where age at dementia diagnosis or age at censoring (31st December 2010) was the dependent variable and age of retirement was the independent variable. Hazard ratios were computed adjusting for gender, marital status, occupational category, type of retirement, pension amount, diagnosis of hypertension, diabetes. Sensitivity analyses to assess potential reverse causation and differential cohort or temporal diagnosis biases were undertaken. **Results:** Among the 429,803 retired self-employed workers alive on December 31st 2010, prevalence of dementia was 2.65%. Workers had been retired on average for more than 12 years. Multivariable analyses showed that the hazard ratio (HR) of dementia was 0.968 (95% Confidence Interval= [0.962-0.973]) per each extra year of age at retirement. After excluding workers who had dementia diagnosed within the 5 years following retirement, the results remained unchanged and highly significant ($p<0.0001$). Results were also similar in further analyses stratified by age categories or year of dementia diagnosis. **Conclusions:** Professional activity may be an important determinant of mental exercise and social integration. Our data show strong evidence of a significant decrease in the risk of developing dementia associated with older age at retirement, in line with the "use it or lose it" hypothesis. This health perspective should be taken into consideration when the age of cessation of professional activity is discussed. Our results thus highlight the importance of maintaining high levels of cognitive and social stimulation throughout work and retiree life and emphasize the need for interventions and policies to help older individuals achieve such cognitive and social engagement.

O2-13-02 ASSESSING LONG-TERM POSTOPERATIVE COGNITIVE DECLINE USING CONVENTIONAL AND QUASI-EXPERIMENTAL APPROACHES: A MONTE CARLO SIMULATION STUDY

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Background: Previous studies of postoperative cognitive decline (POCD) may have produced biased estimates because they did not take into account preoperative cognitive trajectories. We investigated the accuracy of conventional and quasi-experimental approaches in estimating the effect of coronary artery bypass graft surgery (CABG) on POCD in a Monte Carlo simulation study in which the underlying true effects are known. **Methods:** We simulated outcomes for 500 CABG patients and 500 matched controls with 1,000 datasets for each conventional (ordinary least-squares regression) and quasi-experimental (linear-spline regression) analysis. Cognition was generated before (years -5, -3, -1, and 0) and after surgery (years 1, 3 and 5) on a 0-35 point scale. Baseline cognition was lower in CABG patients (25.0 [SD=2.0] versus 27.0 [SD=2.0] points) and annual POCD was higher (0.64 versus 0.4 points). Individual variation was introduced into intercepts (within-group SD of 1.41 points) and slopes (SD of 0.05 points annually). Annual preoperative cognitive decline in controls was held constant at 0.32 points. Annual preoperative cognitive decline in CABG patients was set at 0.36 points in Scenario 1 (-0.2 point true harmful effect of CABG), 0.56 points in Scenario 2 (no true effect of CABG), and 0.76 points in Scenario 3 (0.2 point true protective effect of CABG). **Results:** Conventional analyses suggested surgery increases POCD by a mean of 0.24 points annually (Table 1*): correctly identifying the true association in 100% of simulations in Scenario 1, though 0% in Scenarios 2 and 3. By taking into account the difference-in-differences between preoperative cognitive decline and POCD, quasi-experimental models gave highly accurate estimates across all three Scenarios (94-100% correct). Figure 1 illustrates observed and expected POCD in CABG patients in Scenarios 1 and 3 on the basis of preoperative cognitive trajectories and the difference between prior and postoperative periods in the control group. **Conclusions:** This study demonstrates the potential for bias when the effect of CABG on cognitive decline is estimated from rates of POCD without taking into account preoperative cognitive trajectories. Our findings have important implications for other interventions and health shocks in general, and further research adopting innovative approaches is needed to inform clinical decision-making.

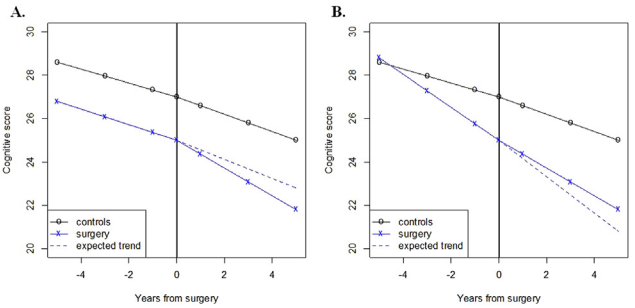

Figure 1. Harmful (A) and protective (B) effects of CABG on postoperative cognitive decline depending upon different trajectories of preoperative cognitive decline in CABG patients.

Table 1
Conventional and quasi-experimental estimates of the effect of CABG on cognitive decline using Monte Carlo simulation*

		Conventional approach				Quasi-experimental approach			
Scenario	True effect†	β	95% CI	p	% of estimates correct‡	β	95% CI	p	% of estimates correct‡
1 (Harmful effect)	-0.2	-0.240	-0.291 to -0.189	6.2×10^{-13}	100.0	-0.200	-0.274 to -0.127	0.00015	99.9
2 (No effect)	0.0	-0.241	-0.292 to -0.190	8.7×10^{-13}	0.0	-0.002	-0.075 to 0.072	0.483	93.7
3 (Protective effect)	0.2	-0.240	-0.291 to -0.189	2.3×10^{-12}	0.0	0.200	0.126 to 0.273	0.00033	99.9

*Based on mean values from 1,000 simulated datasets per scenario by conventional approach (ordinary least squares regression of postoperative cognitive changes adjusted for baseline cognition) and by quasi-experimental approach (difference-in-differences between preoperative and postoperative cognitive changes using linear spline regression).

†True underlying effect on annual rates of cognitive decline on a 0 to 35 point scale where higher values represent better cognition.

‡Percent of simulations where a true negative/positive effect correctly detected at $p<0.05$, or no effect correctly detected at $p\geq0.05$.

O2-13-03 DO SOCIOECONOMIC DISPARITIES EXPLAIN HIGHER DEMENTIA INCIDENCE AMONG BLACK OLDER ADULTS?

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