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Does postponing retirement affect cognitive function? A counterfactual experiment to disentangle life course risk factors

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ABSTRACT

Evidence suggests that contemporaneous labor force participation affects cognitive function; however, it is unclear whether it is employment itself or endogenous factors related to individuals' likelihood of employment that protects against cognitive decline. We exploit innovations in counterfactual causal inference to disentangle the effect of postponing retirement on later-life cognitive function from the effects of other life-course factors. With the U.S. Health and Retirement Study (1996–2014, n = 20,469), we use the parametric g-formula to estimate the effect of postponing retirement to age 67. We also study whether the benefit of postponing retirement is affected by gender, education, and/or occupation, and whether retirement affects cognitive function through depressive symptoms or comorbidities. We find that postponing retirement is protective against cognitive decline, accounting for other life-course factors (population: 0.34, 95% confidence interval (CI): 0.20,0.47; individual: 0.43, 95% CI: 0.26,0.60). The extent of the protective effect depends on subgroup, with the highest educated experiencing the greatest mitigation of cognitive decline (individual: 50%, 95% CI: 32%,71%). By using innovative models that better reflect the empirical reality of interconnected life-course processes, this work makes progress in understanding how retirement affects cognitive function.

Introduction

Concerns about how lengthening life expectancies will affect health care and pension systems have led the U.S. government, like other high-income countries, to postpone the statutory retirement age. There may be a fortuitous unintended consequence of postponed retirement. Evidence accumulates that cognitive engagement is associated with better cognitive function, which would imply that sustained participation in the labor force may be protective against cognitive decline (Adam et al., 2013; Bonsang et al., 2012; Kuiper et al., 2015; Meng et al., 2017; Mosca & Wright, 2018; Roberts et al., 2011; Rohwedder & Willis, 2010). However, because risk factors accumulate and interact over the life course to affect both cognitive function and age at retirement, identifying and quantifying the effect of retirement on cognitive decline has proved elusive. In fact, research often focuses exclusively on single characteristics, such as education or race/ethnicity, not accounting for how life-course factors are dynamically interconnected (Collins, 2015; Diez Roux, 2012). In this study, we approach retirement and cognitive function from the realistic perspective that they both come near the end of a long path beginning with one's social origins in race/ethnicity, gender, and early-life socioeconomic status (SES), through educational and occupational attainment and health behaviors, all the way up to more proximate factors such as partnership status and mental and physical health.

Estimating the effect of retirement on cognitive function is useful not only for understanding cognitive function for the current cohort of retirees or near retirees, but also to anticipate trajectories of cognitive decline for more recent cohorts. For members of cohorts born in or after 1960, the U.S. federal government increased the full statutory retirement to age 67. The statutory retirement age is important because, traditionally, most people retire at the statutory early or full retirement age (Behaghel & Blau, 2012; Rust & Phelan, 1997). Indeed, evidence suggests that statutory full retirement age (FRA) influences retirement behavior in many high-income countries. For example in the US, the establishment of Social Security is correlated with the first (and persistent until the 1980s) spike in age-65 retirement, and the postponement in FRA encoded in 1983 was associated with benefits claiming, self-reported retirement, and labor market participation (Behaghel &

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Blau, 2012). We predict that an unintended consequence of this policy change may be better cognitive function into older ages for these more recent cohorts above and beyond what secular shifts in increased educational attainment would predict. In other words, we hypothesize that postponed retirement will be protective against cognitive decline, accounting also for risk factors that vary within and across cohorts.

There are, however, some methodological challenges related to understanding life course risk factors for cognitive impairment that have prevented conclusive research on this topic. Biased estimates can be a result of 1:) dynamic two-way pathways among predictor and outcome, e.g., if retirement and cognitive function affect each other, 2) interactions between exposures and time-varying mediators, e.g., if retirement's effect on cognitive function differs in its impact through mental health, and 3) mediator-outcome confounding, e.g., if mental health affects cognitive function and retirement, and is itself affected by both.

The parametric g-formula offers a solution to these methodological issues. The g-formula is an innovative statistical approach that enables analysis of time-varying processes, while allowing for selection, reverse causality, and mediation (Bijlsma & Wilson, 2020; Vanderweele & Tchetgen Tchetgen, 2017; Wang & Arah, 2015). As such, the g-formula is a statistically flexible approach that allows us to examine the interdependent influences of life-course processes – such as education, partnership, health, and labor force participation – on later-life cognitive function, irrespective of the functional form that the relationships of mutual influence may take (De Stavola et al., 2015; VanderWeele et al., 2014). An additional benefit of our model is that through simulated interventions, it can also provide estimates of "population-averaged effects" (PAE), which can help us understand how policy changes – such as the postponement of statutory retirement age – may impact the wider population. This, of course, is important from a public health perspective.

With data from the Health and Retirement Study (HRS) (University of Michigan, 2017), we first estimate the effect of postponing retirement to age 67 on later-life cognitive function, accounting for time-variant and invariant sociodemographic, behavioral, and health risk factors. Second, we conduct moderation analyses to identify whether the effect of postponed retirement on cognitive function differs by gender, educational attainment, and/or occupational attainment. Third, we conduct mediation analyses to test whether depressive symptoms or health operate as mechanisms linking retirement and cognitive function.

Background

With Alzheimer's disease's rise in prevalence (Brookmeyer et al., 2018), a significant body of literature has developed, seeking to understand the life-course predictors of cognitive function with a mind to modifiable risk factors. This growing body of research has shown that sociodemographic, behavioral, and health factors throughout the life course are associated with later-life cognitive function.

Later-life cognitive function

We offer only a brief overview of these risk factors, as they have been reviewed extensively elsewhere (e.g., Livingston et al., 2017). Evidence suggests that early-life SES and/or exposure to hardship are associated with both early-life cognitive development (Currie, 2009; Torche, 2018) and later-life cognitive decline, even net of intervening life-course risk factors (Hale, 2017). Consistently, the most important modifiable predictor of cognitive function is educational attainment (Leggett et al., 2017). Cognitive and brain reserve theories suggest that increased education builds reserve (physically and functionally), such that the higher educated can suffer more cognitive decline before the loss passes a clinical threshold (Meng & D'Arcy, 2012).

Of course, in addition to educational attainment affecting cognitive function, it is also associated with many subsequent important life factors that also affect cognitive function, including occupational attainment. Occupation is thought to affect cognitive function through both exposure to positives (e.g., engaging complexities of higher-status work), as well as avoidance of negatives (e.g., health insults characteristic of "bad jobs") (Berr & Letellier, 2019; Kalleberg et al., 2000). Evidence suggests that level of job complexity or intellectual demands are also associated with later-life cognitive function (Andel et al., 2005; Carr et al., 2020; Fisher et al., 2014; Potter et al., 2008; Staudinger et al., 2016).

Partnership status is another important factor that follows from earlier life-course features and predicts cognitive function, though research is inconclusive as to the extent to which the association between partnership and cognitive function is related to selectivity into partnership (Fratiglioni et al., 2000). Nevertheless, the association between partnership and later-life health is robust (Franke & Kulu, 2018). Considering the association between cognitive function and socialization or cognitive engagement (Engelhardt et al., 2010), it is reasonable to expect also an association between partnership and cognitive function at older ages.

All of these life-course factors are associated with exercise, alcohol use, and mental health, which are, in turn, associated with cognitive function (Farina et al., 2014; Hale, 2017; Zhang et al., 2020). Likewise, comorbidities, such as cardiovascular disease, hypertension, diabetes, and stroke are also related to earlier-life factors and associated with cognitive function (Profenno et al., 2010; Stefanidis et al., 2018). Many of these pathways are bidirectional to some degree; for example, depression may both cause and be caused by cognitive decline (Charles, 2004). Finally, each of these factors is likely independently associated not just to cognitive function, but also to age at retirement.

Thus far, we have focused on modifiable risk factors, but one of the most important social predictors of later-life cognitive function is race/ethnicity, with Blacks and Latinx experiencing significantly lower cognitive function than Whites (Zhang et al., 2016). This is partially driven by disparities in educational and occupational opportunities (Reskin, 2012), but is also likely related to exposure to the stresses of discrimination (Das, 2013). Associations between gender and cognitive function are less clear, especially for earlier cohorts where educational and occupational opportunities for women were limited. In sum, the pathway to both the outcome (cognitive function) and the main predictor of interest (age at retirement) involves an entire life course of compounding (dis)advantages (Brown, 2018) that are interconnected and thus have been challenging to model.

Understanding "mental retirement"

Traditional regression analysis has been unable to identify how retirement affects later-life cognitive function because of a set of interrelated methodological barriers related to modeling life courses (Jones et al., 2011). Longitudinal interdependence, intermediate and time-varying confounding, and analyses of intermediate variables and outcome variables with non-linear functional forms all pose problems for standard regression models (Bijlsma et al., 2017).

Researchers have employed a variety of solutions to try and address these methodological challenges. From studies using cross-national variation in the statutory retirement age and instrumental variable techniques, there is mixed evidence that retirement is negatively associated with health, broadly, or cognitive function, specifically (Coe & Zamarro, 2011; Mazzonna & Peracchi, 2012; Rohwedder & Willis, 2010). For example, two studies that use longitudinal regression discontinuity models and eligibility for social security as an instrument find a benefit of continued labor force participation on cognitive function (Bonsang et al., 2012; Clouston & Denier, 2017). As a slight modification of the inverse implied by those findings (i.e., retirement is negatively associated with cognitive function), Celidoni et al. (2017) find that duration spent in retirement (but not retirement, per se) is associated with a larger decline in verbal memory.

At the other end of the spectrum, others find a positive effect of retirement on at least some domains of cognitive function and/or for some subpopulations (e.g., higher educated) (Bianchini & Borella, 2016; De Grip et al., 2015; Engelhardt et al., 2010). For example, studying a more nuanced version of both predictor and outcome, Denier et al. (2017) found that individuals who retired voluntarily or for family reasons had improved abstract memory scores, but those who retired for health reasons had both lower verbal memory and verbal fluency scores. Also taking a more nuanced approach, Carr et al. (2020) use inverse probability weighted regression adjustment and find that the association between retirement and cognitive function depends on occupational complexity: those in lower complexity jobs suffer a significant loss in cognitive function regardless of their retirement pathway, whereas the same was not true for those in higher complexity jobs.

Coe et al. (2012) compare results from ordinary least squares (OLS) regression models with an instrumental variable approach, using the HRS. With the OLS model, they find retirement is associated with lower cognitive function. However, when they use time between interview and offer of early retirement as an instrument, they find no negative causal effect of retirement on cognitive function for white-collar workers and a slight positive effect for blue-collar workers. Bingley and Martinello (2013) show that cross-sectional studies using country variation in the eligibility age for early and old age pension, unless controlling for education, produce negatively biased estimates for retirement. This bias, according to the authors, explains the "mental retirement"' effect found in other studies. Banks and Mazzonna (2012) argue that even controlling for education may not eliminate endogeneity bias because this problem would require simultaneously addressing the endogeneity for both education and retirement. In short, evidence on the association between retirement and cognitive function remains inconclusive, and methodological problems are the primary barrier.

Modeling life courses

We use a different modeling strategy to overcome these methodological challenges. The g-formula estimates effects through simulating interventions, which allows us to disentangle mechanisms across the life course, significantly decreasing the bias likely when using standard regression (Daniel et al., 2013; Vanderweele & Tchetgen Tchetgen, 2017). Thus, it can answer our question of: To what extent does the new statutory full retirement age of 67 delay cognitive decline compared with retiring at younger ages?

Only 37% of U.S. workers actually move directly from full-time work to retirement; patterns often involve some period of part-time work and/or movement in and out of retirement (Hudomiet et al., 2018), especially for Blacks and Latinx and women (Calvo et al., 2017). This model can also account for the added complexity of these phased retirement patterns, while other studies cannot. Furthermore, postponed retirement's effect on cognitive function may differ among subpopulations. This modeling approach allows us to examine whether there are differences in the effect of postponing retirement on cognitive function by gender, educational attainment, and occupational attainment. Gendered differences in how identity is tied to paid employment (men more than women) and in propensity to engage socially (women more than men) may contribute to gender moderating retirement's effect on cognitive decline, suggesting postponing retirement may benefit men more than women (Barnes & Parry, 2004; Kuiper et al., 2015).

The effect of postponing retirement on cognitive function may also depend on level of educational and occupational attainment. Individuals employed in higher-complexity occupations are over-represented among the highly educated, and, conversely, health insults and socioeconomic stressors related to "bad jobs" (Kalleberg et al., 2000) may also hinder benefits of ongoing labor force participation for the lower educated. We expect this combination could mean postponing retirement is more beneficial to the higher educated. If higher-complexity work is associated with better cognitive function, those who work in more monotonous jobs may not experience as much of a benefit from postponing retirement (Potter et al., 2008). At the same time, it is possible that those in non-professional occupations may benefit more from postponed retirement because the working environment, per se, is an important stimulus, especially if retirees have few cognitively-engaging non-work activities (Gow et al., 2016).

We also investigate two mechanisms through which retirement may affect cognitive decline. Those who retire might subsequently experience an increase in depressive symptoms and/or comorbidities that partially explain the effect of retirement on cognitive decline (Brand et al., 2008). Thus, we examine whether depressive symptoms or comorbidities, which are associated with both labor force participation and cognitive function (Calvo et al., 2013; González et al., 2008; Virtanen et al., 2015), operate as mediators.

Hypotheses

Based on this growing body of literature and fully exploiting the flexibility of the g-formula, we derive the following hypotheses:

Hypothesis 1. Compared with retiring between age 55 and 66, postponing retirement until age 67 or older will be protective against cognitive decline, accounting for gender, race/ethnicity, birth cohort, early-life SES, educational and occupational attainment, partnership status, exercise, alcohol consumption, depressive symptoms, and comorbidities.

Moderation analysis

Hypothesis 2. Accounting for all above covariates, postponing retirement to age 67 will be differentially protective against cognitive decline for:

a) men more than women,
b) higher educated more than those with less than a high school education
c) professional occupations more than those in non-professional occupations

Mediation analysis

Hypothesis 3. Depressive symptoms and comorbidities act as mechanisms, mediating the effect of postponing retirement on later-life cognitive function.

Methods

Dataset

The Health and Retirement Study (HRS) (1992-ongoing) is a longitudinal, nationally-representative, biennial survey of U.S. residents age 50 and over and their spouses (regardless of age). The University of Michigan conducts the HRS, which is sponsored by the National Institute on Aging (grant number NIA U01AG009740). We use RAND Version P of the HRS (RAND Center for the Study of Aging, 2017). The HRS includes retrospective data on early-life environment and educational attainment, as well as biennial data on cognitive function, health behaviors, health, and labor force participation. We use data from all waves in terms of collecting retrospective data, but focus on the period 1996 to 2014—the years for which consistent data is available for the cognitive

function measures we include. We extracted individuals who: were age 55–75 years old[1], had self-responses for cognitive function measures (we analyze cognitive function as a continuous outcome, for which proxy responses are not adequate[2], have participated in the labor market at some point in the 1996–2014 period, were not retired prior to their study entry, and have non-missingness on other covariates (less than 1% missing on any covariate) (Bonsang et al., 2012). Respondents do not have to participate in all waves to be included, so the final analytical sample is 96,918 observations from 20,469 individuals.

Key study measures

Cognitive Function. The HRS uses a modified version of the Telephone Interview for Cognitive Status (TICS-m) that was modelled after the Mini-Mental State Examination. The TICS-m was designed to be sensitive to pathological cognitive decline and minimize ceiling and floor effects (Fong et al., 2009). We also use the University of Michigan Survey Research Center's imputed values for the TICS-m measures (Fisher et al., 2017). From these, we extract a subset of questions that represent fluid intelligence – a composite of cognitive domains reflective of neurophysiological health – following a large body of literature (Crimmins et al., 2011; Ghisletta et al., 2012). The final measure sums immediate (0–10 points) and delayed word recall (0–10 points), serial 7s (counting backward from 100 by sevens) (0–5 points), and counting backward from twenty (0–2 points) (Langa et al., 2009). The range is 0–27, where higher values represent better cognitive function. For context, when validated against the clinical assessment from the Aging, Demographics, and Memory Study (ADAMS), cutpoints are 0–6 dementia, 7–11 mild cognitive impairment, and 11–27 no impairment (Crimmins et al., 2011; Langa et al., 2005).

Primary exposure

The primary exposure is employment status. Employment status is a time-varying categorical variable that indicates if an individual is full-time employed, part-time employed/part-time retired, unemployed, disabled, or retired. This measure is not based on hours worked, but the self-reported category. On average, those full-time working report at least 40 hours per week, and those who report being part-time employed or part-time retired work about 25 hours per week.

Time-varying covariates

Covariates include: *partnership status* (partnered, separated/divorced/spouse absent, never married, widowed), *weekly exercise* (1 = exercise more than once per week), *alcohol consumption* (abstinent/rare, light, moderate, heavier), a categorical measure of *depressive symptoms* to allow for a possible non-linear association between depressive symptoms and cognitive function (Center for Epidemiological Studies-Depression, CES-D, 0, 1, 2–4, or 5–8 symptoms), and *comorbidities* (0–4, diagnoses of stroke, diabetes, heart condition, and/or high blood pressure/hypertension).

Time-invariant covariates

Birth cohort follows the HRS cohort structure (AHEAD 1919–1923, Children of the Depression Era 1924–1930, HRS 1931–1941, Warbabies 1942–1947, Early Babyboomers 1948–1953, Mid Babyboomers 1954–1959). HRS reports a binary *Gender* variable (1 = Women). *Race/Ethnicity* is Non-Hispanic White, African American/Black Hispanic, Non-Black Hispanic, and Non-Hispanic Other (henceforth White, Black, Latinx, Other). *Age* is age in years. To provide a more comprehensive picture of the early-life environment, *Early-SES* includes self-reported childhood SES, childhood health, parents' education, father did not contribute economically (unemployed, absent, dead), father's lower-status occupation, childhood family moved due to financial hardship, and/or childhood family borrowed money due to financial hardship (0, 1, 2–5, or 6-7 adversities). *Educational attainment* is defined as less than high school/general equivalency diploma (GED), high school diploma, and some college or higher. *Longest job* ever held is categorized as professional or non-professional (the latter includes sales, administrative, service, manual, farms, forestry, and fishing). *Wealth* is a RAND-generated measure of household wealth that includes household assets and debts, which we average over the study period to get a time-invariant measure meant to be reflective more of later-life SES than yearly income (in debt; 0-$49,999; $50,000-$199,999; $200,000-$499,999; $500,000-$999,999; $1 million or more). In a robustness check, we include quintiles of a measure of pre-retirement income that incorporates individual earnings, unemployment earnings and workers' compensation, other government transfers, and SSDI.

Analytical strategy

The g-formula approach is implemented following four steps, using open-source software (R) (R Core Team, 2020). First, we construct a causal directed acyclic graph (DAG, Fig. 1), which portrays the interrelationships among the factors we consider. We chose a cross-lagged model where variables at t affect variables at t+1. If important effects of variables at t on other variables at t do exist, part of this effect will be lost, resulting in effect attenuation. However, the cross-lagged structure is advantageous due to certainty regarding temporal ordering, which allows us to largely avoid bias due to reverse causality. Additional effects of variables at t on variables at waves beyond t+1 could additionally have been added. However, this results in effects being estimable for fewer waves without having a strong impact on marginal effect estimates. Second, using the DAG as guidance, we estimate a series of multivariable models for the intermediate and outcome variables. Time-varying variables at age a are allowed to be affected by all time-invariant variables and, to limit assumptions on causality within a calendar year, by all time-varying variables in the previous year. We model categorical variables using multinomial logistic regression models and continuous variables with linear regression models. Third, we define intervention scenarios (e.g., what happens to cognitive function scores if people retire at age 67 instead of younger?). Fourth, we simulate an approximation of the empirical data ("the natural course scenario") and an approximation of the sample under an "intervention scenario." More detailed information on the third and fourth steps is provided below.

Natural course vs. intervention

To test Hypothesis 1, we contrast the natural course scenario with the intervention scenario, where we set all individuals who retired younger than age 67 to have the status of full-time employed until at least age 67. After age 67, they can retire following observed retirement risks for individuals with their covariate distribution. This is accomplished in the simulation step of the g-formula, including a 500-iteration bootstrap to produce standard errors and confidence intervals (Efron & Tibshirani, 1994). The simulation process follows that of other longitudinal g-formula implementations elsewhere (Bijlsma et al., 2017; Bijlsma & Wilson, 2020; Robins, 1986). By taking the differences between the intervention scenario and the natural course scenario, we calculate the

[1] We select this age range to represent the twenty-year period over which most Americans retire.

[2] When generating a categorical measure for no, mild-, or severe cognitive impairment, proxy reports on a set of measures can be used, following ADAMS (e.g., Crimmins et al., 2011; Weir et al., 2014). In this case, we use a continuous score for cognitive function. Cognitive impairment is a primary driver of proxy status, so proxy responses are typically very important for analysing cognitive impairment at older ages. However, we focus on younger ages and labor force participation, which should make having to exclude proxies less problematic. Only a small number of responses (<5% of person-waves in our analytical sample) can be categorized as both working and using a proxy.

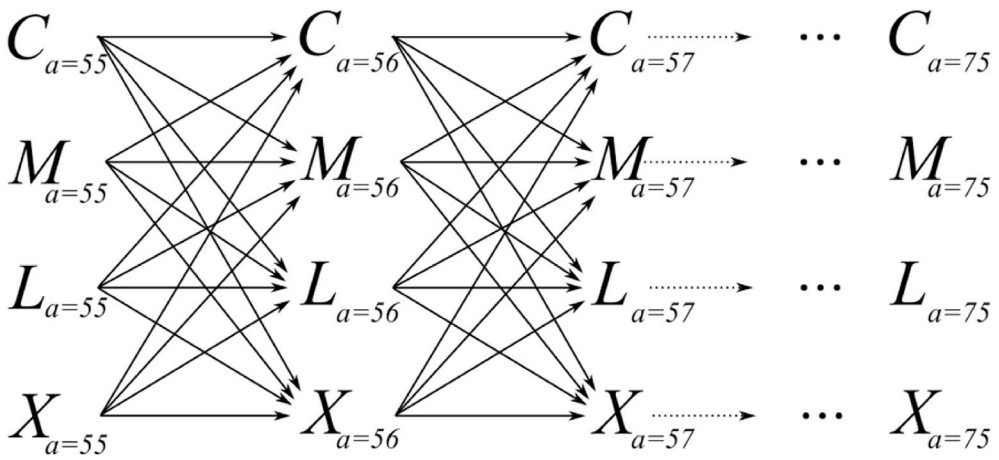

Fig. 1. Simplified directed acyclic graph (DAG) showing the single-year cross-lagged structure whereby cognitive function (C), mediating factors (M), labor force participation (L) and time-varying confounders (X) are associated across age (a) 55 to 75. For simplicity, the DAG does not show time-invariant control variables, but these are included in all models.

total effect of our intervention of postponing retirement to age 67 (Wang & Arah, 2015).

Subgroup analysis: gender, educational attainment, and occupational attainment

Hypothesis 2 is that postponing retirement to age 67 will be differentially protective against cognitive decline for men more than women, the higher educated more than the lower educated, and those in professional more than non-professional occupations. To test this, we allowed for interaction terms between labor force participation and gender, education, and occupation within the multivariable models. This allows for separate effects of employment and retirement on cognitive function by gender, education, and occupational group. Following the g-formula procedure explained above, we then compare subgroup results.

Mediation analysis: depressive symptoms and comorbidities

To test Hypothesis 3, we perform mediation analyses in which we determine to what extent the effect of labor force participation on cognitive function is mediated through depressive symptoms and comorbidities. The direct effect of postponing retirement is determined by performing simulations that are identical to the intervention scenario (as in Hypothesis 1) for the total effect, with the exception that mediators of interest are kept at their natural course levels. Keeping the mediators at their natural course levels prevents the intervention from affecting the mediators, thus eliminating the part of the intervention effect that operates 'via' these mediators. The indirect effect, the portion that does operate via the mediators of interest, is determined by subtracting this direct effect from the total effect. This allows us to examine to what extent the effect of postponing retirement operates through retirement's effect on depressive symptoms or comorbidities. See also Appendix III for more information on the mediation analysis.

Sensitivity analysis

In the intervention scenarios presented above, after postponing retirement until age 67, individuals were "allowed" to retire following empirical expectations conditional on their covariate values. Using this scenario, any difference in cognitive function between the intervention and natural course scenarios that is evident after age 67 could be caused both by the enduring protective effect of postponed retirement at the individual level and by having a larger number of not-yet retired individuals after age 67 in the intervention scenario relative to the natural course.

Therefore, we produced an additional comparison where, in both the intervention and the natural course scenarios, all individuals were "forced" to retire at age 67. This comparison eliminates compositional differences in the number of retired individuals at age 67 and older. This means that any population-level differences in cognitive function at age 67 or older reflect only an enduring protective effect of postponing retirement at the individual level. These results are similar to the main results (Appendix II).

Furthermore, we also performed a minor sensitivity analysis where we additionally adjusted for income. We note that we found similar results with this additional analysis (not reported herein due to substantial missingness (50% of current sample), but results are available upon request).

Results

Table 1 shows descriptive statistics for the analytic sample. About 46% of the observations are in full- or part-time work, while almost 45% are in the retirement state. Retired and disabled individuals have the lowest cognitive function scores. Those who are retired are older and more likely to be women than those who are working. The persistence and interconnectedness of disadvantage over the life course is clear in that Blacks and Latinx are more likely to be unemployed or disabled and less likely to be retired, and those with more early-life disadvantages and lower educational attainment are more likely to be disabled. Those working are more likely to exercise and have fewer comorbidities or depressive symptoms.

Fig. 2 displays the population-averaged effect (PAE) of postponing retirement to at least age 67. Even at the population level (i.e., where even people who did not retire prior to age 67 are in the denominator), there is a positive effect for both women and men of postponing retirement until age 67 or older, accounting for all time-invariant characteristics (cohort, race/ethnicity, early-life SES, and educational and occupational attainment) and accounting for the bidirectional association between labor force participation and time-varying factors (partnership status, exercise, alcohol consumption, depressive symptoms, and comorbidities). This intervention scenario compared to the natural course consistently shows a positive effect throughout the age range, including a positive effect after age 67 up until at least age 74 for both men and women (Fig. 2 and Appendix I). However, as explained above, we cannot attribute the extent of this positive effect only to an enduring protective effect of postponed retirement. The effect could be driven also by a larger number of individuals who continue to work at older ages, as this intervention scenario "allows" individuals to retire

Table 1
Descriptive statistics for the analytic sample by labor force participation.

Variable	FT	PT	Retired	Unemployed	Disabled	NILF	Total
	Mean/%	*Mean/%*	*Mean/%*	*Mean/%*	*Mean/%*	*Mean/%*	*Mean/%*
LFP distribution	29.3	16.9	44.6	2.1	2.3	4.8	100
Cognitive Function	16.9	16.6	15.3	15.8	13.5	15.6	16.0
Number Cognitive Tests	5.9	6.3	6.2	5.2	5.5	6.3	6.1
Age	60.3	64.4	67.3	61.1	60.7	63.4	64.3
Female	48.1	60.1	56.0	49.9	64.2	93.7	56.0
			Rows total to 100%				
Race/ethnicity							n person-waves
White	28.8	17.7	45.9	1.7	1.6	4.4	74,791
Black	29.3	15.3	44.9	2.8	4.6	3.1	17,122
Latinx	31.6	14.2	35.9	3.6	4.0	10.6	9,820
Other	35.4	15.1	38.7	3.6	3.1	4.1	2517
Early-Life Socioeconomic Status							
6–7	23.3	14.2	49.9	2.2	5.9	4.5	1,869
2–5	26.4	16.0	47.4	2.1	2.9	5.1	54,430
1	29.6	17.5	44.4	2.0	1.7	4.8	29,377
0	38.0	18.8	36.0	2.4	1.1	3.7	18,574
Educational Attainment							
Less than HS/GED	20.8	14.1	50.5	2.1	5.0	7.6	24,951
HS Diploma	28.6	16.9	45.8	2.0	1.8	4.8	50,828
Some College+	38.0	19.5	37.2	2.2	0.9	2.2	28,471
Longest Occupation							
Non-professional	27.1	16.2	45.7	2.2	3.0	5.9	71,320
Professional	34.1	18.5	42.3	1.9	0.9	2.3	32,930
Wealth Average							
In debt	26.8	13.4	38.9	5.9	9.3	5.7	4,138
$0–49 K	27.0	14.3	44.3	3.0	5.6	5.7	20,688
$50–199 K	30.8	16.0	44.6	2.0	1.7	4.9	31,860
$200–499 K	29.5	17.9	45.8	1.7	0.9	4.2	25,307
$500–999 K	28.6	18.7	47.0	1.3	0.5	4.0	13,846
$1 mil+	31.0	22.7	40.5	0.9	0.4	4.6	8,411
Partnership Status							
Never Married	33.9	14.8	41.2	3.0	4.9	2.0	3,906
Married/Partnered	30.3	17.4	43.3	1.9	1.7	5.3	72,273
Sep/Divorced/Absent	33.7	15.9	40.1	3.3	4.2	3.0	15,092
Widowed	17.1	16.0	57.9	1.4	2.7	4.9	12,979
Exercise							
Weekly or less	27.0	14.6	47.8	2.0	3.5	5.2	50,726
More than weekly	31.5	19.2	41.6	2.2	1.2	4.4	53,524
Alcohol consumption							
Abstinent/rare	26.7	15.9	46.6	2.0	2.9	5.8	66,957
Light	31.1	19.6	43.4	1.7	1.0	3.2	15,673
Moderate	36.1	19.1	38.6	2.2	1.0	3.0	12,611
Heavier	36.0	16.7	39.8	3.4	1.8	2.3	9,009
CESD							
0	33.1	19.2	41.4	1.7	0.6	3.9	49,250
1	30.1	17.1	44.2	2.1	1.7	4.8	22,858
2–4	24.8	14.1	49.3	2.4	3.9	5.5	22,241
5–8	18.4	11.4	50.7	3.7	8.5	7.3	9,901
Comorbidity Index							
None	37.3	19.1	34.5	2.3	1.4	5.4	36,907
One	29.4	17.4	44.7	2.1	1.9	4.5	38,696
Two	21.6	14.6	53.9	2.0	3.3	4.5	20,938
Three	12.4	11.1	65.5	1.5	5.9	3.7	6,645
Four	5.8	6.2	74.6	1.1	8.6	3.7	1,064

older than 67.

Therefore, as explained above, we conduct sensitivity analyses where we "force" everyone to retire at age 67. Here any effect at age 67 or older is evidence of an enduring protective effect of postponing retirement. Even in this forced-retirement scenario, the protective effect lasts at least five years post retirement, up to at least age 72 for both men and women (Appendix II). This protective effect operates not because labor force participation improves cognitive function, but because in the natural course scenario, those who retire younger than 67 experience faster cognitive decline. This is strong evidence for Hypothesis 1 that postponed retirement is associated with better cognitive function.

In interpreting the meaning of the PAE, it is informative to compare

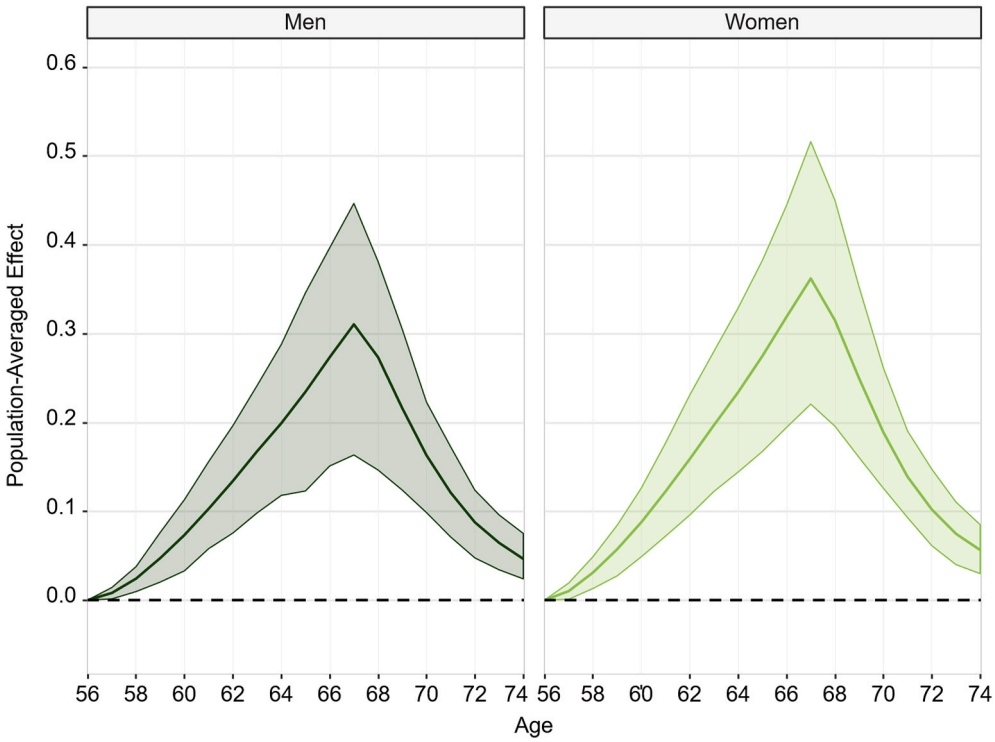

Fig. 2. Population-averaged effect (PAE)—the difference in cognitive functioning between the natural course scenario and the intervention where retirement is postponed until at least age 67.

this protective effect with the number of points lost in the natural course scenario. The average age at retirement for those who retire before age 67 is age 61, which does not differ substantially by subgroup[3](by gender, race/ethnicity, education, or occupational attainment). From age 61 to age 67, the average change in cognition in all subgroups is approximately 1 point on the 0–27 scale, ranging from least lost for those with less than high school (0.92) to most lost for those in professional occupations (1.06). Delaying retirement to age 67 allows men to retain a score that is 0.31 (95% CI: 0.16, 0.45) points higher than if their retirement had not been delayed, and for women this is 0.36 points higher (95% CI: 0.22, 0.52). Therefore, relative to the average decline over the age 61 to 67 period, this represents approximately a one-third reduction in cognitive decline over the relevant time period. Note that only those who retire prior to age 67, approximately 63% of the sample, are affected by the intervention. Individuals who work up to age 67 are not affected by the intervention, but do contribute to the denominator. In other words, they dilute the estimate.

Whereas above we present the population-averaged effect, for Hypothesis 2, we present the average treatment effect on the treated (ATT); that is, the effect of postponed retirement on only those who actually do retire younger than age 67. Fig. 3 shows that, indeed, postponed retirement's substantial effect on the individual (men: 0.42, 95% CI: 0.22, 0.59; women: 0.44, 95% CI: 0.27, 0.63). Again, the protective effect lasts well beyond age 67 (Appendix II). However, we find no support for Hypothesis 2a that men differentially benefit from postponed retirement compared with women.

Similarly, Fig. 4 presents the ATT by education. Those with higher educational attainment have slightly higher point estimates across all ages than those with lower educational attainment (less than high school: 0.31, 95% CI: 0.09, 0.48; high school: 0.46, 95% CI: 0.26, 0.64; some college or higher: 0.50, 95% CI: 0.32, 0.71). Although we find no clear evidence for our Hypothesis 2b that those with higher education would differentially benefit, the effect size for those with at least some college is particularly substantial, suggesting postponed retirement is associated with an approximately 50% reduction in cognitive decline for this group.

Both those in non-professional (0.45, 95% CI: 0.27, 0.64) and in professional occupations (0.37, 95% CI: 0.16, 0.57) appear to benefit from postponed retirement, with a slightly greater, but non-significant benefit to the former (Fig. 5). Thus, our findings do not support Hypothesis 2c for a professional advantage.

We next test Hypothesis 3, where we conduct mediation analyses to identify to what extent depressive symptoms or comorbidities operate as mechanisms through which retirement affects cognitive function. We find no evidence for Hypothesis 3 that retirement's negative effect on cognitive function is because retirement causes depressive symptoms or health problems that, in turn, cause cognitive decline (Appendix III).

Discussion

Interdependent life-course processes influence cognitive function in terms of "achieved" cognitive function, as well as rate of decline. However, there are critical gaps in our knowledge about the modifiable factors that may be protective against later-life cognitive decline, a shortcoming related both to a lack of research that takes into consideration that life-course factors are dynamically interconnected and the related methodological barriers. The g-formula is a more flexible modeling strategy that better approaches the empirical reality of the life course's influence on later-life cognitive health.

Accounting for demographic and early-life factors, as well as the longitudinal interdependence between educational and occupational attainment, labor force participation, and health, we find evidence for Hypothesis 1 that postponing retirement to age 67 provides an insulative effect against cognitive decline. Even the population-averaged effect of the intervention shows a 30–34% reduction, for men and women,

[3] Note: this is the average age at retirement for those people who retire in the age 55–66 range. Hence, there is not much space for differentiation. Differentiation does exist at the decimal level.

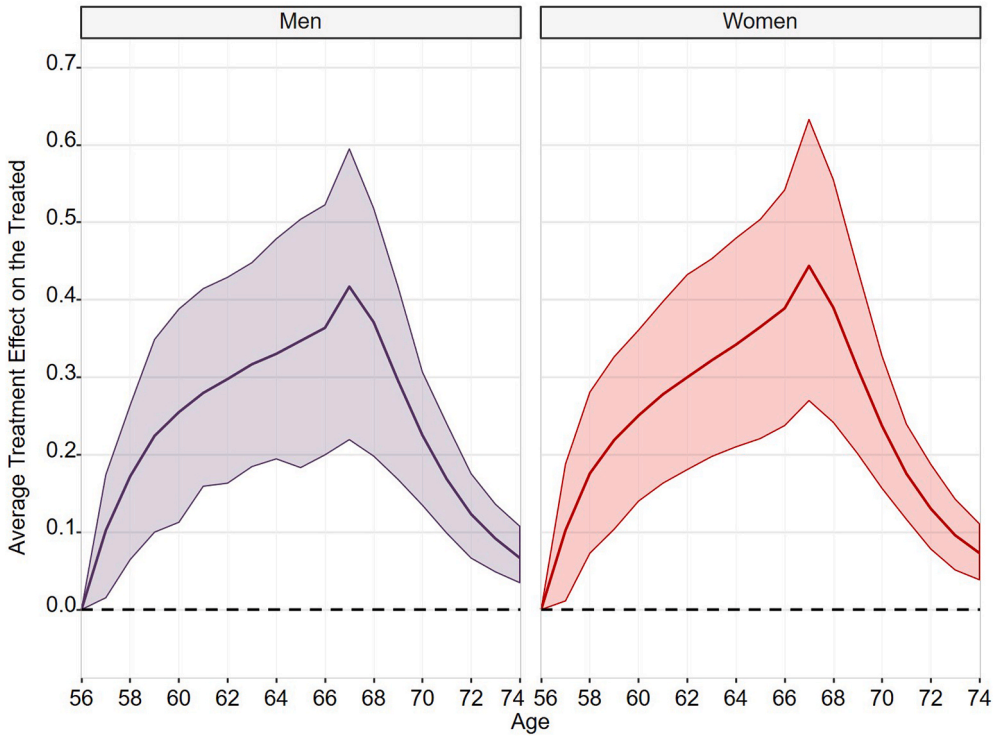

Fig. 3. Average treatment effect on the treated by gender— the difference in cognitive function score between the natural course and intervention scenarios only for those who retired prior to age 67.

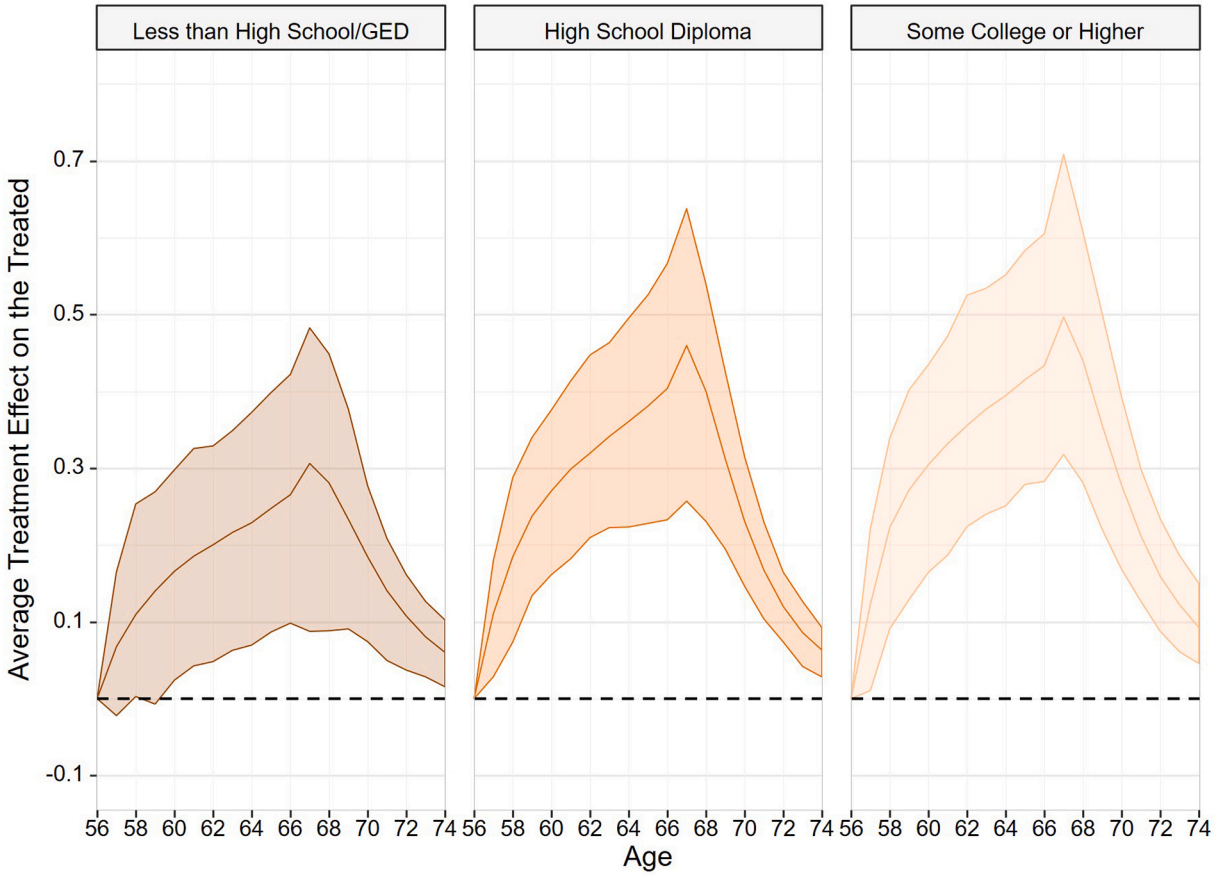

Fig. 4. Average treatment effect on the treated by educational attainment—the difference in cognitive function score between the natural course and intervention scenarios only for those who retired prior to age 67.

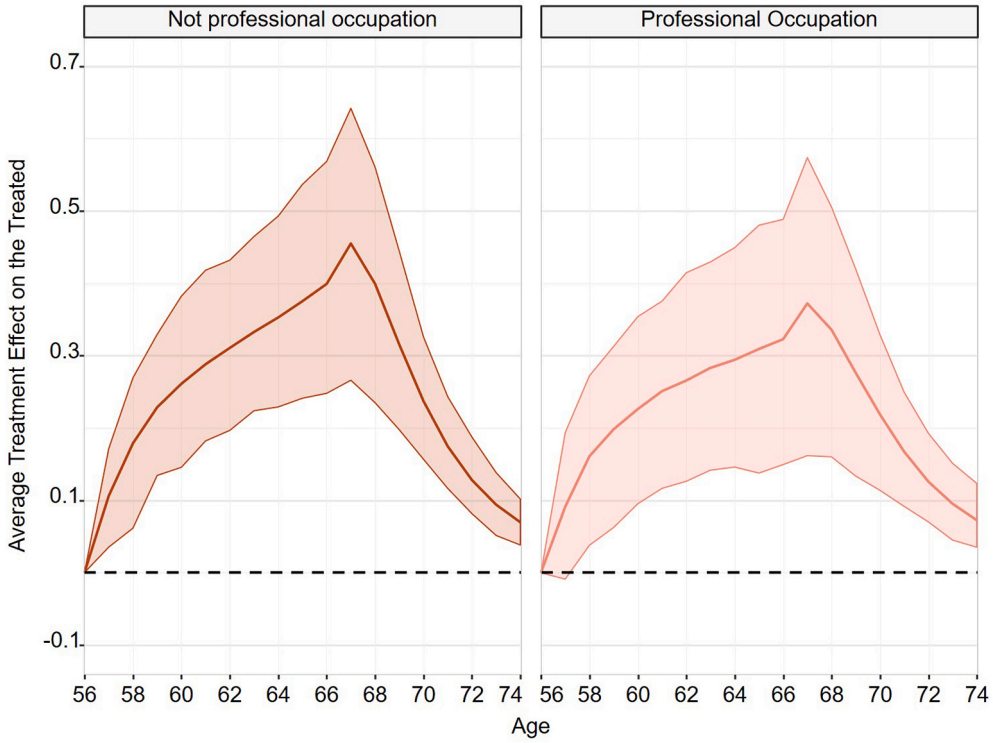

Fig. 5. Average treatment effect on the treated (ATT) by occupational attainment—the difference in cognitive function score between the natural course and intervention scenarios only for those who retired prior to age 67.

respectively, in cognitive decline associated with remaining employed compared with retiring younger than age 67. The effect is related to a slowed rate of cognitive decline versus a "boost" in cognitive function. The protective effect appears to hold regardless of gender, educational attainment, or occupational attainment, thus we find no clear evidence for Hypothesis 2 that certain subgroups would differentially benefit from postponed retirement. We hypothesized that a mechanism through which retirement may affect cognitive function may be related to experiencing some level of depression and/or health insults as a result of retirement. We did not find evidence that either of these explained much of the association between retirement and cognitive function.

This study accounts for time-variant and time-invariant predictors of cognitive function and age at retirement, as well as their dynamic interactions over the life course; however, there remain distinct limitations. Our ability to test for alternative mechanisms is limited by the data. Indeed, for analytical reasons (power) and data constraints (no information, e.g., on job complexity), we are forced to oversimplify what are important distinctions, such as dichotomizing occupation as professional versus non-professional. Work tasks and conditions also contribute to cognitive function. Data that allow for a more detailed analysis of occupation, such as considering current job tasks, e.g., complexity (Andel et al., 2005; Berr & Letellier, 2019), embedded within a life course approach would offer a strong contribution.

For example, Carr et al. (2020) investigate the role occupational complexity plays in the association between alternative retirement pathways and change in cognitive function, exploring alternative transitions, e.g., downshifting to part-time work, retiring, and returning to work. That approach yields interesting results showing the detrimental impact of retirement on those working in lower-complexity jobs; these are akin to our findings with regard to the lower-educated suffering more loss than the higher educated. However, Carr et al. do not address age trajectories of cognitive decline, while we estimate both the natural course and the counterfactual trajectories of cognitive decline across age. Read together, both papers indicate that retirement affects subgroups differently, potentially further disadvantaging disadvantaged groups, suggesting more research is warranted in this area. Furthermore, we note that effects in our study may be underestimated compared to Carr and colleagues' due to the modeling of a cross-lagged structure, including effects via mediators, in the presence of wave-to-wave idiosyncratic variation in cognitive score.

Relatedly, other research suggests that socially- or cognitively-engaging activities that are not recorded as labor market participation, e.g., grandparenting or volunteering, may also be protective (Arpino & Bordone, 2014; Gow et al., 2016). It is plausible that the relationship between retirement and cognitive function is driven by other changes in cognitive engagement that we were unable to identify with the available data. In order to study the interplay of market and non-market activities, future research should exploit other datasets that include more detailed measures of unpaid labor and social activities, such as the National Social life, Health, and Aging Project (NSHAP).

Also, unlike Denier et al. (2017), we are not able to identify the reason for retirement, which means we cannot determine to what extent retirement is voluntary. Whether retirement is voluntary versus involuntary is likely to impact depressive symptoms and general health, and likely also cognitive function. We do take into consideration depressive symptoms and health status as mediators. Similarly, we are unable to capture all the lowest cognitive function scores (as many respondents with impairment require a proxy), which may bias results. More than double those in the analytical sample who have less than a high school diploma or GED have a proxy compared with those with college or more, and more than double those with manual jobs have a proxy than those with professional occupations. This could potentially contribute to explaining part of the high benefit of postponed retirement for the low occupational group. Further research investigating reason for retirement could provide insight.

Limitations

There are some methodological limitations. The causal claims of this study rely on three fundamental assumptions: positivity, consistency,

and exchangeability (Greenland & Robins, 2009; Petersen et al., 2012; Rehkopf et al., 2016). The positivity assumption requires that individuals who were hypothetically intervened on had in fact a non-zero chance of receiving such an intervention in the real world. In our study, this requires that individuals who hypothetically had their retirement postponed could in fact be employed. Our intervention was not performed on those individuals who were disabled or out of the labor force. However, there may have been other reasons for individuals to retire for which we do not have information. Since the intervention would not have been possible for these individuals, the true population-averaged effect would be smaller. Nevertheless, this issue will not have a substantial effect on the treatment effect for the treated.

The consistency assumption requires that the hypothetical intervention of interest is well-defined and that the variable representing it in the dataset corresponds to this definition. In our study, our hypothetical intervention was a postponement of retirement to age 67 for those individuals who retired before age 67. The variable used to hypothetically implement this intervention was a variable indicating employment status, and its effect is, by definition, drawn from those individuals who are employed at each age (relative to those who are not). Individuals who continue working at certain ages may do so for a variety of reasons, e.g., for health and financial reasons. Since we use information from these individuals, our hypothetical intervention does not represent a forced intervention, but a scenario that represents individuals choosing to work longer of their own accord (for any reason). An important limitation is that some individuals have chosen to retire because of causes that are not represented (to the same degree) in the population of individuals who continue to work. This brings us to the exchangeability assumption.

Applied to our study, the exchangeability assumption requires that individuals who are employed are comparable, in terms of factors that affect cognitive function, to individuals who are retired, conditional on the measured covariates. For example, individuals may choose to retire because their health has deteriorated, and they are no longer able to perform full- or part-time work. If this deteriorated health has also affected their cognitive function, then this effect of health on cognitive function – if it has not been adjusted for in the study – will bias the association between employment and cognitive function away from the causal effect of employment on cognitive function. In our study, we adjust for a large number of potential confounding variables. However, it is likely that the models have not adjusted for some important confounding variables or that those that are measured do not perfectly capture salient matters (an issue known as residual confounding). Hence, although the aim of this study is to approximate a causal effect, we cannot eliminate all biases.

Conclusion

This work has significant implications. Longer life expectancies and population aging have motivated many high-income countries to postpone the statutory retirement age for more recent cohorts. In the U.S., the government raised the statutory retirement age for successive cohorts, and for those born after 1960, that age is 67. Prior evidence was inconclusive as to whether labor market participation is protective against cognitive decline because working is dynamically interconnected with other life-course factors that influence cognitive function. Using advanced counterfactual modeling allows us to test the effect sizes of hypothetical intervention scenarios. This study examines both what the U.S. postponed retirement age may mean at the population-level, as well as what it means to those who themselves postpone retirement.

Our findings suggest that postponed retirement is beneficial to cognitive function for all genders, races/ethnicities, educational levels, and regardless of professional or non-professional occupational status. The clear implication is that more recent cohorts, who have an older statutory retirement age, may, indeed, enjoy an enduring protective effect of postponed retirement against cognitive decline.

Author statement

All authors contributed to the study conception and design. Data management: JMH, AL; Methodology: MJB, AL; Formal analysis and investigation: MJB; Visualization: AL, JMH; Writing - original draft preparation: JMH, MJB; Writing - review and editing: JMH, MJB, AL; all authors read and approved the final manuscript.

Ethical approval

This study uses only secondary data analysis of the publicly available data in the Health and Retirement Study, a longitudinal project sponsored by the National Institute on Aging (NIA U01AG009740) and the Social Security Administration.

Ethical approval for the Health and Retirement Study was obtained from the University of Michigan Institutional Review Board.

Declaration of competing interest

The authors declare that they have no conflict of interest.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ssmph.2021.100855.

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