement with the compression of morbidity hypothesis (Fries, 1980, 2005), predicting shorter mortality-related deterioration phases in later born cohorts.

There are several causal pathways that may explain the observed findings. As a consequence of a generally healthier lifestyle, as example in terms better nutrition (Samuelsson et al., 2019), less smoking (Ahacic et al., 2008), and more frequent physical exercise (Vilhelmson et al., 2022), as well as improved and more aggressive treatments of cardiovascular risk factors (Zhong, et al., 2016; e.g., hypertension and hypercholesterolemia) we may expect, on average, a more healthy cerebral blood flow among members of the 1930

cohort. Such is central for nutrients and oxygen supply to the brain, which is crucial for intact cognitive functioning.

Also, higher proportion of the members of the 1930 cohort have operated during their life-course within the context of more cognitively demanding environments (e.g., in terms of education, occupation, leisure activities, and everyday life functioning), continuously being exposed to new knowledge and technologies. Such a lifetime exposure to cognitive stimulation may leads to increased gray matter volume (e.g., Draganski et al., 2004), improved white matter integrity (e.g., Scholz et al., 2009), larger synaptic density and dendritic complexity, as well as more flexible cognitive strategies, providing resilience against accumulation of age-related brain pathologies, that is contributing to greater *cognitive reserve* (Stern, 2009). Such explanation is also in line with findings of steeper decline within the TD phase among members of the 1930 cohort (and for those with more education). Presumably, the pathology accumulation was larger at the time point of TD onset for the members of this cohort, leading to a steeper cognitive decline after a relatively higher reserve threshold had been reached. Improved sleeping pattern in the 1930 cohort (Skoog et al., 2019), as mediated through the glymphatic system (Jessen et al., 2015), may also be part of the explanation.

To our knowledge, this is the first study reporting population-based estimates of cohort differences in TD onset. Two studies (i.e., Gerstorf et al., 2011; Hülür et al., 2013) have reported steeper decline in later born cohorts when analyzing data as a function of time-to-death. However, these studies did not provide a distinct TD onset estimate, but rather modeled the process as a continuous polynomial function, which may explain the revealed discrepancy (see e.g., discussion in Sliwinski et al., 2006). Our findings were partly in agreement with these findings, as we found a steeper decline in the 1930 cohort, but only

after the TD onset. Therefore, our findings suggest that cohort differences in rate of change is likely to vary as a function of time to death.

A main strength of our study was the age-homogeneity of our population-based samples, which were systematically drawn in a similar manner from the same city population, almost 30 years apart. Another strength was the availability of information of date of death from population register. An obvious limitation was right censoring (i.e., to April 2023) of date of death for the 1930 cohort. A significant TD by age of death by cohort interaction may jeopardize study conclusions (i.e., if TD will be significantly different among those that are still alive in the 1930 cohort). These findings should be attested when date of death is available for the entire 1930 cohort. We note though, that our sensitivity analyses, using same age of measurement occasions across birth cohorts, revealed similar pattern of findings, which should strengthen confidence in the presented evidence. Another limitation was the relatively long time-intervals between subsequent measurements, which may lead to bias and imprecision in change point estimates. However, given similar study design set-up, that should have affected estimates in both cohorts equally.

Our findings demonstrate generational differences in TD, with later born cohort showing delayed TD onset and steeper decline within the TD phase. This supports the compression of morbidity hypothesis (Fries, 1980, 2005). Future studies need to evaluate TD onset for other functional variables (e.g., mobility and sensory functioning) and more specific morbidity markers (e.g., those related to cardio-vascular and neurological health). Such findings may have significant implications for our general view of aging and functioning, as well as for future health care planning.

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Table 1. Descrptives for the analyzed H70 study sample

Variables	Cohort 1901-02 (n = 755)	Cohort 1930 (n = 347)
Females (%)	481 (64)	168 (48)
More than formal education (%)	247 (35)[a]	156 (45)
Cognitive composite M (SD)		
Age 70	46.50 (8.29) n=362	54.67 (8.46) n=183
Age 75	45.46 (8.11) n=287	51.23 (7.92) n=259
Age 79	44.22 (8.16) n=197	49.72 (7.78) n=191
Age 85	38.27 (8.14) n=475	43.81 (8.61) n=93
Age 88	39.56 (7.32) n=194	42.65 (8.42) n=31
Age 90	38.90 (8.03) n=131	
Age 92	40.22 (6.85) n=66	
Age 95	35.94 (6.52) n=36	
Age 97	34.84 (5.14) n=19	
Age 99	34.33 (5.72) n=8	
Age 100	38.70 (3.98) n=4	
Age of death M (SD)	87.24 (7.01)	85.08 (4.99)[b]
Years to death M (SD)		
Age 70	13.38 (7.43)	14.53 (5.02)
Age 75	10.38 (6.55)	10.14 (4.53)
Age 79	8.91 (5.29)	7.00 (3.47)
Age 85	5.56 (3.84)	3.32 (2.10)
Age 88	4.40 (3.14)	2.39 (1.22)
Age 90	3.68 (2.69)	
Age 92	2.99 (2.44)	
Age 95	2.59 (2.21)	
Age 97	2.09 (1.87)	
Age 99	1.57 (0.57)	
Age 100	2.22 (1.92)	

Notes. [a]There were 41 and 2 with missing data on the education variable in cohort 1901-02 and 1930, respectively. [b]Last date of death for cohort 1930 was in April 2023.

Table 2. Estimates from a random change point model evaluating differences in onset and rate of cognitive terminal decline across two cohorts born in 1901-02 (n=755) and 1930 (n=347)

Parameter	Estimates [a]	95%HDI[b]
Level of performance[c]	42.36	[39.67, 45.15]
Cohort 1930	4.82	[2.06, 7.25]
Female	-1.37	[-3.39, 0.76]
More than formal education	3.26	[1.11, 5.51]
TTD[d] prior to CP[e]	-0.37	[-0.59, -0.17]
TTD prior to CP x Cohort 1930	-0.08	[-0.22, 0.09]
TTD prior to CP x Female	0.07	[-0.08, 0.23]
TTD prior to CP x More than formal education	-0.07	[-0.21, 0.08]
CP	5.17	[2.54, 8.05]
CP x Cohort 1930	-2.53	[-5.68, -0.10]
CP x Female	1.16	[-1.04, 3.47]
CP x More than formal education	-1.23	[-3.78, 1.30]
TTD post CP	-1.64	[-2.22, -1.16]
TTD post CP x Cohort 1930	-0.68	[-1.14, -0.27]
TTD post CP x Female	-0.04	[-0.48, 0.32]
TTD post CP x More than formal education	-0.67	[-1.21, -0.21]
Variance components[f]		
Intercept	6.80	
TTD prior to CP	0.31	
CP	6.08	
TTD post CP	0.94	
Residual	3.97	

Notes. The outcome was a composite score of global cognition derived from measures of spatial ability, perceptual and motor speed, and reasoning. [a]Mean of the marginal posterior distribution. [b]95% highest density of the marginal posterior distribution. [c]Conditioned at time of the change point. [d]Time to death in years (defined as age of measurement – age of death). [e]Change point reflecting onset of terminal decline. [f]Covariance across random effects were estimated as part of the models but omitted from the table. All parameters had a Gelman and Rubin (1992) convergence diagnostic value below a cut-off of <1.2.

Figure caption

Figure 1. Histogram showing number of data points as a function of years to death (binwidth = ½ year) by birth cohort.

Figure 2. Raw score trajectories and fixed effect estimates from a random terminal decline change point model fitted to the H70 study data.

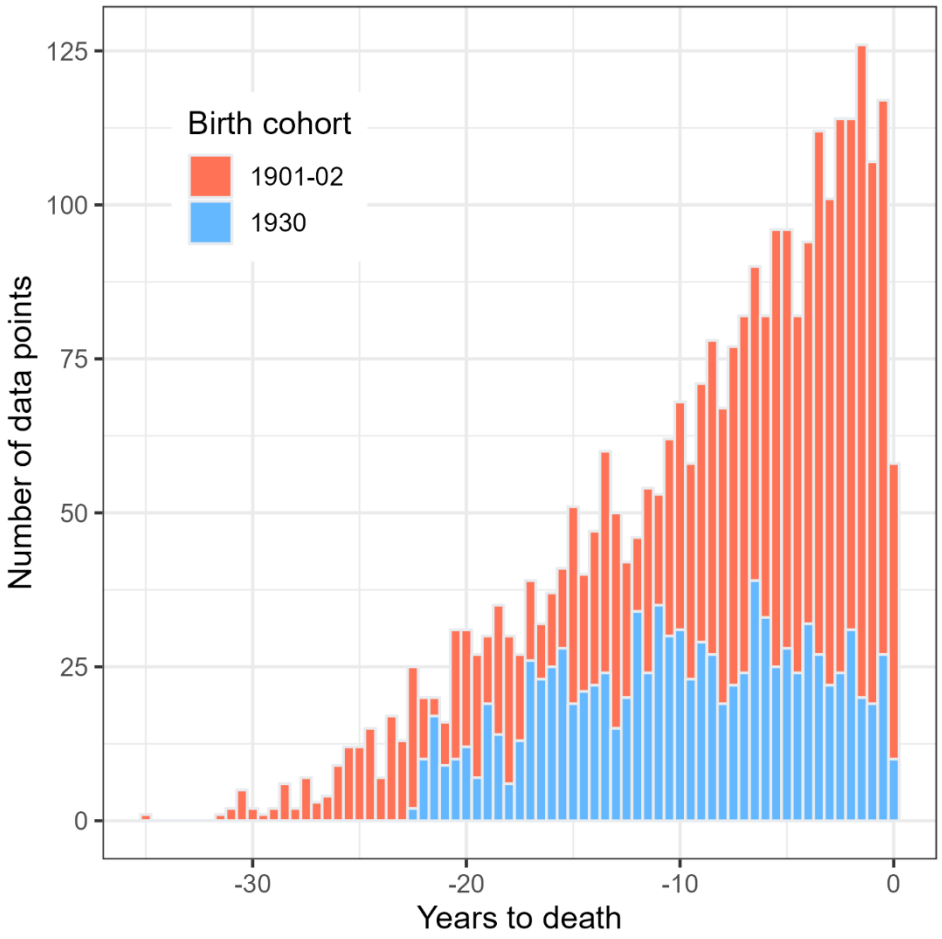
Birth cohort
1901-02
1930
Number of data points
125
100
75
50
25
0
-30
-20
-10
0
Years to death

Figure 1.

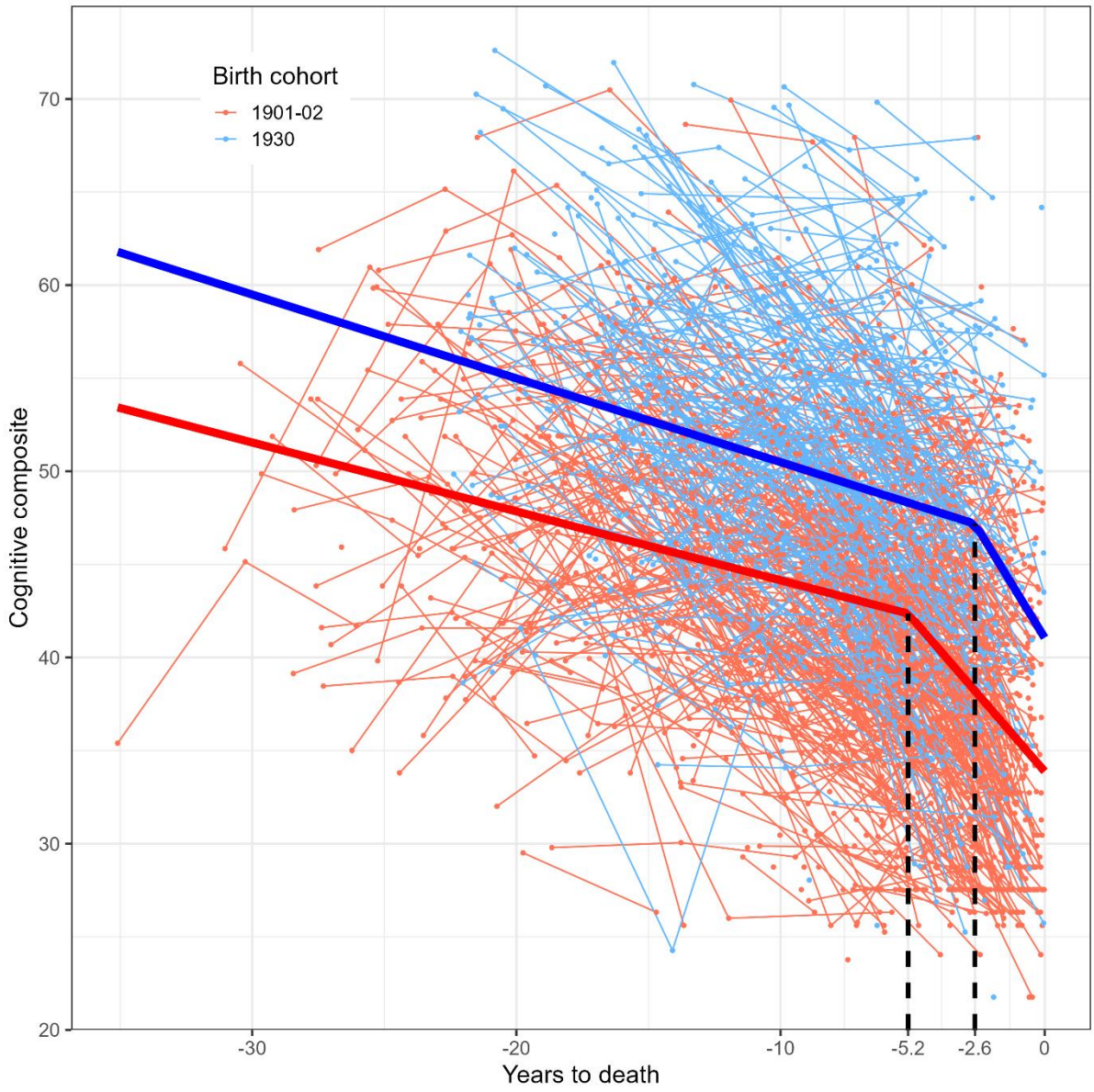
Birth cohort
1901-02
1930
Cognitive composite
70
60
50
40
30
20
-30
-20
-10
-5.2
-2.6
0
Years to death

Figure 2.