MONASH University

ECONOMICS OF AGEING ACROSS THE LIFE-CYCLE

NICOLAU MARTIN BASSOLS

B.SC. AND M.SC. ECONOMICS

A THESIS SUBMITTED FOR THE DEGREE OF DOCTOR OF PHILOSOPHY

AT MONASH UNIVERSITY IN 2022

MONASH BUSINESS SCHOOL, CENTRE FOR HEALTH ECONOMICS

Abstract

This thesis explores how different old-age related problems can be minimised by taking preventive measures during different stages of the life-cycle of individuals. Three essays examine independent research questions and outcomes. The first essay explores if very short-term events of extreme air pollution can cause long-term health effects that can extend to older adulthood and if socioeconomic status (SES) can compensate for them. The main hypothesis is that individuals exposed to high levels of air pollution in-utero or at early childhood could suffer long-lasting detrimental respiratory health problems. It is found that individuals exposed to short episodes of intense air pollution in-utero present significantly worse respiratory health outcomes throughout adulthood. The effects are most substantial for exposure to air pollution during the first trimester of pregnancy and males born in low SES areas. The second essay explores whether cognitive work activities involved in individuals' most prolonged lifetime occupations could affect their likelihood of suffering dementia at an older age. The main hypothesis is that individuals working in occupations with high levels of cognitive work activities build up their cognitive reserve throughout their lives, protecting them from dementia at an older age. The results suggest a robust association between cognitive activity at work and predicted dementia. The third essay explores the relationship between age-related cognitive deterioration with risky and non-risky financial investments. The main hypothesis of this essay is that individuals' financial investments are determined by their levels of cognition and cognitive deterioration. It is found that investing in financial assets is strongly associated with cognition, cognitive deterioration reduces the probability of holding risky financial investments, and cognition does not moderate households' financial reactions to stock market fluctuations.

Declaration

This thesis is an original work of my research and contains no material which has been accepted for the award of any other degree or diploma at any university or equivalent institution and that, to the best of my knowledge and belief, this thesis contains no material previously published or written by another person, except where due reference is made in the text of the thesis.

Nicolau Martin Bassols

08.02.22

Nicolau Martin Bassols

Dedication

To Trinidad Rouse Olle

AKNOWLEDGMENTS

This research was supported by an Australian Government Research Training Program (RTP) Scholarship. I thank Judit Vall Castello, who introduced me to research and showed great patience and generosity at the first stage of my career. I also thank Anna Garcia Altes and Rennan Goetz, who helped me throughout my PhD application. During my PhD, I have been greatly supported by my fantastic PhD colleagues and Senior Researchers of the Centre for Health Economics of Monash University. I want to especially thank my panel members Johannes Kunz, Daniel Avdic, and Gang Chen, who took the utmost care of my progress. My supervisors David Johnston, Sonja de New, and Michael Shields have also been incredibly supportive at all moments. I have grown immensely as a researcher and as a person, thanks to them. I also want to thank Arie Kapteyn for hosting me at the University of Southern California in the most inspiring research visit. In addition, I greatly value the work of Angie Martin for proofreading the thesis. Outside the academic field, too many friends and family members have helped me throughout this process to mention. I want to thank every single one of them, but especially my parents. You always encouraged me to start exciting projects and to dream big. I always could count on you and never felt alone throughout this journey.

Contents

Introduction

1 Historical perspective

In the past century, there has been the highest increase in the world GDP experienced in human history (Treasury *et al.*, 2001; Fund. and Fund., 2000). The annual average rate worldwide was three per cent, while worldwide GDP increase had been only moderate throughout history. Estimations show that annual average GDP growth from the year 1000 to 1500 was 0.2%, from the year 1500 to 1820 it was 0.3%, and from 1820 to 1900 it was 2.1% (Maddison, 2000). This sharp growth in modern history has been mainly driven by the scientific and industrial revolution that raised productivity levels unprecedentedly. It had a strong cumulative effect, leading to exponential growth. This revolution started in western countries and caused the Great Divergence. This event was characterised by a constant evolution of worldwide GDP throughout most history, which was close to zero, but a significant growth divergence across countries after the scientific and industrial revolution. A group of countries proliferated (developed countries), while others grew at low rates (developing countries). This scenario has changed in the last 50 years, and developing countries are catching up and experiencing unprecedented large economic growth. Therefore, there is a consensus of having arrived at a milestone of the human history of economic progress never experienced before, which should keep increasing in the near future. Staff (2000) show that by the year 2000, countries at the 25th to 50th percentile of global GDP per-capita were richer than countries at the 75th to 100th percentile at 1900; while countries at the 25th percentile or lower in the year 2000 were richer than countries at the 50th to 75th percentile in 1900. This suggests that even the most impoverished countries have experienced a significant increase in their GDP throughout the last century.

A worldwide rise in GDP directly impacted education, health, mortality, and similar welfare indicators, with all of them experiencing significant improvements. These variables are closely linked and have a significant impact on each other (Fernandez, 2019). Life expectancy has been greatly prolonged due to progress in health and public policies, better education, and rising incomes. All these factors improved individuals diets, hygiene standards, and similar health-promoting behaviours, shifting global life expectancy

to 73.9 years in 2019, with an increase in six years since 2000 (World Health Organization, 2020). In the least developed countries, life expectancy in 2018 was 63 years (The World Bank, 2020), while for OECD countries at the same year it was 80.6 (OECD, 2021). Compared to historical values, in 1870, the longest life expectancies worldwide were in Norway and Australia and were only 49.3 and 48 years, respectively, while in 1880 in India, it was 25 years. These enormous changes show that globally there has been an extraordinary increase in life expectancy.

Not only has there been an increase in life expectancy, but also a great fertility decline. Thanks to better education, women empowerment, and access to contraceptive methods, fertility worldwide has experienced a significant decrease (United Nations, Department of Economic and Social Affairs, Population Division, 2015). Developing countries experienced the strongest decrease: in Asia from 1950 to 1955 there were 5.8 births per woman, which fell to 2.2 births from 2010 to 2015; in Latin America and the Caribbean from 1950 to 1955 there were 5.9 births per woman, which fell to 2.2 births from 2010 to 2015. In the least developed countries, these changes were more moderate but still considerably large, with 6.6 births per woman from 1950 to 1955 dropping to 4.3 births per woman from 2010 to 2015. Birth rates in Europe from 1950 to 1955 were 2.7 births per woman and decreased to 1.6 births from 2010 to 2015, while in northern America, birth rates from 1950 to 1955 were 3.4 births per woman and decreased to 1.9 births from 2010 to 2015. This demographic change has caused many countries to experience non-replacement fertility rates, which means that births do not counter-balance mortality. Countries with non-replacement birth rates would experience a decrease in their total population if they did not experience other demographic conditions such as immigration. In 1970 there were only 22 countries ($\approx$15% of the world population) that had transitioned into below-replacement birth rates, while by 2013, these numbers had almost quadruplet to 83 countries ($\approx$50% of the world population) (United Nations, Department of Economic and Social Affairs, Population Division, 2017). It is worth noticing that many middle-income countries that recently reached these below-replacement fertility rates did a much faster transition than high-income countries that reached them in the past. Therefore, this demographic process is accelerating, and it is expected that developing countries will experience a much faster fertility change than the one developed countries went through in the past century. This change will lead worldwide fertility in the future to fall from 2.5 births per woman from 2010 to 2015 to 2.3 births by 2045 to 2050 and two births by 2095 to 2100 (United Nations, Department of Economic and Social Affairs, Population Division, 2015). As such, below replacement, worldwide fertility rates are predicted in the near future.

From this, it can be inferred that even if human progress in the last centuries has brought enormous advances in welfare, it also involves new and vital challenges. Almost doubling worldwide life expectancy is a very positive milestone in human history, as it reflects an enormous improvement in welfare. Still, combined with the natural transition to lower fertility rates, it leads to a growth in ageing population and a

problem never confronted before. By definition, a longer life expectancy will lead to a sharp rise in old age-related problems such as physical- and neuron-degenerative health problems and an increasingly growing long-term care system. This shift in the young-old demographic ratio will lead to fewer younger individuals taking care of more older individuals. Due to the enormous scale of this problem, its exponential nature, and its global scale, it is likely to become one of the most significant challenges of the current and future generations.

If no solution is found to this demographic dis-adjustment, this could lead to one of the most outstanding achievements in human history, the sharp increase in life expectancy, to become one of its worse failures. Future generations of older individuals could be condemned to receiving insufficient care and support to confront natural old-age related health problems, suffer financial deprivation, and experience other hardships. These problems are already a dramatic reality in the present moment, but the previously described trends could escalate this issue to a whole new level. The most devastating effect would be experienced in low- and middle-income countries. These are predicted to experience the sharpest decrease in fertility rate, they have higher marginal gains in life expectancies, and already struggle to fulfil the basic needs of older individuals (Brodsky *et al.*, 2002; Dutt, 1998). In such societies, older individuals had typically entirely relied on support by family members, while the transition towards urbanisation, migration, and women empowerment are depriving them of that. Given that this demographic trend seems unstoppable, there is the urge to improve knowledge regarding old-age related problems, to minimise their impact, and to smoother the change into societies with higher life expectancies, but with adequate quality of life for all its members.

This thesis follows this line of arguments, acknowledging that adapting to these demographic changes is one of the most urgent challenges of contemporary history. It explores three different old-age related problems and finds preventive solutions that could mitigate their impact. These problems are addressed in the form of three independent essays that consider independent stages of the life-cycle of individuals. Each essay evaluates a highly relevant old-age related problem using empirical evidence to validate its hypothesis. Different data sources and analysis methods address these questions and shed some light on these challenges. This thesis aims to understand these problems better to help policymakers, private enterprises, and single individuals take preventive measures to minimise old age-related problems. This improved understanding aims to contribute to smoother the transition to the demographic change of an ageing population.

2 Prevention throughout life-cycle

The different essays examine independent research questions and outcomes, but all of them consider the life-cycle of individuals as a linear process formed by distinct stages from birth until death. Each of these stages greatly influences old-age related outcomes, and therefore preventive measures should be taken on each of

them. Even before individuals are born, when they are in-utero, preventive behaviours that minimise old age-related problems have to be implemented. The environment of individuals has to be protected at that early stage since there are determinants that can already leave their print on them. These can lead to detrimental health effects that can extend until an older age. Similarly, their environment has to be protected after birth, at early childhood. During the young- and adult-life, healthy lifestyle practices have to be implemented to minimise detrimental outcomes at an older age. At late adulthood or older age, preventive behaviours still have to be sustained to optimise the welfare of individuals until their death. Each essay presented in this thesis addresses a vital stage of the life-cycle of individuals.

2.1 Essay 1

The first essay explores if very short-term events of extremely high air pollution can cause long-term health effects that can extend up to older adulthood (ages 47–70) and if socioeconomic status (SES) can compensate for them. For this aim, the Great London Smog of 1952 (GLS) is examined. It was a 5-days severe air pollution event that occurred in London in 1952 and is estimated to have caused 4,000 deaths in the week following it and 12,000 deaths in the following year (Bell *et al.*, 2004; Bell and Davis, 2001; Wilkins, 1954). Data is sourced from the UK Biobank, which includes self-reported, objective, and matched administrative health information on a large sample of people. By comparing the health of exposed and non-exposed people, conceived before and after the event, it is found that the GLS significantly impaired adulthood respiratory health. Using a difference-in-difference (DID) methodology, it is shown that the GLS caused individuals exposed to the GLS in-utero to be 0.08 standard deviations more likely to suffer respiratory health problems at older adulthood, while it caused individuals exposed during the first trimester of gestation to be 0.19 standard deviations more likely to suffer respiratory health problems. The effect had an enormous SES gradient, suggesting that there could have been large differences in the compensatory behaviours of parents and the environment in which individuals developed. This large gradient also suggests that the effect captured is a lower bound, since the UK Biobank is a non-representative sample and is biased towards high SES individuals. The effect on respiratory health developed at young age and extended towards older adulthood. Supplementary analysis suggests that the adulthood effects were carried over from childhood rather than caused by behavioural effects that created adulthood differences in exposure to pollutants, such as in smoking behaviours or exposure to residential or workplace air pollution. This essay provides comprehensive evidence that early life exposure to short-term pollution events can cause significant life-long health problems. It has implications for public health policies and individuals behaviour towards air pollution.

2.2 Essay 2

The second essay explores whether cognitive work activities involved in individuals' most prolonged lifetime occupations could affect their likelihood of suffering dementia at an older age (ages 50–85). The main hypothesis of this essay is that individuals working in occupations with high levels of cognitive work activities build up their cognitive reserve throughout their life cycle, which protects them from suffering dementia at an older age. Data from the Health and Retirement Study (HRS) is analysed to test this hypothesis, which for a nationally representative sample of older individuals in the US contains information about cognitive ability over time and occupation histories. This information is combined with the O*NET database, which has detailed information about the work activities performed at many occupations. A factor capturing the level of cognitive activities involved in individuals' most prolonged lifetime occupation is generated, and its relation with dementia is estimated, running multivariate linear and non-linear regression models. The main findings show that having had the most prolonged lifetime occupation with high loads in cognitive activities reduces the probability of dementia at an older age. These findings are robust to demographic, education, occupation, industry, work context and genetic controls. Genetic predisposition towards dementia does not affect the results. Using the panel structure of the HRS and running models with individual-level fixed effects, it is also found that decline towards dementia is affected by cognitive work activities.

2.3 Essay 3

The third essay explores the relationship between age-related cognitive deterioration with risky and non-risky financial investments. The main hypothesis of this essay is that individuals' investments in risky and non-risky financial investments are determined by their levels of cognition and cognitive deterioration. Data from the English Longitudinal Study of Ageing (ELSA) is analysed to test this hypothesis. This data is a nationally representative and longitudinal sample of older individuals, containing information about cognitive ability and ownership of financial investments. Its longitudinal nature makes it possible to measure changes in cognition and financial investments over time. Multivariate regression models with and without individual fixed-effects show a strong and positive relation of cognitive level with risky and non-risky financial investments. Individuals with higher levels of cognition invest more in risky- and non-risky financial investments, while old age-related cognitive deterioration only affects risky financial investments, reducing them. There are no significant differences in reactions to stock-market fluctuations due to the level of cognition. These results show that financial investments of individuals differ significantly due to their levels of cognition and cognitive deterioration.

3 Conclusions

This thesis has explored potential paths on how to minimise old-age related problems through preventive actions during the whole life-cycle of individuals. The demographic shift towards an ageing population is one of the most significant contemporary challenges, and this thesis examines three problems that are considered particularly relevant due to their scale, exponential nature, and gaps in the literature. This analysis is done in three empirical essays that combine economic theory with data analysis to shed light on these problems. Since these essays are independent, this thesis does not have a final chapter of conclusions or discussion. Each essay has an independent conclusion and discussion section that focuses on the specific problems considered. For the same reason, the different essays can be read without an order of preference. The chosen structure shows a continuous line of preventive behaviours that should be taken from the moment individuals are in gestation until old age, but the problems considered differ in their nature. It is shown that at each stage of the life-cycle of individuals, it is crucial to take preventive actions to minimise old-age related problems.

The first essay estimates the causal, long-term effects of a very short-lived event of extreme air pollution in-utero or early childhood on respiratory health at an older age. Individuals exposed to air pollution in-utero or at early childhood have worse respiratory health outcomes at an older age. The second essay explores the relationship between cognitive work activities and dementia. It shows that sustaining cognitive intensive work activities during the whole labour-life of individuals protects them from suffering dementia at old age. The third essay examines the effect of natural old-age related cognitive decline and investments in risky and non-risky financial investments. Individuals have to be protected when they start suffering old-age related cognitive deterioration to optimise their wealth throughout old age.

These unrelated essays converge in emphasising the great importance of prevention to address the challenges of an ageing population. They show that prevention has to be promoted during the whole life-cycle of individuals before they are born until their death. It seems impossible to stop the current demographic trend, but there is sufficient time to reduce its impact and minimise related problems. This thesis provides empirical evidence on different fields where this should be driven. Many other essential outcomes and scenarios could have been explored due to the broadness of this field. Because of time limitations, only the previously mentioned ones have been considered, with their selection having been carefully scrutinised. The problems exposed in each essay have been chosen due to their deep impact in terms of their economic cost, their exponentially growing nature, and the existence of gaps in the literature that made knowledge around them incomplete. This thesis provides theoretical and empirical evidence about them and improves their understanding.

Preventive measures can significantly reduce the impact of these problems. Research can identify new problems and point at new ways to solve them. This is done in this thesis, which analyses three extremely relevant old-age related problems and offers potential ways to prevent them. As mentioned in the historical perspective, the demographic change to an ageing population is a highly worrying problem and could transform one of the most outstanding achievements of human history into an enormous failure. Nevertheless, there is still time to avoid this. With the effort of researchers and the implication of policymakers, private enterprises, and single individuals, this dramatic scenario can be avoided.

References

BELL, M. L. and DAVIS, D. L. (2001). Reassessment of the lethal London fog of 1952: novel indicators of acute and chronic consequences of acute exposure to air pollution. *Environmental Health Perspectives*, **109** (suppl 3), 389–394.

—, — and FLETCHER, T. (2004). A retrospective assessment of mortality from the London smog episode of 1952: the role of influenza and pollution. *Environmental Health Perspectives*, **112** (1), 6–8.

BRODSKY, J., HABIB, J., HIRSCHFELD, M. and SIEGEL, B. (2002). Care of the frail elderly in developed and developing countries: the experience and the challenges. *Aging Clinical and Experimental Research*, **14** (4), 279–286.

DUTT, D. (1998). Care for the growing number of elderly people in developing countries needs to be addressed. *British Medical Journal*, **316** (7141), 1387.

FERNANDEZ, R. M. (2019). Health and GDP. Springer.

FUND., I. M. and FUND., I. M. (2000). *World economic outlook, May 2000*. International Monetary Fund Washington, DC.

MADDISON, A. (2000). *Economic Progress: The Last Half Century in Historical Perspectice*. Academy of the Social Sciences in Australia.

OECD (2021). Life expectancy at birth (indicator). Accessed: 2021-03-02.

STAFF, I. (2000). Globalization: threat or opportunity. *Tratto il giorno Settembre*, **27**, 2016.

THE WORLD BANK (2020). Life expectancy at birth, male (years) - least developed countries: UN classification. https://data.worldbank.org/indicator, accessed: 2021-03-02.

TREASURY, A. *et al.* (2001). Global poverty and inequality in the 20th century: turning the corner? *Economic Roundup*, (1), 1–52.

UNITED NATIONS, DEPARTMENT OF ECONOMIC AND SOCIAL AFFAIRS, POPULATION DIVISION (2015). World population prospects: the 2015 revision. United Nations: New York. http://www.un.org/en/development/desa/population/publications/dataset/fertility.shtml., accessed: 2021-04-02.

UNITED NATIONS, DEPARTMENT OF ECONOMIC AND SOCIAL AFFAIRS, POPULATION DIVISION (2017). World fertility report 2015 highlights. https://www.un.org/development/desa/pd/content/world-fertility-report-2015-highlights, accessed: 2021-03-02.

WILKINS, E. (1954). Air pollution aspects of the London fog of December 1952. *Quarterly Journal of the Royal Meteorological Society*, **80** (344), 267–271.

WORLD HEALTH ORGANIZATION (2020). Global health estimates: Life expectancy and leading causes of death and disability. https://www.who.int/data/gho/data/themes/mortality-and-global-health-estimates, accessed: 2021-03-02.

Essay 1: Long-term health effects of a short-run pollution shock

1 Introduction

Air pollution is one of the most prevalent global hazards, with nine out of 10 individuals worldwide being exposed to high air pollution levels that can cause severe respiratory diseases (World Health Organization, 2018). Meteorological conditions cause high variability in air pollution levels (Yang *et al.*, 2015), and therefore even very polluted areas suffer high variability in air quality levels, ranging from healthy towards extremely hazardous. This can lead to events of extreme air pollution in developing countries, such as that experienced by New Delhi in November 2019, when the city recorded a week of the worst air pollution levels ever recorded in human history. The city had to shut down completely, and planes could not land due to visibility problems (Xu and Xu, 2020). Developed countries have overall healthier air quality levels, but natural disasters or other extreme events can also cause pollution peaks. An example of this was the Australian bushfires of 2019, with Melbourne experiencing overnight one of the highest air pollution levels in the world. From having air pollution levels considered to be healthy, on Tuesday 14th January 2020, pollution levels raised to the 'worst of the world' (Australian Associated Press, 2020). Climate change and global warming will shift towards more dramatic climatic conditions and this will cause an increase in the number of days with extreme air pollution levels (Archer *et al.*, 2019).

Air pollution is directly linked with economic activity and industrialization (Kubiszewski *et al.*, 2013). This fact is especially true in the case of developing countries where pollution rates are rising most dramatically. Therefore air pollution is the indirect product of development, which is closely linked with welfare and well-being. Still, air pollution on its own can cause many hazardous effects on health outcomes such as pneumonia, stroke, ischaemic heart disease, chronic obstructive pulmonary disease (COPD), lung cancer (World Health Organization, 2018), diabetes (Andersen *et al.*, 2012), dementia (Bishop *et al.*, 2018; Carey

et al., 2018), cardiovascular diseases (Ha *et al.*, 2007), and on economic outcomes such as educational attainment (Roth, 2017) or labour productivity (He *et al.*, 2019).

While it is well known that exposure to air pollution can cause short-term adverse outcomes, much less is known about the long-term effects of short-term pollution shocks and about the capacity of individuals to recover from such events (Almond and Currie, 2011). It is crucial to determine if individuals only suffer short-term effects or if these effects: extend over time; are triggered in older adulthood; or are carried throughout an entire life cycle. Compensatory or environmental measures that could compensate for these effects are also considered.

This essay asks if short-term events of extreme air pollution can cause long-term health effects that could extend up to older adulthood and if Socioeconomic Status (SES) could compensate for them.

To shed light on the issue, the Great London Smog (GLS) of 1952 is examined. The GLS was an episode of severe air pollution that affected London for five days of December 1952. Retrospective assessment has attributed 4,000 deaths in the week following that event and 12,000 deaths in the year following that event (Bell *et al.*, 2004; Bell and Davis, 2001; Wilkins, 1954). Therefore the GLS had immediate and extremely hazardous health consequences. In the case of pregnant women, air pollution could have reached the placenta (Bové *et al.*, 2019) and affected the development of the fetus. There is consensus about the development of the lungs being one of the most affected organs by exposure to air pollution in-utero (Korten *et al.*, 2017; Bettiol *et al.*, 2021; Johnson *et al.*, 2021).

This essay's main hypothesis is that individuals who experienced the GLS in-utero or at early childhood could have suffered long-lasting detrimental respiratory health effects and that SES could have modulated the magnitude of these. To test this hypothesis, respiratory health outcomes of exposed and non-exposed individuals, conceived before and after the GLS are compared using a difference-in-difference (DID) specification. Individuals exposed to the GLS were born in London and in-utero or early childhood during that event, while individuals non-exposed to the GLS were born in the rest of England or conceived after that event. The research is made feasible by the unique data from the UK Biobank. From 2006 to 2010, 502,505 individuals aged 40 to 69 completed detailed surveys, underwent health assessments, provided saliva and blood samples, and agreed to have have their health records matched. The large sample size means that there are detailed health records on 43,823 individuals born from December 1950 to August1953 and March 1954 to July 1956. This allows for relatively precise estimates on a range of respiratory health outcomes such as: hospitalizations due to respiratory causes; medical diagnosis as to ever having suffered respiratory health problems; consumption of prescription drugs for respiratory health; and spirometry measurements.

The results show that individuals exposed to the GLS in-utero had significantly worse respiratory health throughout adulthood. Being exposed to the GLS in-utero increased the value of an indicator that measures the extent to which an individual suffers respiratory health problems throughout adulthood by 0.08 standard

deviations, while being exposed to the GLS at age one increased it by 0.1 standard deviations. The size of this effect is equivalent to the mean difference in the indicator of respiratory problems between individuals regardless of their exposure to maternal smoking around birth; it is three times as large as the mean difference between individuals with or without a college or university degree; and it is twice as large as the mean difference between being a male or a female.

The most sensitive period of exposure was the first trimester of pregnancy. Experiencing the GLS during that development period increased the value of the respiratory health problems indicator at an older age in 0.19 standard deviations. Considering heterogeneity effects by gender and SES, the effects were most substantial for males born in low SES boroughs. These effects are statistically larger compared to low SES females, high SES males, and high SES females.

The results are robust to many alternative specifications considering different control groups and even variation within London. The effect of the GLS on other health outcomes is also explored, and it shown that even if some other health outcomes were affected as well, the most substantial effect was on respiratory health. It is also ruled out that the effect of the GLS affected individuals' health through some indirect channels. The GLS did not affect the probability of individuals being exposed to risky environments or taking risky behaviours throughout their life cycles, that could have caused respiratory health problems on their own. There is no evidence of exposure to the GLS increasing the probabilities of living in areas with high levels of air pollution, smoking, or holding occupations subject to exposure to air-born contaminants.

This paper contributes to the literature of the 'fetal origin hypothesis' (Almond and Currie, 2011). This literature argues that the most critical period of human development is the gestational phase. During that period, the main organs are developed, and any disruption can have very long-lasting effects. Several hazardous conditions have been explored in this context, such as exposure to maternal stress (Aizer *et al.*, 2016), alcohol and tobacco consumption (von Hinke Kessler Scholder *et al.*, 2014; Cupul-Uicab *et al.*, 2012), or different types of pollutants (Almond and Currie, 2011). The later literature is the most relevant for this essay, and particularly the papers considering the long-run effects of exposure to pollution in-utero (Singh *et al.*, 2019; Sanders, 2012; Almond *et al.*, 2009; Bharadwaj *et al.*, 2017; Ferrie *et al.*, 2011; Isen *et al.*, 2017; Nilsson *et al.*, 2009; Black *et al.*, 2013). The pollutants considered include: air pollution; radiation; and water pollution. The outcomes are: different health and cognition measures; academic achievements; and labour force status. There is robust evidence about different pollutants having a detrimental effect on all these outcomes. The size of these effects is economically significant. The longest run effects reported are for labour outcomes at age 30 (Isen *et al.*, 2017). It remains unclear if short term events of extreme air pollution levels could have similar long-run effects to the pollutants considered in this literature.

Only two papers so far have considered short-term events of extreme pollution (Bharadwaj *et al.*, 2016; Ball, 2018b). Both of them also explore the GLS and estimate its long-term effects on health and economic

outcomes. Bharadwaj *et al.* (2016) show that the GLS had detrimental effects on childhood health outcomes but that it did not have significant effects in adulthood. Ball (2018b) shows that exposure to the GLS at childhood had significant long-run effects on health outcomes, while exposure to the GLS in-utero had no significant long-run effects. These non-significant long-run effects for exposure to the GLS in-utero could be driven by individuals' health recovering over time, but they could also be driven by some limitations of these papers in terms of sample sizes and noisy outcomes. The analysis of Bharadwaj *et al.* (2016) only includes 42 individuals exposed by the GLS in-utero, and the outcome considered is a self-reported measure of ever having suffered asthma. Ball (2018b) has an indicator of having had cancer as an outcome, which is very broad health outcome, and could be capturing very diverse health conditions. This essay contributes to both papers by analysing data from the UK Biobank, which has an extensive set of high-quality health variables for a large sample of individuals.

2 Context

2.1 Air-pollution, health and economics outcomes

Air pollution is a combination of solid particles and gas in the air generated from burning fuels in industrial activities, daily consumption and similar sources, and pollen, mould, and similar naturally produced processes. The Clean Air Act (CAA) of 1970 recognised six common air pollutants that can have detrimental effects on human health (Currie and Neidell, 2005). (1) Particulate matter (PM) refers to tiny particles from ash, dust, and the exhaust of motor vehicles. Its most minute elements can get captured in the lungs and generate respiratory problems. (2) Carbon monoxide (CO) is a poisonous gas with no colour or odour and is generated by fuel combustion in industrial processes, vehicle exhausts, or natural events such as bush fires. Its concentrations are highest in cold weather. (3) Ozone (OZ) is a gas that is not directly emitted to the air but results from reactions of nitrogen oxides and volatile organic compounds. (4) Sulfur dioxide (SO2) is a gas that has no colour and is generated by burning coal and oils. Under the right circumstance, sulfur dioxide transforms into tiny droplets of sulfuric acid, which is particularly hazardous for human health. (5) Lead (Pb) is a toxic metal that can be introduced into the air through industrial activities. (6) Nitrogen dioxide (NO_2) is an oxide of nitrogen that gets into the air by burning fuel.

These substances cause respiratory health problems when inhaled and pass to the blood system. In pregnant women, they can reach the placenta once they are in the blood system. Bové *et al.* (2019) found black carbon in the placenta of pregnant women, with its load being positively correlated with residential black carbon exposure during pregnancy. Currently, it is still unknown how those pollutants affect the development of the fetus (Korten *et al.*, 2017). There are two main theories. (1) Pollution crosses the alveoli

and placental barriers, directly affecting the fetus. (2) Pollution generates a systemic immune reaction of the mother, which leads to a decrease in the supply of nutrients and oxygen to the fetus.

Regardless of the mechanism, there is consensus about air pollution affecting the development of the fetus. The development of the lungs and its subsequent respiratory health problems have received the most attention so far (Korten *et al.*, 2017; Bettiol *et al.*, 2021; Johnson *et al.*, 2021), although there is a growing literature considering other outcomes such as cognition and metabolic dysfunctions. Regarding respiratory health, a strong positive association has been found between air pollution and chronic respiratory problems.

Air pollution can affect the lungs at different phases of development. These are defined as: (1) the Embryonic phase (zero to eight weeks of gestation), where the first differentiation of lung tissues occurs; (2) the pseudo glandular phase (nine to 16 weeks of gestation), where the air-conducting bronchial tree is formed; (3) the canalicular phase (16 to 24 weeks of gestation), where the branching of the respiratory portion of the lung from the terminal bronchiole occurs; (4) the saccularm phase (24 to 36 weeks), where airways and blood vessels are formed along with the conducting airways and related blood vessels; (5) the alveolar phase (36 weeks to two years after birth), where the alveoli develop.

Because fetuses and newborns are at a crucial moment of development, any disruption at that stage can have a long-lasting impact. This impact can be extended throughout the whole life-cycle of individuals until old age. Therefore it can affect their current health and also their potential development. It remains unclear if they can recover from that initial exposure to extreme air pollution in-utero.

This essay confirms the existence of such long-term effects, and it finds the most prominent effects for exposure to air pollution at the first, second, and fifth developmental phases of the lung. The most substantial effect is found at the first and second phases of development when the placenta protects the fetus, but the lungs are at an early developmental stage. However, a strong effect is also found in the first year of life, at the fifth development phase. At this stage, the baby is still developing its lungs and is not protected by the placenta.

Such gestational health effects can have a very substantial long-run impact on economic outcomes, since according to the theories of dynamic complementarity or self-productivity (Conti and Heckman, 2012; Cunha and Heckman, 2007), early conditions and environments are crucial for individuals' ability to learn and they also affect the productivity of later years. Considering development as being composed of many stages, skills produced at one stage can raise the productivity of investment at subsequent stages. As such, complementarity leads investments at different stages to bolster each other. Exposure to pollution in-utero could have had the opposite effect hindering development at a very early stage, reducing the return of investments at any other further stage.

2.2 Historical context GLS

The GLS was an event of extreme air pollution that occurred in London from 5th September (Friday) to 9th September (Tuesday) of 1952. London was the world's largest city at the time, with eight million inhabitants. Most of them used open coal fires and burned low-quality coal. As an anticyclone developed featuring a distinct lack of wind, a cold-weather blanket swept across the continent and became stationary over the capital. The cold weather increased the emission of pollutants, and those got trapped under the atmospheric anticyclonic conditions causing a thick ground-level layer of smog, also called a 'pea-souper' (Bell *et al.*, 2004). It was a short-term event that only lasted five days, and it was generated mainly due to meteorological conditions that were unpredictable at the time.

During each day of the GLS, 1,000 tonnes of smoke particles, 2,000 tonnes of carbon dioxide, 140 tonnes of hydrochloric acid, and 14 tonnes of fluorine compounds, and 370 tonnes of sulfur dioxide were emitted (metoffice.gov.uk, n.d.). The emissions of sulfur dioxide were converted into 800 tonnes of sulphuric acid, which is argued to have been the most dangerous pollutant (Bell and Davis, 2001). Other measures show that during the days of the GLS, black smoke concentrations rose from 0.49 milligrams per cubic metre (mg/m^3) to 4.46mg/m^3 and sulphur dioxide rose from 0.41mg/m^3 to 3.83mg/m^3. As wind speed rose, by 7th and 8th of December, smoke and SO_2 dropped to 1.22mg/m^3 and 1.35mg/m^3. In comparison, the previous December mean smoke concentration among 12 different sites remained between 0.12mg/m^3 and 0.44mg/m^3 (Mayor of London, 2002).

The smog led to a significant reduction in visibility, disrupting transport in the city and caused the cancellation of some sports events. Still, it generated no panic overall since Londoners were used to smog episodes of lower intensity and were not fully aware of how serious the situation was until the following week. According to some reports of that time, individuals stayed in their homes, which generally were not well-sealed from outdoor air pollution, or just continued with their everyday lives (Ball, 2018b).

The surprise came the week following the event when an excess mortality of 4,000 individuals was reported as being directly linked to air pollution (Logan *et al.*, 1953). Current research raises the mortality caused by the GLS to 12,000 individuals that died the following year (Bell *et al.*, 2004)[1]. These statistics shed some light on the true impact of the event and had great repercussions. The public awareness of the impact of this event led to the implementation of the Clean Air Act (CAA) of 1956, which prohibited the emissions of dark smoke. This policy was very effective, and 70% of the decline in infant mortality from 1957 to 1973 has been attributed to it (Nanna, 2020). Figure 1 presents a scheme of these different events.

[1] Harrison (2006) finds consistent results regarding mortality in the months after the GLS, and 59-76% of these deaths were related to respiratory diseases (Mayor of London, 2002).

Figure 1: Scheme individuals born before-after the GLS

Source: Own elaboration.

The literature suggests that the GLS did not discriminate by SES and that high levels of air pollution were not only present at financially deprived boroughs (Ball, 2018b; Wilkins, 1954; Mayor of London, 2002). An example of this is that the most affected areas were Chelsea and Kensington, two of the wealthiest areas in London at the time, and South East, one of the most deprived areas at the time .

The air pollution levels of the GLS were very similar to the ones that are experienced in developing countries on regular basis. For instance, Wang *et al.* (2016) found that the Chinese megacities like Xi'an and Beijing suffer pollution peaks very similar to the GLS in terms of pollutants and intensity of the emissions.

3 Literature Review

Due to the broadness of this research field, in the following review, only papers providing causal effects of the exposure to pollution in-utero or early childhood are discussed[2]. A summary of these can be found in Table A.1 in the appendix.

[2] A strong association has been found between the exposure to air pollution in-utero (Leon Hsu *et al.*, 2015; Clark *et al.*, 2010; Phillips *et al.*, 2018; Farmer *et al.*, 2014) or early childhood (Shankardass *et al.*, 2009; James Gaudermann *et al.*, 2000; Gehring *et al.*, 2015; Brauer *et al.*, 2007; Farmer *et al.*, 2014) on respiratory health during childhood. These papers are not discussed in detail since they only find associations and not causal effects. Air pollution could be capturing the variability of other variables affecting respiratory health. Korten *et al.* (2017), Bettiol *et al.* (2021), Johnson *et al.* (2021) offer a summary of this literature.

3.1 Literature GLS

Only two papers have explored the long-term effects of being exposed to the GLS in-utero or at early childhood (Bharadwaj *et al.*, 2016; Ball, 2018b).

Bharadwaj *et al.* (2016) analyse the effect of having been exposed to the GLS in-utero or at early childhood on asthma prevalence during childhood and adulthood. They use the English Longitudinal Study of Ageing (ELSA), which has information about individuals' place and date of birth and self-reported information about suffering asthma. The methodology employed is a DID strategy that considers individuals born in London as treated and individuals born in the rest of England as non-treated, and the time frame considered goes from 1945 to 1955. Their results show that exposure to the GLS in-utero or during the first year of life strongly affects childhood respiratory health. Being exposed to the GLS increases the likelihood of suffering asthma during childhood by 19.87 percentage points. No long-run effects are found, and being exposed to the GLS has no significant effect on asthma prevalence as an adult. These non-significant effects can be driven by the small sample size (42 individuals exposed to the GLS in-utero and 15 individuals exposed at age one or younger) and the likelihood of a large reporting error posed by the self-reporting nature of the outcomes.

Ball (2018b) estimates the effect of exposure to the GLS in-utero or early childhood on miscarriage and childhood mortality (short-run effect); educational attainment; labour outcomes; and cancer prevalence (long-term effect). The data analysed comes from the Office of National Statistics Longitudinal Study for the years 1971, 1981, 1991, 2001, and 2011 (42,000 total observations and an average of 730 individuals born in London each year) and a DID strategy similar to the one of Bharadwaj *et al.* (2016) is used. Individuals born in London are considered treated and individuals born in the rest of England as non-treated, and the time frame goes from 1950 to 1958. Their results show that the GLS reduced the size of the affected in-utero cohort by 2%. It also reduced the likelihood of holding a degree-level qualification by 3% for individuals affected by the GLS in-utero, and monthly hours worked in 1971 and 1991 diminished by eight hours for the same group. In addition, individuals exposed to the GLS as infants were 3% more likely to develop cancer before 2011, while no effect is found for individuals exposed to the GLS in-utero. This ambiguous health result could be driven by the broadness of the cancer outcome, which captures many diverse health conditions.

In both papers, the long-term effects on health outcomes are not statistically significant. Bharadwaj *et al.* (2016) found strong effects of the GLS on childhood asthma prevalence outcomes but was not able to confirm those for older adults. Ball (2018b) found substantial and statistically significant effects for economic outcomes but less clear effects for cancer. The most substantial effects on cancer are found for exposure to the GLS at age one, and this is not consistent with the literature (Almond *et al.*, 2009), which

shows that the most vulnerable period of human development is the gestational phase and particularly the first trimester. This could be driven by an absence of long-term effects or by some limitations of these papers, such as small sample sizes (Bharadwaj *et al.*, 2016) or noisy health outcomes (Bharadwaj *et al.*, 2016; Ball, 2018b). This essay contributes directly to these points considering a set of high-quality health outcomes and the largest sample considered so far.

One further paper considering the GLS is that of Hanlon (2018), which explores the short-term effect on mortality of smog events in London from 1866 to 1965. These smog episodes in London include the GLS, and the primary outcome is mortality at the time of the events for all age groups (including the gestational period). Analysing mortality data from Weekly Reports generated by the Registrar General's General's office, indicates that these extreme air pollution events caused one out of every 200 deaths in London during that time. The paper focuses on the short-term events of extreme air pollution and finds a substantial effect on mortality, which is consistent with the results of Logan *et al.* (1953); Bell *et al.* (2004). This essay expands the results of Hanlon (2018) in that it considers if individuals surviving the GLS suffered respiratory health problems into older adulthood.

3.2 Literature on the short-term effect of exposure to air pollution in-utero

Other papers have explored the effects of being exposed to other pollutants in-utero or early childhood making use of the variability in wind directions, natural disasters, or the gradual implementation of environmental policies. Most of these papers have estimated the short-term effects of pollution in-utero or early childhood on fetal and early childhood mortality (Agarwal *et al.*, 2010; Beach and Hanlon, 2018; Chay and Greenstone, 2003; Clay *et al.*, 2016; Currie and Schmieder, 2009; Currie *et al.*, 2015; Jayachandran, 2009; Knittel *et al.*, 2016; Luechinger, 2014; Tanaka, 2015; Khawand *et al.*, 2015; Foster *et al.*, 2009) and birth weight (Altindag *et al.*, 2017; Currie and Walker, 2011; Yang and Chou, 2015; Vyas, 2019; Currie and Schwandt, 2016). Consistent and robust results show that exposure to high levels of pollution can increase the likelihood of suffering miscarriages, intrauterine death, and mortality at early childhood and can lead to lower birth weights.

3.3 Literature on the long-term effect of exposure to air pollution in-utero

Papers that consider medium- or long-term effects of pollution are of most relevance for this essay. These estimate the effects of exposure to pollution in-utero or early childhood on childhood or adulthood outcomes. Again, different outcomes are considered, such as height and weight (Rosales-Rueda and Triyana, 2019; Singh *et al.*, 2019), cognition, academic achievement (Rosales-Rueda and Triyana, 2019; Sanders, 2012; Almond *et al.*, 2009; Bharadwaj *et al.*, 2017; Ferrie *et al.*, 2011; Molina, 2021), earnings, labour force

status, and the average number of hours worked (Isen *et al.*, 2017; Nilsson *et al.*, 2009; Black *et al.*, 2013; Peet, 2016).

Singh *et al.* (2019) explore the effect of exposure to air pollution in-utero on child height and weight. They use an instrumental variable (IV) strategy relying on the upwind biomass of burning events in neighbouring areas. In addition, satellite PM2.5 data and the geo-coded Demographic and Health Survey of India (2016) is analysed. Their findings show that exposure to air pollution during the first trimester of pregnancy significantly reduces height-for-age by 6.7% and weight-for-age by 7.8%.

Sanders (2012) estimate the effects of exposure to total suspended particulate in-utero on high school test scores in the United States (US). They use county-level variation in the timing and severity of the 1980 recession in the US as an IV and analyse data from the monitored Texas Assessment of Academic Skills (TAAS) from 1994 to 2002. Their results show that a decrease in one standard deviation in prenatal total suspended particulate in a students year of birth increases high school scores by 6%.

Bharadwaj *et al.* (2017) examine the effects of exposure to pollution in-utero on high school test scores in Chile. They consider in-sibling variation to account for location sorting and time-invariant family characteristics. Using data from a national test run for every student in the country (SIMCE) from 2002 to 2010, they find that one additional standard deviation of exposure to CO in-utero is associated with a 0.036 standard deviation in fourth-grade math scores and a 0.042 standard deviation decrease in fourth-grade language test scores.

Isen *et al.* (2017) estimate the effect of a reduction in childhood exposure to pollution led by the CAA on labour force participation and wages at age 30. They use an IV approach and analyse data from the US Census Bureau'sBureau's Longitudinal Employer Household Dynamics for the years 1998 to 2007 and air pollution monitoring data from the US Environmental Protection Agency (EPA). It is shown that a 10% reduction lead by the CAA significantly increased earnings at age 30 in 1%.

Molina (2021) explores the effect of exposure to air pollution in Mexico. Air pollution is measured with thermal inversion, and data of the Mexican Family Life Survey is analysed. It is found that exposure to air pollution in the second trimester of gestation leads to significantly lower cognitive ability in adulthood for both genders but lower high school completion and income for women only. This paper highlights the role of gender in understanding the long-term effects of gestational insults.

Nilsson *et al.* (2009) study the effects of lead reductions due to the phasing of leaded gasoline in Sweden on high school Grade Point Average (GPA) and cognition test scores, high school completion, and educational attainment. The Swedish moss survey sample for the years 1975, 1980 and 1985 is used for the pollution measurements, and data from the Institute for Labor Market Policy Evaluation (IFAU) and military enrolment test scores in Sweden for the year 2004 is used for the educational, labour, and cognition outcomes. Estimating OLS models with municipality birth fixed effects, they find that reductions in lead

during the observation period reduced the probability of ending in the lower end of the GPA distribution by 3.3%, increased high school completion by 0.9%, and reduced the probability of suffering welfare dependency at ages 20 to 32 by 0.6 percentage points. There is an SES gradient favouring low SES children, who benefit most from the lead reduction.

Rosales-Rueda and Triyana (2019) estimate the causal effect of being exposed in-utero or early childhood to the Indonesian bushfires of 1997. They analyse data from the TOMS and the IFLS, which were collected three, ten, and 17 years after that event. Their results show that exposure to that event in-utero or early childhood reduced height and lung capacity, while cognitive functioning remained unaffected. The lung capacity of individuals recovered throughout age, and no significant effect was is found at age 17.

This literature has found large and significant causal effects of different sources of pollution on a diverse set of outcomes. The sizes of the effects on the health, educational, and labour outcomes are economically meaningful. Even if there is evidence about long term-effects, it remains unclear if individuals can recover from these over time. The longest-run effect reported considers labour outcomes at age 32 (Nilsson *et al.*, 2009). Given this limited time span, it remains unclear if these effects persist over age, or if individuals can eventually recover from them at older age. Another crucial point of the literature is that individuals with low SES suffer a more detrimental effect from exposure to air pollution in-utero, compared to individuals with high SES (Nilsson *et al.*, 2009). This gradient is explored at an individual level exploring parental earnings. Using residential segregation data it is shown that this gradient can not be explained by different levels of exposures due to SES. It is also shown that there are large differences in these effects by gender (Molina, 2021).

3.4 Literature on the long-term effect of exposure to other pollution sources in-utero

Other pollutants such as radiation and water pollution have also been considered by the literature (Black *et al.*, 2013; Almond *et al.*, 2009; Ferrie *et al.*, 2011). These papers are relevant for this essay since they use very similar identification strategies and methods. Still, their results are less relevant since different sources of pollution could affect individuals in a very different manner.

Black *et al.* (2013) estimate the effects of exposure to radiation at the period of gestation on IQ scores, education, earnings, and adult height in Norway. They explore variations driven by nuclear weapon testing during the 50s and 60s and analyse data from the Norwegian Registry Data and Norwegian military records (up to 2009). Their findings show that individuals exposed in-utero to radiation suffer a drop in their IQ score at age 18 of about 0.06 or one IQ point. Furthermore, there is an intergenerational transmission of 0.65, of this lower IQ.

Almond *et al.* (2009) study the effect of exposure to radiation in-utero on health and school outcomes in Sweden. They use variation in the exposure to Chernobyl's radioactive fallout and analyse administrative data on compulsory school records for cohorts born from 1983 to 1988. Their findings show that exposure to radiation in-utero does not affect birth weight; hospitalisations during the twenty years after the accident; neoplasms and diseases of the blood. However, they found that radiation exposure in-utero does affect academic achievement, leading to a significant 2.5 percentile drop in the grade distribution, a 6% decrease in mathematics scores, and a reduction in the likelihood of entering high school by 3%. Considering the parental level of education, they found that the reported effect is concentrated among parents with low levels of education.

Ferrie *et al.* (2011) consider the effect of lead plumbing and water chemistry on cognition test scores of US army enlistees during World War II. They analyse a 5% public use sample (IPUMS) drawn from the 1930 US Census of Population and the Army General Classification Test (AGCT). Their results show that higher exposure to water-borne lead is associated with lower test scores, with changes from pH 6 to pH 5.5 reducing scores in one-quarter standard deviation.

These papers present strong and robust effects of other sources of pollution on cognitive and academic outcomes. They confirm that exposure to other sources of pollution also can have strong long-term effects on economic outcomes. This literature also confirms that the effects of exposure to pollution in-utero are largest for individuals with low SES (Almond *et al.*, 2009).

3.5 Contributions to the literature

This essay contributes to the existing literature of the 'fetal origin hypthesis' (Almond and Currie, 2011), which estimates the causal, long-term effects of being exposed in-utero or during early childhood to short-lived air pollution peaks. A very robust and relevant health outcome is considered, and its SES gradient is explored.

It explores the long-term effects of in-utero stressful events. The GLS offers the perfect scenario since it was an intense short-term (five days) event conducive to a clean identification. Data from the UK Biobank is also ideal for this purpose since it combines administrative data, objective measures, and self-reported information for a large sample of individuals. This data makes it possible to estimate the long-term effects of the GLS, considering the variables of hospitalisations 47 to 70 years after the GLS and self-reported outcomes or spirometry measure 48 to 60 years after the GLS. This long term dimension shows that disruptive events in-utero can greatly impact individuals until into adulthood.

This essay considers if there are differences in the effect of the GLS due to SES or gender. It is shown that the effect is strongest for low SES males. Accordingly, SES and gender are good predictors of the impact of stressful events in-utero. These results highlight the inequality gap individuals are constricted

to from the moment they are born. It also suggests that gender is a crucial dimension to consider when exploring the 'fetal origin hypothesis'.

The event considered is also highly relevant since it is a short period (five days) of extreme air pollution. As mentioned in the introduction, such events occur regularly in developing countries and exceptionally in developed ones. However, little has been done so far regarding these short-term air pollution peaks. Finding robust evidence of large, long-term effects of such short time windows of air pollution can lead policymakers to target their interventions to these particularly harmful events.

4 Data

4.1 Main database – UK Biobank

For the empirical analysis, data from the UK Biobank is used. This data contains health and genetic variables for 502,505 individuals aged 40 to 69 living in the United Kingdom (UK). The initial enrolment took place from 2006 to 2010, when individuals went to an assessment centre where they completed an automated questionnaire; a verbal interview; several cognitive tests. Physical measures and biological samples were also taken. Although most of the UK Biobank sample only has information about that first enrolment in a cross-sectional manner, some of the participants have been followed over time, and additional information has been collected. In addition, there is detailed administrative data about hospitalizations for 87% of the UK Biobank sample. It captures individuals that had at least one hospitalization episode. There is also detailed administrative data about mortality for 6% of the sample, who passed away after the initial interview. This essay only uses the information of the first enrolment and the linked hospitalisations and deaths registries.

The UK Biobank is not a representative sample from the UK since the sampling is volunteer-based and over-represents high SES individuals. This could bias the results of this essay since the most substantial effect of the GLS is found for individuals with low SES. Having an over-representation of high SES individuals would reduce the size of the treatment average effect. As such, a lower bound of the actual effect of the GLS would be captured.

4.2 Outcomes – UK Biobank

This essay explores how the GLS affected respiratory health problems in adulthood. Respiratory health problems constitute a broad outcome composed of many conditions. As such, it is measured from the combination of different health outcomes. Individuals suffering from respiratory health problems are likely to receive a diagnosis by a doctor; in more extreme cases, they may even be hospitalised. They are also

likely to consume prescription drugs for respiratory health and to have worse lung capacity. These health indicators can be found in four respiratory health outcomes available in the UK Biobank as follows[3].

(1) Self-reported information about ever having been diagnosed by a doctor to suffer respiratory health problems is obtained through a touchscreen questionnaire and a verbal interview conducted by a trained staff member, both being completed at the assessment centre. In a first stage, the participants report in a touch screen if they ever were diagnosed by a doctor to having suffered: a heart attack; angina; stroke; high blood pressure; blood clot in the leg (DVT); blood clot in the lung; emphysema/chronic bronchitis; asthma or diabetes. In a second stage, the interviewer confirms with the participants this previously reported information, and if further details are required, the interviewer asks: "In the touch screen you selected that you have been told by a doctor that you have other serious illnesses or disabilities, could you now tell me what they are?". This information is coded into ICD-10 data, from which a dummy variable is generated, capturing if individuals ever had been diagnosed by a doctor as having suffered from respiratory problems.

(2) Hospitalisation data was obtained through linkage to external data providers and ranges from 1997 to 2020[4]. Inpatients are defined as individuals who are admitted to a hospital and occupy a hospital bed. These hospitalisations include both admissions where an overnight stay is planned and day cases. A dummy variable is generated from this information, capturing if individuals were hospitalised from 1997 to 2020 due to respiratory causes.

(3) Self-reported information about current consumption of respiratory prescribed medications is obtained through a touchscreen questionnaire and a verbal interview conducted by a trained staff member, both being completed at the assessment centre. In the first stage, the participants report on a touch screen if they currently are taking regular prescription medications. In a second stage, the interviewer confirms with the participants this previously reported information. If further details are required, the interviewer asks: "In the touch screen you said you are taking regular prescription medications. Can you now tell me what these are?". It considers prescription drugs taken regularly (taken weekly, monthly), while short-term consumption of medications is excluded. This information is coded into National Health Service (NHS) data, from which a dummy variable is generated, capturing if individuals consume prescription drugs for respiratory problems at the date of the interview.

(4) Data on breath spirometry is measured using a Vitalograph Pneumotrac 6800. Individuals record two to three blows, depending on the reproducibility of the first two, within six minutes. The z-score of the FEV1/FVC ratio (Tiffeneau-Pinelli index) is calculated. This is a measure used to diagnose obstructive and restrictive lung disease. This measure takes into account the gender, age, and height of individuals and

[3] These outcomes are described in Table B.2 in the Appendix

[4] For England, this data are obtained by the Access Request Service (DARS), managed by NHS Digital. The dataset is called Hospital Episode Statistics (HES) and Admitted Patient Care (APC). The HES comes from routine data from providers to the NHS Digital for payment and commissioning healthcare in England. This data are linked to 87% of the UK Biobank sample.

is restricted to white ethnicity (Gupta and Strachan, 2017). A dummy variable is generated, capturing if individuals are at the fifth percentile or lower of this ratio, which is a standard predictor of obstructive and restrictive lung diseases[5] (Pellegrino *et al.*, 2005).

Following Baranov *et al.* (2020); Janke *et al.* (2020), an indicator for respiratory health problems is generated by combining the four outcomes in a factor analysis[6]. Only one factor with an Eigenvalue above one is retained. A scree plot of these factors and their rotated loadings can be found in the Appendix in Figure B.1 and Table B.3. This factor is standardised with a mean zero and a standard deviation one. Higher values capture worse respiratory health in adulthood.

This indicator has two advantages to considering the individual health outcomes. First, it helps to interpret the results since one primary outcome is considered instead of four. This reduction helps to consider the big picture of respiratory health rather than focusing on individual outcomes. Second, it maximises statistical power, which helps capture the long-run causal effects of an event that occurred 50 to 70 years before measuring the outcomes. In any case, the results for the individual outcomes are also estimated and the results are highly consistent with the main results.

It has the disadvantage that it restricts the sample to observations that have non-missing values in all outcomes. Due to that, the self-reported information about ever having been diagnosed by a doctor to suffer respiratory health problems loses 32.6% of its observations, the hospitalisation data loses 18.7% of its observations, the self-reported information about current consumption of respiratory prescribed medications loses 32.6% of its observations, and the spirometry outcome loses 17.6% of its observations. Still, for the previously exposed arguments the instrument for respiratory health is preferred[7].

4.3 Control variables – UK Biobank

The year and place of birth of individuals are obtained through self-reported information. This information captures if individuals were born in London or the rest of England and before or after the GLS. Individuals are asked, "What is the town or district you first lived in when you were born?".

Individuals are given a pre-visit 'aide-memoire' questionnaire, which they are asked to complete at home before the interview, to improve the quality of the self-reported information. Then, when they attend the assessment centre, they can bring it to help them answer questions related to health history, prescription medications consumed, and birth details.

[5] Further details of this outcome can be found in Appendix C.
[6] The results are also highly consistent when an index is used that adds up the four outcomes (ranges from zero to four).
[7] Results for the largest sample for each individual outcome are provided in Table C.5 in the Appendix. The coefficients are similar in size and more significant to the ones with the restricted sample in Table 3.

An indicator of SES at the place of birth is generated from the mean age of completion of formal education in different counties or boroughs[8]. The average age of completion of formal education of individuals is taken at the county or borough level for a sample of individuals born from 1956–1966 (individuals born ten years after the primary sample). This SES indicator is split into the 25th percentile (low SES), 25-75 percentile (medium SES) and 75 percentile (high SES) of completion of formal education and merged at a county or borough level with the primary sample. This categorisation into SES groups is done for London and the rest of England separately[9]. Each county or borough in England has been assigned an SES based on the average age of completion of formal education.

4.4 Sample restriction – UK Biobank

The initial sample of the UK Biobank is composed of 502,508 individuals and observations. The sample is restricted to individuals born from December 1950 to August 1953 and from March 1954 to July 1956, which reduces it to 85,660 observation. Individuals born from December 1950 to August 1953 were in-utero or aged zero to one at the GLS. The sample is restricted to children younger than two at the GLS because this is an important milestone in human development (Iliff and Lee, 1952; Casale and Desmond, 2016; Manji *et al.*, 2015; Hamadani *et al.*, 2014). The sample of individuals conceived after the GLS goes from March 1954 to July 1956. It is limited to August 1956 because the CAA of 1956 was introduced, affecting respiratory health outcomes. The months from September 1953 to February 1954 are excluded from the sample to avoid a potential bias driven by short-term demographic changes that the GLS could have caused. Parents affected by the GLS could have changed their decisions about having children. Such short lived demographic changes after natural disasters or other extreme events have been extensively reported in the literature (Evans *et al.*, 2010; Rodgers *et al.*, 2005; Nandi *et al.*, 2018). It is argued that parents either need some time to recover from the economic and emotional impact of such extreme events, or if they experience miscarriage or child loss they might feel pressured to have another child. A scheme of this time frame can be seen in Figure 1, and more details are provided in the methodology section.

The sample is further restricted to individuals with non-missing information about their place of birth and born in England, reducing the sample to 64,639 individuals. Individuals born in England are more comparable to those born in London than individuals born in Scotland and Wales. The sample is also restricted to non-missing information in all respiratory health outcomes reducing it to 43.823 individuals

[8] Table C.1 in the Appendix shows that this indicator of SES is a good predictor of income level. This table suggests that the indicator is truly capturing SES

[9] The size of the percentiles is not identical, since the same counties and boroughs have values above and below a certain percentile. For example, some individuals born in the borough of Brent are above the 75th percentile and some individuals below it. As the SES is measured at the county level, taking the exact percentiles would assign some of these individuals to Medium SES and others to High SES, even if both were born in the same borough. Therefore, individuals born in the first county below the 75th percentile are categorised as High SES. An equivalent approach is taken with the other SES indicators. Therefore Low and High SES have a few observations more than a quartile, and Medium SES is a bit less than half of the sample.

and observations. This restriction makes it possible to construct the respiratory health problems indicator at older adulthood with complete information in each outcome area. However, it restricts the sample to individuals of white ethnicity since the estimations of the z-scores of the spirometry test are restricted to them (Gupta and Strachan, 2017). Table C.2 in the Appendix shows that the final sample is composed of 43,823 observations. Of these, 3,227 individuals were born in London and in-utero or aged zero to one at the GLS (*Treat*Pre*), while 2,418 individuals were born in London and conceived after the GLS (*Treat*Post*). Further, 21,223 individuals were born in the rest of England and in-utero or aged zero to one at the GLS (*Control*Pre*), while 16,955 individuals were born in the rest of England and conceived after the GLS (*Control*Post*).

4.5 Complementary databases – MERRA-2

Contemporaneous air pollution data are merged with the place of residency of UK Biobank respondents to explore the potential mechanisms of the GLS on respiratory health[10]. These air pollution measurements come from the MERRA-2, a NASA-driven database that combines satellite and weather stations information to generate hourly meteorological data at a global scale. This data goes back to 1980 and has detailed information about temperature, dust, and similar variables. PM2.5 data from 2005 to 2010 for the area of England is used to capture contemporaneous air pollution. This data are merged with the residency of the UK Biobank respondents at the time of the interview. The yearly average PM2.5 is taken to capture the average levels of air pollution at the year previous to the interview.

4.6 Complementary databases – O*NET

Information about the context of the main occupations of individuals is linked with the UK Biobank to explore the potential mechanisms of the GLS on respiratory health. This information regarding the context of work activities comes from the O*NET[11]. It is a database containing detailed information about the work context of many occupations in the US. Among others, it provides information about the exposure to contaminants in different occupations. These contaminants include pollutants, gases, dust, and odours. Given the high quality of the O*NET and the similitude of the UK and US labour markets, the O*NET has already been used by other papers in the UK context (Lekfuangfu and Lordan, 2018; Dickerson and Morris, 2019). The O*NET includes US SOC 2000 codes, while the UK Biobank has UK SOC 2000. The O*NET occupations in the highest decile of exposure to contaminants are manually cross-walked to the UK SOC 2000 to match both codes. This conversion generates a variable that captures the main occupation at the highest decile of exposure to air-born contaminants.

[10] The MERRA-2 database is independent from the UK Biobank and is linked by the author.
[11] The O*NET database is independent from the UK Biobank and is linked by the author.

5 Methodology

5.1 Identification

To capture the long-term causal effect of in-utero or early childhood exposure of the GLS on respiratory health, a slightly modified DID model is run. For a better understanding of this model, Figure 1 presents a scheme of the GLS scenario. This event occurred from the 5th to the 9th of December 1952. Therefore individuals born from December 1950 to the 4th of December 1952 were aged zero to one at the GLS. Individuals born from the 5th of December 1952 to August 1952 were in-utero during the GLS. Individuals born right after the GLS had been conceived many months before and were at a late uterine stage during that event and vice versa. Individuals born after August 1953 had not been conceived at the GLS. Considering the notation of a traditional DID setting, individuals born in August 1952 or before (*Pre*) were exposed to the GLS in-utero or early childhood. Individuals born after August 1952 (*Post*) were not exposed to that event. Individuals born in London were treated by the GLS, while individuals born in the rest of England were not treated[12]. From this assessment it can be inferred that the coefficient of interest in this scenario is *Pre*Treat*, while in a traditional DID setting this would be *Post*Treat*. Other than this conceptual difference, the model considered can be interpreted as a DID, and has the same identification power. The main specification is:

$$\begin{aligned} Y_i = \beta_0 + \beta_1 BornLondon_i + \beta_2 YearMonthBirthFE_i' + \\ \beta_3 BornLondon_i * InUteroEarlyChildGLS_i' + \beta_4 X_i' + \epsilon_i \end{aligned} \tag{5.1}$$

In this model, Y is the indicator of respiratory health problems. $BornLondon$ is a dummy variable equal to one for individuals born in London and zero for individuals born in the rest of England. $YearMonthFE$ is a vector of year-month of birth fixed effects, which captures if individuals were conceived before or after the GLS and time-dependent characteristics. This variable captures cohorts, seasonal and time fixed effects. $BornLondon_i * InUteroEarlyChildGLS'$ is a vector that includes the interaction between being born in London and being in-utero or early childhood at the GLS. β_3 is the coefficient of interest and captures the causal effect of in-utero or early childhood exposure to the GLS.

The vector X_i includes controls that could have affected the exposure of individuals to the GLS and later life outcomes. These include a categorical variable for gender and fixed effects for the year and month of the interview. Gender is included because of the existence of survival bias of in-utero events (Ball, 2018a), with females having higher survival rates compared to males (Buckberry *et al.*, 2014; Lyster, 1974). Gender affects selection in-utero and later-life health outcomes. A year and month interview fixed effects combined

[12] In Table C.3 other time windows are considered. The results show no major differences compared to the main specification.

with the year-month birth fixed effects are included to capture the age of individuals. Individuals born at an earlier stage are older and should present more health problems than those born later. Similarly, individuals attending the assessment centre at an earlier stage are younger and should present fewer health problems than individuals attending the assessment centre at a later stage. These time fixed effects also control for seasonal effects and time-dependent variables at the time of birth and the attendance to the interview centre. Other controls, such as education, wealth, and similar variables, could affect the health outcomes but are endogenous and could have been affected by the GLS. The GLS occurred when individuals were in-utero or early childhood and triggered individuals' education, wealth, and similar variables. Including these controls would capture the variability that is explored in this specification.

The interpretation of this model is that having been born in London and having been exposed to the GLS in-utero or early childhood causally increases the probability of experiencing respiratory health problems at older adulthood in β_3 standard deviations[13].

The causal and long-term effects of the GLS on respiratory health hold under the assumption that the GLS was exogenous and not related to other variables that could have affected it and that London and the rest of England had similar time-trends in their respiratory health before the GLS.

The first point holds since the GLS was an unpredictable event mainly driven by atmospheric conditions. As mentioned in the description of the GLS, its actual impact was not well known until one week later, and individuals in London were used to smogs of much lower intensity in the past. The literature also suggests that air pollution did not discriminate by SES (Ball, 2018b; Wilkins, 1954). Therefore, it affected individuals in London in an unpredictable and homogeneous manner[14].

The second condition is shown to hold in Figure 2. This condition is described in more detail in the results section.

Due to data limitations, these models do not explore the variation in air pollution levels during the GLS within London. Although some registers of the air pollution levels experienced during that event exist, these are very limited. There is only information about certain pollutants, and there are relatively few registers within London. For example, Figure C.1 shows that there are only registers available for 10 of the 32 boroughs of London. This limited information reduces the sample exposed by the GLS from 3,227 individuals to 1,184, of which only 324 individuals were exposed in-utero. Consequently, it greatly reduces the sample size and statistical power, making it impossible to estimate long-term effects. Therefore this

[13] This analysis considers overall exposure to the GLS and not exposure to different levels of air pollution. There is some information about air pollution levels within London during the GLS (Ball, 2018b; Wilkins, 1954), but it is constrained. A shortage of air measurement stations and missing information within these drives this limitation. For this reason, there is not enough variability to run an analysis considering different levels of exposure to air pollution. This limitation can be seen in Figure C.1, which shows that for most London, there were no data registers.

[14] Information about air pollution levels within London during the GLS is limited. A shortage of air measurement stations and missing information within these drives this limitation. No correlation is found between SES and air pollution, considering the limited available information.

essay does not focus on any particular pollutant, nor does it consider intensities of exposure. Instead, it examines short-term peaks of high-intensity air pollution as a single event and in a binary manner.

It can be hypothesised how the results would change if variation in air pollution levels within London could be explored. If the significant variation in air-pollution levels reported by Wilkins (1954) held for the rest of London, the average treatment effects reported in this essay would be capturing the average of different air pollution levels. As such, the reported results would only capture a lower bound, and the effects should be even more prominent for the boroughs most heavily affected by the GLS.

5.2 Biases

Even if the main DID conditions hold, some potential biases could have affected the identification strategy. In most cases, these biases can be solved, and if this is not possible, it can be hypothesised how they could be affecting the main results.

The GLS could have led to short-term demographic changes in the population, affecting the decision to have children. These changes could go in either directions. Individuals that had a miscarriage or suffered child loss could have experienced pressure to have another child. On the contrary, individuals could have been traumatised by the event or experienced some economic loss and so needed time to recover before having another child. Robust evidence exist for both scenarios finding demographic changes after natural disasters or other extreme events (Evans *et al.*, 2010; Rodgers *et al.*, 2005; Nandi *et al.*, 2018). To account for this bias, the main specification in this essay excludes individuals conceived six months after the GLS. Individuals born from December 1950 to August 1952 are considered in-utero or early childhood at the GLS, while individuals born from March 1954 to July 1956 are considered individuals conceived after the GLS. The months from September 1953 to February 1954 are excluded from the main sample since they could have been affected by the demographic bias[15].

Excluding these six months after the GLS also accounts for high levels of air pollution that persisted after the GLS (Mayor of London, 2002).

The CAA of 1956 could also have affected the respiratory health outcomes of individuals since it had a great impact on individuals' health (Nanna, 2020). Therefore the sample is restricted to August 1956 to exclude individuals born thereafter.

Some individuals exposed to the GLS in-utero could have experienced a long pregnancy and be born after August 1952. These individuals would not be considered in the main specification since the observations after August 1952 until March 1954 are excluded from the primary sample. Other individuals conceived after the GLS could have been born prematurely before August 1952. These would be erroneously classified

[15] Alternative specifications with different time frames are run in Table C.3 in the Appendix, and the results are highly consistent. The main specification of this essay is considered to account for this well-known demographic bias, and that it captures the true causal effect of the GLS.

as individuals having been exposed to the GLS in-utero. Both scenarios are rare, and it is reasonable to assume most pregnancies to have lasted nine months. Any modifications of the time windows considered would exacerbate this error instead of reducing it. Therefore, it is assumed that this error exists in the estimation, but the chosen time window minimises it. It can be hypothesised how the existing error affects the results. Because of this error, the group of individuals exposed to the GLS includes some individuals conceived after that event and not affected by it. These individuals should have healthier outcomes, and therefore the results present a lower bound.

Due to their deteriorated health, individuals exposed to the GLS may have been less likely to participate in the UK Biobank, creating a participation bias. As described in the data section, the UK Biobank is a non-representative sample from the UK, and it is volunteer-based. Individuals that suffered respiratory health problems due to the GLS could be less likely to participate in the study compared to healthy individuals. Their participation cost, such as travelling to the assessment centre, would be higher due to their health condition. This selection bias is confirmed in Table C.4 in the Appendix[16]. Individuals exposed in-utero or at early childhood to the GLS were less likely to participate in the study. The direction in which this bias affects the main estimates can be hypothesized. If the least healthy individuals affected by the GLS refused to participate in the UK Biobank, the group affected by the GLS and that participated in the UK Biobank should be biased towards healthier individuals. For this reason, the results of the main specification capture a lower bound.

There also is a survival bias driven by the GLS being the cause of miscarriages and reducing the size of the affected cohort (Ball, 2018a). If individuals who survived exposure to the GLS in-utero were fitter than those who did not, the sample exposed to the GLS would be biased towards healthier individuals. In such a case, the results would capture a lower bound of the effect of the GLS.

Another bias could be driven by individuals exposed to the GLS migrating out of London due to their deteriorated respiratory health. In such a case, these individuals would report being born in London, but they would attend an assessment centre outside London. To control for this, Table C.6 in the Appendix includes dummy controls for the different assessment centres and boroughs of birth. Again, there are no differences with the main results, suggesting that this potential bias is not a problem.

The rest of England could also have been affected by the smog of the GLS and not be a clean control group. Ball (2018b) and Mayor of London (2002) show that this is not the case and that during the time of the GLS, air pollution levels in the rest of England remained constant. Furthermore, consistent results are found using different control groups.

[16] Table C.4 estimates if the GLS had a main effect on participation in the UK Biobank. The outcome is the number of participants born in London and the rest of England at different years and months. The model is equivalent to the main specification of this essay, excluding the year and month of the interview. This model captures the effect of the GLS on participation in the UK Biobank.

After the GLS, air pollution levels in London remained high (Jon, 2015) but had a much milder effect on an individuals health. These high levels of air pollution limit the interpretation of the GLS, as it is impossible to compare individuals exposed to very high levels of pollution with those exposed to low levels of pollution. Instead, this scenario considers individuals exposed to high levels of pollution (not exposed to the GLS) and compares them to individuals exposed to very high levels of pollution (exposed to the GLS). When interpreting the results, it has to be kept in mind that these capture the effects of the GLS in the context of London at that time and not the effect of very high pollution compared to low pollution. It can be hypothesised that these effects would differ if the comparison group considered low levels of pollution. In such a scenario, the effect reported should be larger since individuals exposed to low pollution levels should present better health outcomes than those born after the GLS. Therefore, this essay identifies the effects of the GLS in the London context and a lower bound of the effects of exposure to very high pollution compared to exposure to low pollution.

6 Results

6.1 Main Results

The results of the main specification can be found in Table 1, which is divided into two panels that consider the effect of the GLS at different moments of development. Panel A estimates the effects of being exposed to the GLS in-utero, age zero or age one, while Panel B estimates the effects of being exposed to the GLS at different trimesters of gestation. The different columns present the results for different sample groups. All control variables are constant across the different models, and only the interaction of having been born in London and being in-utero or aged zero to one at the GLS (*Treat*Pre*) is presented. It is reported as standard deviations from the mean and interpreted as the causal effect of the GLS on respiratory health in older adulthood.

Table 1: Multivariate linear regressions estimated through OLS – Indicator of respiratory health problems as outcome and being born in London during the GLS as interest variable

	(1)	(2)	(3)	(4)	(5)
VARIABLES	Total	Low SES and Male	Low SES and Female	High SES and Male	High SES and Female
Panel A					
Born in London*In-utero at GLS	0.08*	0.37***	-0.02	0.03	0.14
	(0.039)	(0.129)	(0.074)	(0.109)	(0.088)
Born in London*Age 0 at GLS	0.10**	0.03	0.18*	0.03	0.15
	(0.037)	(0.106)	(0.096)	(0.073)	(0.111)
Born in London*Age 1 at GLS	0.05	0.13	0.00	0.12	-0.03
	(0.038)	(0.099)	(0.057)	(0.094)	(0.098)
Observations	43,823	5,170	7,110	4,528	6,310
Panel B					
Born in London*1st trim. preg. at GLS	0.19***	0.56***	0.08	0.28	0.14
	(0.072)	(0.178)	(0.194)	(0.216)	(0.210)
Born in London*2nd trim. preg. at GLS	0.06	0.58***	0.09	-0.06	-0.12
	(0.064)	(0.174)	(0.110)	(0.114)	(0.212)
Born in London*3rd trim. preg. at GLS	-0.02	-0.02	-0.23*	-0.07	0.31*
	(0.055)	(0.162)	(0.119)	(0.158)	(0.167)
Observations	25,436	2,970	4,161	2,599	3,672
Baseline covariates	✓	✓	✓	✓	✓

Note: Standard errors clustered at the postal district level in parenthesis, *** p<0.01, ** p<0.05, and * p<0.10. Sample restricted to non-missing observations for all outcomes, white ethnicity and from December 1950 (Panel A) or December 1952 (Panel B) to August 1953 and from March 1954 to July 1956. The outcome is a standardized factor of having been hospitalized due to respiratory problems from 1997 to 2020, having ever been diagnosed by a doctor as suffering respiratory health problems, presenting spirometry values in the lowest fifth percentile of the sample at the time of the interview, and consuming prescription drugs for respiratory health problems at the time of the interview. Higher values indicate worse respiratory health and vice-versa. Baseline covariates control for being born in London or the rest of England, year-month birth fixed effects, gender and year and month of the interview fixed effects. *Source*: UK Biobank.

Considering the overall sample, Panel A Column 1 shows that individuals exposed to the GLS in-utero were 0.08 standard deviations more likely to suffer respiratory health problems at an older age. In comparison, individuals exposed to the GLS at age zero were 0.1 standard deviations more likely to suffer respiratory health problems at an older age. Panel B Column 1 shows that the effect of the GLS was strongest for exposure at the first trimester of pregnancy. Individuals exposed to the GLS during the first trimester of gestation were 0.19 standard deviations more likely to suffer respiratory health problems at an older age. Part of the effect is captured at age zero, but overall it seems to become smaller and even disappear after birth. Accordingly, even if some individuals could potentially be affected by the GLS after birth, this effect gradually disappeared as they developed. It is consistent with the literature that argues that the gestational phase, and particularly the first trimester of gestation are the most vulnerable periods of human development (Almond and Currie, 2011).

The size of these effects is very substantial when compared to other risk factors. For instance, the mean difference of the indicator of respiratory health problems for individuals exposed and not exposed to maternal smoking around birth is 0.11, for individuals having and not having a college or university degree, it is 0.04, and between males and females, it is 0.03[17].

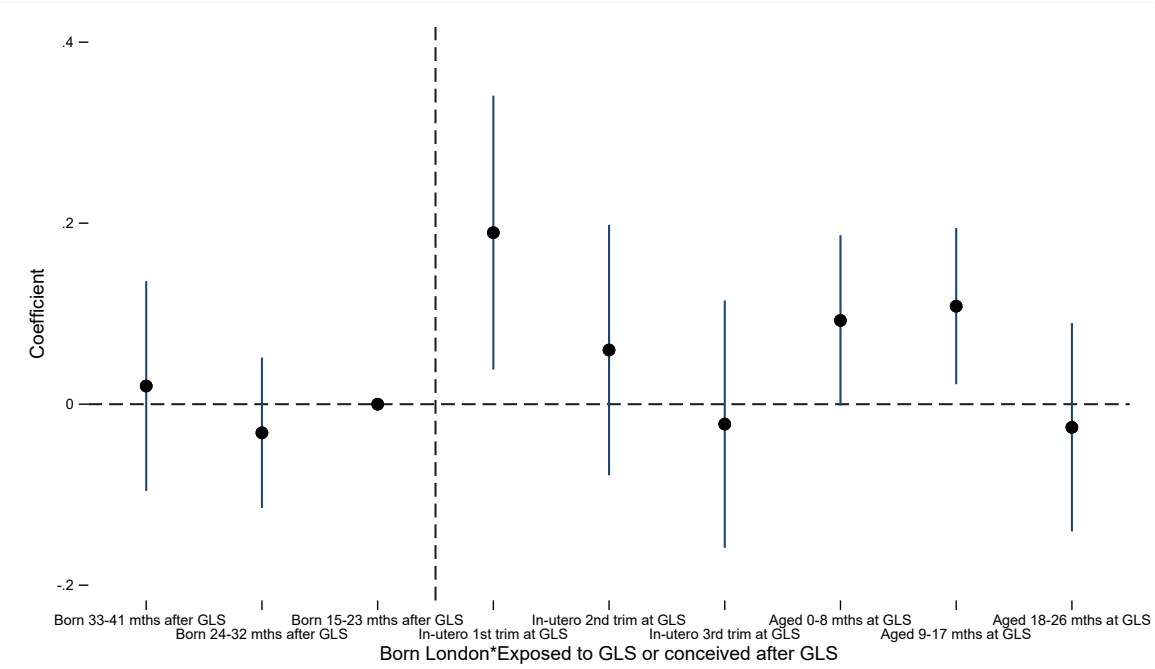
These results can be interpreted as causal under the following two conditions. First, the exposure to the GLS was exogenous to the outcomes. Second, individuals conceived after the GLS have similar trends

[17] Respondent are asked, "Did your mother smoke regularly around the time when you were born?".

in respiratory health regardless of having been born in London or the rest of England. As argued in the methodology section, the first condition holds because: the GLS was a short-term event mainly driven by atmospheric conditions; no preventive measures were taken at the time; and it did not discriminate by SES. The second condition also holds, as can be seen in Figure 2[18], which presents a coefficient-plot of a regression equivalent to the one of Panel B, dividing the time before and after the GLS into periods of nine months (three months for the gestation trimesters). This interval is chosen because it is equivalent to the gestation period and makes it possible to have a homogeneous time unit to evaluate the time trend of the outcomes. Thus, the categorical variables capture exposure to the GLS at different trimesters of gestation or zero to eight months and nine to 17 months after birth. It also considers non-exposure to the GLS, having been conceived seven to 15, 16 to 24, and 25 to 33 months after birth the GLS. As expected, being exposed to the GLS in-utero or early childhood has a strong and significant effect on the indicator of respiratory health problems, compared to the baseline category of being conceived after the GLS. More relevant is that being conceived long after the GLS has no significant effect on the respiratory health problems indicator compared to being conceived only shortly after the GLS. This non-significant difference shows that there is no time-trend (*Post*) that could be driving the results.

[18] Figure C.2 shows that this is also consistent for the different sub-samples divided by gender and SES.

Figure 2: Parallel trend assumption



Note: Standard errors clustered at the postal district level and 95% confidence intervals. Sample restricted to non-missing observations for all outcomes, white ethnicity and from December 1952 to August 1953 and from March 1954 to July 1956. The outcome is a standardised factor of having been hospitalised due to respiratory problems from 1997 to 2020, having ever been diagnosed by a doctor as suffering respiratory health problems, presenting spirometry values in the lowest fifth percentile of the sample at the time of the interview, and consuming prescription drugs for respiratory health problems at the time of the interview. Higher values indicate worse respiratory health and vice-versa. Baseline covariates control for being born in London or the rest of England, year-month birth FE, gender and year and month of the interview fixed effects. *Source*: UK Biobank.

In Table 1 it is also explored if the results vary by gender and SES. Columns (2) to (5) consider the effects for: males born in low SES boroughs; females born in low SES boroughs; males in high SES boroughs; and females born in high SES boroughs. A strong and significant effect is found for males with low SES. All other groups seem to be non-significantly affected by the GLS. The difference between low SES male and low SES female is tested for statistical significance, as is the difference between low SES male and high SES male, and low SES male and high SES female. These null hypotheses can be rejected[19]. These results are consistent with the literature that argues that males are more vulnerable during the gestational phase than females (Kraemer, 2000; Eriksson *et al.*, 2010), and with the literature that has found the most substantial effects of gestational insults on low SES individuals (Almond *et al.*, 2009; Nilsson *et al.*, 2009).

[19] The difference between low SES males and low SES females for exposure in-utero is significant at a 95% confidence level, for exposure during the first trimester of gestation at a 90% confidence level, and for exposure during the second trimester of gestation at a 99% confidence level. The difference between low SES males and high SES males for exposure in-utero is significant at a 95% confidence level and for exposure during the second trimester of gestation at a 99% confidence level. The difference between low SES male and high SES female is not significant for some periods, but very close to the 90% confidence level cut-off for all of them. For exposure in-utero the p-value of this difference is 0.13. For exposure during the first trimester of gestation the p-value is 0.11, for exposure during the second trimester of gestation the difference is significant at a 99% confidence level, and for exposure during the third trimester of gestation the p-value is 0.12.

Kraemer (2000); Eriksson *et al.* (2010) discuss why male are more vulnerable than female during the gestational phase. The growth of the fetus is limited by the capacity of the mother and the placenta. As male tend to be longer and grow faster than female, they have less reserve capacity and suffer a greater risk of becoming undernourished. As such they are more exposed to gestational insults, which can reduce the capacity of the mother to produce nutrients and oxygen. In the case of the GLS, male would suffer a more detrimental effect compared to female, what gets reflected in these results. There are also strong arguments on why low SES individuals should be most affected by the GLS. Three potential mechanisms could be driving this SES gradient.

This SES gradient has been attributed mainly to three different mechanisms. First, compensatory behaviours of parents could have differed due to their SES (Almond and Mazumder, 2013). Parents with higher SES levels could have had higher resources to diagnose and treat the respiratory health problems of their children. Second, the environment in high SES areas could have allowed children to recover, while the environment of children born in low SES areas could have exacerbated their respiratory health problems (Cutler *et al.*, 2008). A third potential mechanism would be individuals receiving different levels of exposure to the GLS due to their SES. The limited air pollution registries of that time period suggest otherwise, since both high and low SES boroughs were heavily affected by the GLS (Ball, 2018b). Even if individuals with higher SES would have been capable to avoid the worst aspects of the GLS (Sun *et al.*, 2017; Jin *et al.*, 2020), this seems insufficient to explain the very substantial size of its effect.

The fragility of male during the period of gestation in combination with these three mechanisms could have driven this substantial gradient. Unfortunately, data limitations do not make it possible to identify these single channels. Still, this finding contributes to the literature by confirming large gender and SES differences in the effect of exposure to air pollution in-utero (Nilsson *et al.*, 2009; Almond *et al.*, 2009).

6.2 Alternative specifications and robustness

These results are robust to alternative specifications considering different control groups, as can be seen in Table 2. Column 1 presents results equivalent to Table 1, Column 1, Panel (B) and is used for ease of comparison; the control group is the rest of England. Column 2 only includes the six biggest cities in England at the time of the GLS (Birmingham, Liverpool, Manchester, Sheffield, Leeds, and Bristol) in the control group. Column 3 only includes London's counties (Essex, Kent, Surrey, Bucks, and Hertford) in the control group. Column 4 only includes the biggest city in England during the GLS and its metropolitan area (Birmingham). Column 5 does not include a control group and only explores changes within London. These models present highly consistent results to the specification using the rest of England as a control group. The effect identified by these models is slightly larger than the baseline specification in Column 1. The specification using the rest of England as the control group is the preferred one since it considers the

most complete comparison group, and it is consistent with the previous literature (Bharadwaj *et al.*, 2016; Ball, 2018b).

Table 2: Multivariate linear regressions estimated through OLS – Indicator of respiratory health problems as outcome and being born in London during the GLS as interest variable considering different control groups

VARIABLES	(1) Baseline rest of England as controls	(2) Six biggest cities as controls	(3) Counties surrounding London as controls	(4) Birmingham and metropolitan area as control	(5) Within London no control
Born in London*1st trim. preg. at GLS	0.19***	0.24***	0.23**	0.27**	0.27*
	(0.072)	(0.082)	(0.110)	(0.130)	(0.147)
Born in London*2nd trim. preg. at GLS	0.06	0.04	0.21**	-0.09	0.10
	(0.064)	(0.095)	(0.083)	(0.094)	(0.150)
Born in London*3rd trim. preg. at GLS	-0.02	-0.10	0.05	0.08	-0.13
	(0.055)	(0.072)	(0.151)	(0.066)	(0.137)
Observations	25,436	8,379	4,248	4,699	3,193
Baseline covariates	✓	✓	✓	✓	✓

Note: Standard errors clustered at the postal district level in parenthesis, *** p<0.01, ** p<0.05, and * p<0.10. Sample restricted to non-missing observations for all outcomes, white ethnicity and from December 1952 to August 1953 and from March 1954 to July 1956. The outcome is a standardised factor of having been hospitalised due to respiratory problems from 1997 to 2020, having ever been diagnosed by a doctor as suffering respiratory health problems, presenting spirometry values in the lowest fifth percentile of the sample at the time of the interview, and consuming prescription drugs for respiratory health problems at the time of the interview. Higher values indicate worse respiratory health and vice-versa. Baseline covariates control for being born in London or the rest of England, year-month birth FE, gender and year and month of the interview fixed effects. *Source*: UK Biobank.

Results for the individual outcomes can be found in Table 3[20]. The specification is equivalent to the one of Table 1, Panel B, Column 1, but substituting the respiratory health problems indicator by the individual variables that generate it. The effect is reported in percentages, and it is large and positive for all the outcomes. It is significant for being hospitalised due to respiratory causes from 1997 to 2020 and consuming prescription drugs for respiratory health at the interview. Being exposed to the GLS during the first trimester of gestation increases the probabilities of being hospitalised due to respiratory causes from 1997 to 2020 by 6.03 percentage points and the probabilities of consuming prescription drugs for respiratory health at the moment of the interview by 3.53 percentage points. Relative to the mean, this increases hospitalisations by 27.05% and prescription drugs 66.48%.

[20] Results with the largest sample size for each outcome can be found in Table C.5. The results are consistent and the significance level of the estimates raises.

Table 3: Multivariate linear regressions estimated through OLS – Individual respiratory health outcomes and being born in London during the GLS as interest variable

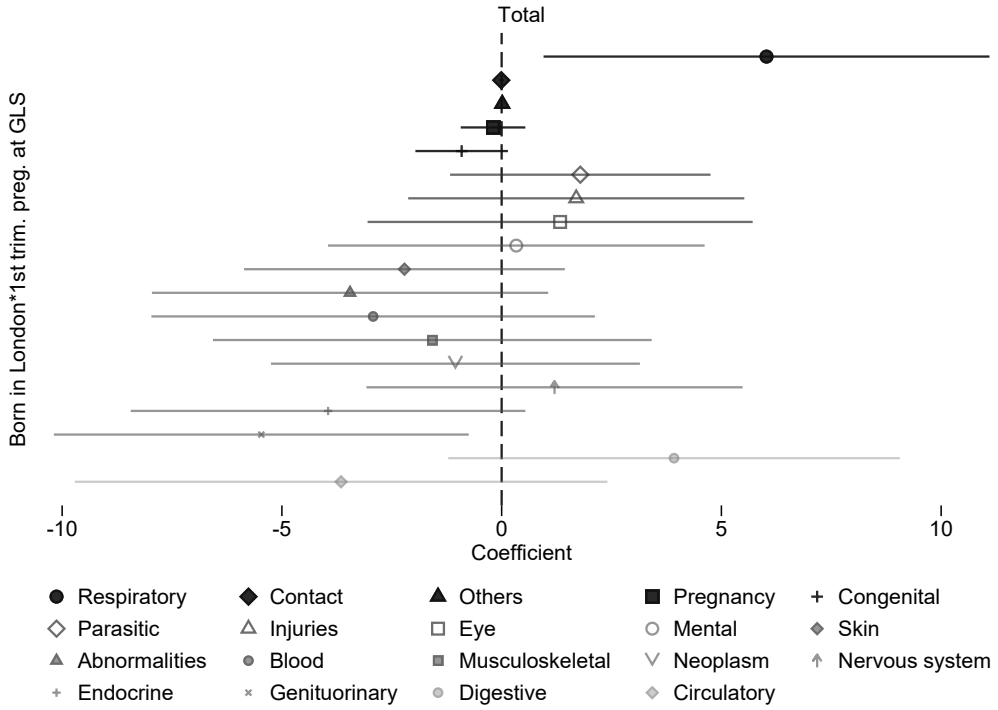
VARIABLES	(1) Diag. Dr. respi (ever)	(2) Hospi. respi. from 1997-2020	(3) Current self-reported drugs respi.	(4) 5th pct. spiro z-score FEV1/FVC at interview date
Born in London*1st trim. preg. at GLS	4.37	6.03*	3.53**	1.31
	(2.864)	(3.056)	(1.450)	(1.688)
Born in London*2nd trim. preg. at GLS	3.41	3.00	-0.08	1.57
	(2.252)	(2.892)	(1.490)	(1.118)
Born in London*3rd trim. preg. at GLS	1.20	-0.27	-1.02	1.12
	(2.492)	(2.407)	(1.043)	(1.495)
Observations	25,436	25,436	25,436	25,436
Baseline covariates	✓	✓	✓	✓
Mean	66.43	22.29	5.31	5.07

Note: Standard errors clustered at the postal district level in parenthesis, *** p<0.01, ** p<0.05, and * p<0.10. Sample restricted to non-missing observations for all outcomes, white ethnicity and from December 1952 to August 1953 and from March 1954 to July 1956. The outcome is a standardised factor of having been hospitalised due to respiratory problems from 1997 to 2020, having ever been diagnosed by a doctor as suffering respiratory health problems, presenting spirometry values in the lowest fifth percentile of the sample at the time of the interview, and consuming prescription drugs for respiratory health problems at the time of the interview. Higher values indicate worse respiratory health and vice-versa. Baseline covariates control for being born in London or the rest of England, year-month birth FE, gender and year and month of the interview fixed effects. *Source*: UK Biobank.

6.3 Other outcomes

There exists a strong link between air pollution and respiratory health (Kajekar, 2007; Selgrade *et al.*, 2013; Harding and Maritz, 2012), while there is a much weaker link with other health outcomes. Still, the GLS could have affected other respiratory health outcomes as well. Figure 3 considers the results of different regressions with a specification equivalent to the one of Table 3, Column 2 and estimating the effect of the GLS on the rest of hospitalisation ICD-10 categories. The most substantial effect is found for hospitalisations due to respiratory causes. For most other outcomes, the effect is non-significant. Only hospitalisations caused by genitourinary causes present a significant effect as well. The result for genitourinary causes could be driven due to chance, as 18 hypotheses (excluding hospitalisations due to respiratory causes) are tested at a 90% confidence level, or there could also be a causal effect of the GLS on this outcome. Regardless of that outcome, this figure shows that the most substantial effect was captured on hospitalisations due to respiratory causes, while most other outcomes remained unaffected. This most significant effect on respiratory health is consistent with the literature (Korten *et al.*, 2017; Bettiol *et al.*, 2021; Johnson *et al.*, 2021).

Figure 3: Tree-plot with all hospitalization ICD-10 outcomes – Exposure first trimester of gestation



Note: Standard errors clustered at postal district level in parenthesis, *** p<0.01, ** p<0.05, and * p<0.10. Sample restricted to non-missing observations for all outcomes, white ethnicity and from December 1952 to August 1953 and from March 1954 to July 1956. The outcome is a standardised factor of having been hospitalised due to respiratory problems from 1997 to 2020, having ever been diagnosed by a doctor as suffering respiratory health problems, presenting spirometry values in the lowest fifth percentile of the sample at the time of the interview, and consuming prescription drugs for respiratory health problems at the time of the interview. Higher values indicate worse respiratory health and vice-versa. Baseline covariates control for being born in London or the rest of England, year-month birth FE, gender and year and month of the interview fixed effects. *Source*: UK Biobank.

Individuals attended the UK Biobank from 2006 to 2010, and mortality data was linked to them. An outcome is generated from this mortality data that considers early mortality due to respiratory causes from ages 50 to 70. Table 4 presents a specification equivalent to the one of Table 3 for the outcome of early mortality due to respiratory causes. Both Columns 1 and 2 present a small and non-significant coefficient. They suggest that even if a strong and significant effect of the GLS was found on respiratory health problems, this was not strong enough to be captured by mortality.

Table 4: Multivariate linear regressions estimated through OLS – Mortality outcomes and being born in London during GLS as interest event

VARIABLES	(1) Mortality respi. (2006–2020)	(2) Mortality respi. (2006–2020)
Born in London*In-utero at GLS	-0.00	
	(0.003)	
Born in London*1st trim. preg. at GLS		0.00
		(0.006)
Born in London*2nd trim. preg. at GLS		0.00
		(0.004)
Born in London*3rd trim. preg. at GLS		-0.00
		(0.005)
Observations	43,823	25,436
Baseline covariates	✓	✓
Mean	0.01	0.01

Note: Standard errors clustered at postal district level in parenthesis, *** p<0.01, ** p<0.05, and * p<0.10. Sample restricted to non-missing observations for all outcomes, white ethnicity and from December 1952 to August 1953 and from March 1954 to July 1956. The outcome is mortality after having participated at the UK Biobank. Baseline covariates control for being born in London or the rest of England, year-month birth FE, gender and year and month of the interview fixed effects. *Source*: UK Biobank

6.4 Mechanisms

Respiratory health problems could have affected individuals throughout their life cycle or triggered at an older age. To explore this hypothesis, Table 5 presents the results of self-reporting having ever been diagnosed with respiratory health problems by a doctor for different age groups. The specification is equivalent to the one of Table 3, Column 1. The effect of exposure to the GLS during the first trimester of gestation is most substantial at age 20, while exposure during the second trimester of gestation is strongest at ages 30, 40 and 50. Thus, the effects of the GLS started at a young age and extended over time until older adulthood. This result suggests that individuals exposed to the GLS suffered respiratory health problems throughout their whole life cycle.

Table 5: Multivariate linear regressions estimated through OLS – Self-reported respiratory problems by age as outcomes and being born in London during GLS as interest variable

VARIABLES	(1) Diag. Dr. respi. before age 20	(2) Diag. Dr. respi. before age 30	(3) Diag. Dr. respi. before age 40	(4) Diag. Dr. respi. before age 50
Born in London*1st trim. preg. at GLS	3.28**	1.11	1.11	1.98
	(1.638)	(2.025)	(2.337)	(3.548)
Born in London*2nd trim. preg. at GLS	3.63	5.85**	6.27**	7.27**
	(2.410)	(2.822)	(2.720)	(3.039)
Born in London*3rd trim. preg. at GLS	1.75	0.23	1.53	3.00
	(3.431)	(2.626)	(2.752)	(3.238)
Observations	25,436	25,436	25,436	25,436
Baseline covariates	✓	✓	✓	✓
Mean	14.50	22.39	31.41	49.53

Note: Standard errors clustered at postal district level in parenthesis, *** p<0.01, ** p<0.05, and * p<0.10. Sample restricted to non-missing observations for all outcomes, white ethnicity and from December 1952 to August 1953 and from March 1954 to July 1956. The outcome is a standardized factor of having been hospitalized due to respiratory problems from 1997 to 2020, having ever been diagnosed by a doctor suffering respiratory health problems, presenting spirometry values in the lowest fifth percentile of the sample at the time of the interview, and consuming prescription drugs for respiratory health problems at the time of the interview. Higher values indicate worse respiratory health and vice-versa. Baseline covariates control for being born in London or the rest of England, year-month birth FE, gender and year and month of the interview fixed effects. *Source*: UK Biobank

The GLS could have directly affected individuals' respiratory health, or it could have affected their exposure to risky environments and behaviours. Ball (2018b) found that the GLS caused individuals to have lower levels of education and to face higher unemployment rates throughout adulthood. Individuals with lower levels of education and who are unemployed are more likely to live in areas with high levels of air pollution (Lipfert, 2004); engage in risky behaviours such as smoking (Wetter *et al.*, 2005); and be exposed to risky work environments (Brown *et al.*, 2021). Therefore the effect of the GLS on respiratory health could have been driven by these.

To explore this hypothesis, Table 6 estimates whether the GLS affected the levels of pollution and traffic intensity at the place of residency of UK Biobank respondents at the time of the interview, their probabilities of ever smoking, and their probabilities of having the main occupation in a sector with high exposure to air contaminants[21]. Residential air pollution is obtained through the linkage of the MERRA-2 database with the place of residency of the UK Biobank respondents. The average levels of PM2.5 air pollution at the place of residency the year previous to the interview are estimated. Traffic intensity is a UK Biobank derived variable that measures traffic levels at the closest major road to individuals' residency. Ever having been a smoker is a self-reported indicator. Exposure to air contaminants at the workplace is obtained through the linkage of the O*NET database and the longest lifetime occupation of the UK Biobank respondents.

Table 6: Multivariate linear regressions estimated through OLS – Mechanisms of respiratory health as outcomes and being born in London during the GLS as interest variable considering different control groups

VARIABLES	(1) Yearly average air pollution at residency	(2) High traffic intensity	(3) Ever smoked	(4) Occupation high pollution
Born in London*1st trim. preg. at GLS	-0.08	0.01	0.04	-0.04
	(0.074)	(0.024)	(0.040)	(0.024)
Born in London*2nd trim. preg. at GLS	-0.11*	-0.01	0.02	-0.03
	(0.064)	(0.012)	(0.035)	(0.027)
Born in London*3rd trim. preg. at GLS	-0.07	0.02	0.03	-0.06**
	(0.071)	(0.015)	(0.032)	(0.025)
Observations	24,424	25,436	25,436	25,434
Mean	0.00	0.06	0.44	0.21
Baseline covariates	✓	✓	✓	✓

Note: Standard errors clustered at postal district level in parenthesis, *** p<0.01, ** p<0.05, and * p<0.10. Sample restricted to non-missing observations for all outcomes, white ethnicity and from December 1952 to August 1953 and from March 1954 to July 1956. Yearly average air pollution at the place of residency at the interview is a standardised variable that measures PM2.5 using MERRA-2 data. Traffic intensity at the nearest road measures the average total number of road vehicles per 24 hours on the nearest major road using data from the Road Traffic Statistics Branch at the Department of Transport. High traffic intensity considers being two standard deviations above the mean. Occupation high pollution measures if the main occupation of individuals was in the 90th percentile or above of occupations with exposure to contaminants such as pollutants gases, dust or odours, using data from the O*NET. Baseline covariates control for being born in London or the rest of England, year-month birth FE, gender and year and month of the interview fixed effects. *Source*: UK Biobank, MERRA-2, O*NET.

The effect of the GLS on air pollution at the place of residency is small and consistently negative, but overall not statistically significant. Only for the total sample and exposure at the second trimester of

[21] Pollution at the place of residency during the UK Biobank interview is highly and significantly correlated with contemporaneous measures of respiratory health (spirometry), while ever having been a smoker and having a main occupation with high exposure to air-born contaminants is highly and significantly correlated with historical measures of respiratory health (an indicator of respiratory health problems). In addition, living close to a road with high traffic intensity is strongly correlated with residential air pollution.

gestation, the effect is significant at a 90% significance level. The effect of the GLS on having high traffic intensity at the nearest road and ever having been a smoker is small and not statistically significant. The effect of the GLS on having the main occupation with high exposure to air born pollutants is negative and significant. Overall, these results do not present very consistent coefficients, and most of them are not statistically significant. They suggest that the GLS did not affect the environment nor the risky behaviours of individuals. In the case it did, this effect was negative, as seen for having an occupation with exposure to a high level of air-born contaminants. Individuals suffering from respiratory health problems could have chosen occupations with better air quality standards. These results show that later life exposures to risky environments or behaviours did not drive the effect of the GLS.

7 Conclusions

This essay has estimated the causal effect of the GLS on respiratory health outcomes at an older age. Using the UK Biobank and a DID strategy, it has shown that exposure to the GLS in-utero or early childhood had large health effects at an older age. Being exposed to the GLS in-utero increased the probability of suffering respiratory health problems in older adulthood by 0.08 standard deviations. Exposure at age zero increased the probability of suffering respiratory health problems in 0.1 standard deviations, and exposure during the first trimester of gestation increased in 0.19 standard deviations. There were significant differences in the exposure to the GLS by gender and SES, with low SES males suffering the most prominent effect.

Only two papers so far have examined the long-term health effects of the GLS. Bharadwaj *et al.* (2016) explored the effect of the GLS on asthma prevalence during childhood and adulthood. They find a moderate effect of the GLS on asthma prevalence during childhood but no significant effect on adulthood. This result can be driven by a very small sample and a self-reported outcome subject to error. Ball (2018b) expands this analysis exploring the effect of the GLS on economic and health outcomes, only finding strong long-run effects for the first ones. For the long-term health outcomes, no significant effects are found for exposure to the GLS in-utero, while significant effects are found for exposure to the GLS at early childhood. An explanation for the consistent results on educational and labor outcomes is the theory of dynamic complementarity or self-productivity (Conti and Heckman, 2012; Cunha and Heckman, 2007). According to this theory, early conditions and environments are crucial for individuals' ability to learn and can deeply affect their productivity in later years. Being exposed by the GLS in-utero or ages zero to one negatively impacted the development of individuals at a very early life stage. This reduced their return of investments at any other further stage. As such, not only health outcomes were affected by the GLS, but also a large range of economic ones.

This essay contributes to these papers by finding very strong and highly significant long-term effects of the GLS on respiratory health outcomes, and it did so by analysing an extensive database, and a set of objective and self-reported health outcomes.

This essay also contributes to the rest of the literature in this field, reporting the longest-run effects found so far. It finds that exposure to pollution in-utero can significantly affect health outcomes up to 70 years after the exposure. It also finds that gender and SES are a strong determinants of the magnitude of the effect of exposure to pollution in-utero. A short-term period of air pollution is also considered, which last only a couple of days instead of prolonged exposure to pollution. Finally, very relevant and robust health outcomes are explored, which lead to very clear conclusions.

The differences in the effect of the GLS by SES is a particularly interesting results in terms of policy making. Three mechanisms have been proposed that could be driving the gradient.

The first mechanism is individuals receiving different levels of exposure to the GLS due to their SES. The limited air pollution registries of that time period suggest that this was not the case, since both high and low SES boroughs were heavily affected by the GLS (Ball, 2018b). Still, individuals with higher SES could be more likely to avoid the worst aspects of the GLS (Sun *et al.*, 2017; Jin *et al.*, 2020). Even if not possible to rule out, this option seems limited since the technology for the protection from air pollution was primitive at that time, and even nowadays only offers limited protection (Cherrie *et al.*, 2018). Some behaviours could have clearly differed by SES. For example, pregnant women with low SES could have been more likely to leave their houses to work during the days of extreme pollution and thus experience a more substantial exposure. This could drive part of this gradient, but it seems insufficient to explain its very substantial size.

The second mechanism is that compensatory behaviours of parents could have differed due to their SES (Almond and Mazumder, 2013). Ambiguous results have been found so far considering how parents respond to their children's endowments (Almond and Mazumder, 2013). A group of papers found that parents compensate the endowments of their children (Black *et al.*, 2010; Del Bono *et al.*, 2012; Bharadwaj *et al.*, 2010). This compensation means that parents take measures if their children experience developmental difficulties. In the scenario of the GLS, parents could promote healthy behaviours in their children affected by that event to reverse the long-term detrimental consequences. Other papers found reinforcing responses (Aizer and Cunha, 2012; Venkataramani, 2012; Datar *et al.*, 2010; Almond *et al.*, 2009; Rosenzweig and Zhang, 2009), suggesting that parents receive larger marginal gains from investing in the children that have greater endowments. In the scenario of the GLS, parents would invest less in children having been exposed in-utero, reinforcing its detrimental effects. Another group found mixed evidence of compensatory and reinforcing responses (Conti *et al.*, 2011; Restrepo, 2011; Parman *et al.*, 2012; Hsin, 2012), which have been attributed to different dimensions of children's capabilities and investments. A last group has found

no responses (Bharadwaj *et al.*, 2013; Royer, 2009; Currie and Almond, 2011; Kelly, 2011), implying that parents might have difficulties in recognising their children endowments and do not react to them. The ambiguity of these results can be driven by the variability in parental investments and initial endowments and by the difficulty of recognising children's endowments. SES can limit the range of compensatory or reinforcing tools used and change the capacity of recognising children's endowments. In the case of the GLS, parents with more resources were more likely to access private health services. As a result, they would be more likely to recognise their children's respiratory problems and treat them properly.

The third mechanism is that the environment in high SES areas could have allowed children to recover, while the environment of children born in low SES areas could have exacerbated their respiratory health problems (Cutler *et al.*, 2008). Individuals with low SES are more likely to grow up in neighbourhoods with high levels of pollution; be exposed to violence; have less access to social capital; suffer conflicts within their families; live in uninhabitable houses; suffer psychological stress; and take risky behaviours (Chen and Miller, 2013). All these factors have detrimental effects on their health and they could be very harmful to individuals with an initial unhealthy status. The GLS could have triggered the respiratory problems, and the SES environmental factors would have exacerbated or reduced these. High SES individuals would be exposed to a few of these risk factors and could recover from the effects of the GLS. However, medium or low SES individuals would be exposed to these factors, which would impede their recovery and even worsen the effects of the GLS.

One of the most vital points of this essay is the detailed, individual-level database of the UK Biobank, which is ideal for exploring health impacts associated with exposure to the GLS because the data are conducive to clean identification of linkages. This data makes it possible to capture causal, long-term effects on a group of very reliable health outcomes. Its main limitation is that it is impossible to differentiate among different pollutants and intensities of air pollution due to data limitations. A significant gap remains in the literature regarding this dimension.

Distinguishing between different pollutants is an essential point since the results of this essay can be directly compared to developing countries but less so to developed ones[22]. Developing countries experience regular air-pollution peaks in which the levels of different kinds of pollutants are similar to the ones of the GLS (Wang *et al.*, 2016). Developed countries can experience peaks of extreme pollution during natural disasters, but it is unlikely for them to present similarly high levels of sulfur dioxide. Data limitations make it impossible to distinguish between the effect of different air pollutants in the analysis. Therefore,

[22] Currently, sulfur dioxide emissions are highly restricted in developed countries, as they can have very detrimental effects on human health. Emissions of sulfur dioxide have been greatly reduced in the last century, and when nowadays overall air pollution is high in developed countries, it is likely sulfur dioxide levels remain at safe levels (Grennfelt *et al.*, 2020). In developing countries, this is still a major problem and happens quite regularly.

for developed countries, the results are less applicable and require further research, while they are directly applicable for developing countries.

This limitation affects the policy implications of this work, and these differ significantly between developing and developed countries.

The results are directly applicable to the context of developing countries since the GLS presented similar air pollution levels to the ones that they regularly experience (Wang *et al.*, 2016). Every time that areas in developing countries experience air pollution levels equivalent to those of the GLS, the respiratory health of a whole cohort of individuals is compromised. Therefore, the most vital implication of these results is that pregnant women and young children must be protected from such events. For this aim, air quality data has to improve in developing countries. Without this information, it is impossible to protect vulnerable individuals at episodes of high air pollution, and developing countries often lack access to quality data, or this data are systemically misreported. Roychowdhury and Somvanshi (2020) highlights that India does not have a standard official method for reporting air pollution levels and that this lead to missing data and data gaps. Turiel and Kaufmann (2021) go a step further and find evidence of Chinese air quality data being systematically misreported in five different cities from 2015 to 2017. They compared air quality measurements of the Chinese government-controlled monitoring stations with measurements of the US embassy-controlled monitoring stations. Divergences were more frequent than those expected by random chance; they were biased towards better air quality reports from the government-controlled stations, and misreports were more likely to occur during periods of high pollution. It is reasonable to assume that similar problems occur in many other developing countries. Such misinformation could directly mislead individuals and contribute to pregnant women and young children exposing themselves and their children to extreme air pollution, leading to very long-run detrimental problems. Once having reliable air quality data, different policies could be thought of to protect pregnant women and young children at periods of high air pollution levels. Their efficacy would depend on resource limitations and cultural context.

In developed countries, the results are not directly applicable since one of the most harmful pollutants of the GLS was sulphur dioxide, which is restricted there. Most fuels are purified in developed countries to avoid the emission of these substances. Due to data limitations, it is impossible to identify different air pollutants' emissions during the GLS. Therefore this question remains unsolved, and further research will have to establish if extreme air pollution driven by PM or other substances that developed countries experience during natural disasters and other extreme events has a similar effect.

Despite these limitations, this essay sends a powerful message to policymakers, pointing at their responsibility to protect vulnerable groups of the population during short-term periods of extreme air pollution.

Furthermore, it highlights the risk of not protecting vulnerable individuals during periods of extreme air pollution; the long-term costs will most certainly outmatch the cost of protecting them. Therefore, prevention of individuals health outcomes at older age begins before these individuals are born.

References

AGARWAL, N., BANTERNGHANSA, C. and BUI, L. T. (2010). Toxic exposure in America: Estimating fetal and infant health outcomes from 14 years of TRI reporting. *Journal of Health Economics*, **29** (4), 557–574.

AIZER, A. and CUNHA, F. (2012). The production of human capital in childhood: endowments, investments and fertility. *Unpublished manuscript, Brown University*.

—, STROUD, L. and BUKA, S. (2016). Maternal stress and child outcomes: Evidence from siblings. *Journal of Human Resources*, **51** (3), 523–555.

ALMOND, D. and CURRIE, J. (2011). Killing me softly: The fetal origins hypothesis. *Journal of Economic Perspectives*, **25** (3), 153–72.

—, EDLUND, L. and PALME, M. (2009). Chernobyl's subclinical legacy: prenatal exposure to radioactive fallout and school outcomes in Sweden. *The Quarterly Journal of Economics*, **124** (4), 1729–1772.

— and MAZUMDER, B. (2013). Fetal origins and parental responses. *Annual Review of Economics*, **5** (1), 37–56.

ALTINDAG, D. T., BAEK, D. and MOCAN, N. (2017). Chinese yellow dust and Korean infant health. *Social Science & Medicine*, **186**, 78–86.

ANDERSEN, Z. J., RAASCHOU-NIELSEN, O., KETZEL, M., JENSEN, S. S., HVIDBERG, M., LOFT, S., TJØNNELAND, A., OVERVAD, K. and SØRENSEN, M. (2012). Diabetes incidence and long-term exposure to air pollution: a cohort study. *Diabetes Care*, **35** (1), 92–98.

ARCHER, C. L., BRODIE, J. F. and RAUSCHER, S. A. (2019). Global warming will aggravate ozone pollution in the US Mid-Atlantic. *Journal of Applied Meteorology and Climatology*, **58** (6), 1267–1278.

AUSTRALIAN ASSOCIATED PRESS (2020). Melbourne's air quality 'worst in the world' as bushfires continue to burn across Victoria.

BALL, A. (2018a). Hidden costs of the Great London Smog: evidence from missing births.

— (2018b). The long-term economic costs of the Great London Smog.

BARANOV, V., BHALOTRA, S., BIROLI, P. and MASELKO, J. (2020). Maternal depression, women's empowerment, and parental investment: evidence from a randomized controlled trial. *American Economic Review*, **110** (3), 824–59.

BEACH, B. and HANLON, W. W. (2018). Coal smoke and mortality in an early industrial economy. *The Economic Journal*, **128** (615), 2652–2675.

BELL, M. L. and DAVIS, D. L. (2001). Reassessment of the lethal London fog of 1952: novel indicators of acute and chronic consequences of acute exposure to air pollution. *Environmental Health Perspectives*, **109** (suppl 3), 389–394.

—, — and FLETCHER, T. (2004). A retrospective assessment of mortality from the London smog episode of 1952: the role of influenza and pollution. *Environmental Health Perspectives*, **112** (1), 6–8.

BETTIOL, A., GELAIN, E., MILANESIO, E., ASTA, F. and RUSCONI, F. (2021). The first 1000 days of life: traffic-related air pollution and development of wheezing and asthma in childhood. A systematic review of birth cohort studies. *Environmental Health*, **20** (1), 1–10.

BHARADWAJ, P., EBERHARD, J. and NEILSON, C. (2010). Do initial endowments matter only initially? Birth weight, parental investments and academic achievement in school. *University of California at San Diego, Department of Economics*.

—, GIBSON, M., ZIVIN, J. G. and NEILSON, C. (2017). Gray matters: Fetal pollution exposure and human capital formation. *Journal of the Association of Environmental and Resource Economists*, **4** (2), 505–542.

—, LØKEN, K. V. and NEILSON, C. (2013). Early life health interventions and academic achievement. *American Economic Review*, **103** (5), 1862–91.

—, ZIVIN, J. G., MULLINS, J. T. and NEIDELL, M. (2016). Early-life exposure to the great smog of 1952 and the development of asthma. *American Journal of Respiratory and Critical Care Medicine*, **194** (12), 1475–1482.

BISHOP, K. C., KETCHAM, J. D. and KUMINOFF, N. V. (2018). *Hazed and confused: the effect of air pollution on dementia*. Tech. rep., National Bureau of Economic Research.

BLACK, S. E., BÜTIKOFER, A., DEVEREUX, P. J. and SALVANES, K. G. (2013). *This is only a test? Long-run impacts of prenatal exposure to radioactive fallout*. Tech. rep., National Bureau of Economic Research.

—, DEVEREUX, P. J. and SALVANES, K. G. (2010). Small family, smart family? family size and the IQ scores of young men. *Journal of Human Resources*, **45** (1), 33–58.

BOVÉ, H., BONGAERTS, E., SLENDERS, E., BIJNENS, E. M., SAENEN, N. D., GYSELAERS, W., VAN EYKEN, P., PLUSQUIN, M., ROEFFAERS, M. B., AMELOOT, M. *et al.* (2019). Ambient black carbon particles reach the fetal side of human placenta. *Nature Communications*, **10** (1), 1–7.

BRAUER, M., HOEK, G., SMIT, H., DE JONGSTE, J., GERRITSEN, J., POSTMA, D. S., KERKHOF, M. and BRUNEKREEF, B. (2007). Air pollution and development of asthma, allergy and infections in a birth cohort. *European Respiratory Journal*, **29** (5), 879–888.

BROWN, S., BROOKS, R. D. and DONG, X. S. (2021). Injury inequalities among US construction workers. *Journal of Occupational and Environmental Hygiene*, **18** (4-5), 159–168.

BUCKBERRY, S., BIANCO-MIOTTO, T., BENT, S. J., DEKKER, G. A. and ROBERTS, C. T. (2014). Integrative transcriptome meta-analysis reveals widespread sex-biased gene expression at the human fetal–maternal interface. *Molecular Human Reproduction*, **20** (8), 810–819.

CAREY, I. M., ANDERSON, H. R., ATKINSON, R. W., BEEVERS, S. D., COOK, D. G., STRACHAN, D. P., DAJNAK, D., GULLIVER, J. and KELLY, F. J. (2018). Are noise and air pollution related to the incidence of dementia? A cohort study in London, England. *British Medical Journal open*, **8** (9), e022404.

CASALE, D. and DESMOND, C. (2016). Recovery from stunting and cognitive outcomes in young children: evidence from the south African Birth to Twenty Cohort Study. *Journal of Development Origin Health Disabilities*, **7** (2), 163–71.

CHAY, K. Y. and GREENSTONE, M. (2003). The impact of air pollution on infant mortality: evidence from geographic variation in pollution shocks induced by a recession. *The Quarterly Journal of Economics*, **118** (3), 1121–1167.

CHEN, E. and MILLER, G. E. (2013). Socioeconomic status and health: mediating and moderating factors. *Annual Review of Clinical Psychology*, **9**, 723–749.

CHERRIE, J. W., APSLEY, A., COWIE, H., STEINLE, S., MUELLER, W., LIN, C., HORWELL, C. J., SLEEUWENHOEK, A. and LOH, M. (2018). Effectiveness of face masks used to protect Beijing residents against particulate air pollution. *Occupational and Environmental Medicine*, **75** (6), 446–452.

CLARK, N. A., DEMERS, P. A., KARR, C. J., KOEHOORN, M., LENCAR, C., TAMBURIC, L. and BRAUER, M. (2010). Effect of early life exposure to air pollution on development of childhood asthma. *Environmental Health Perspectives*, **118** (2), 284–290.

CLAY, K., LEWIS, J. and SEVERNINI, E. (2016). *Canary in a coal mine: Infant mortality, property values, and tradeoffs associated with mid-20th century air pollution*. Tech. rep., National Bureau of Economic Research.

CONTI, G. and HECKMAN, J. J. (2012). *The economics of child well-being*. Tech. rep., National Bureau of Economic Research.

—, —, YI, J. and ZHANG, J. (2011). Early health shocks, parental responses, and child outcomes. *Unpublished manuscript, University of Chicago*.

CUNHA, F. and HECKMAN, J. (2007). The technology of skill formation. *American Economic Review*, **97** (2), 31–47.

CUPUL-UICAB, L. A., SKJAERVEN, R., HAUG, K., MELVE, K. K., ENGEL, S. M. and LONGNECKER, M. P. (2012). In utero exposure to maternal tobacco smoke and subsequent obesity, hypertension, and gestational diabetes among women in the MoBa cohort. *Environmental Health Perspectives*, **120** (3), 355–360.

CURRIE, J. and ALMOND, D. (2011). Human capital development before age five. In *Handbook of Labor Economics*, vol. 4, Elsevier, pp. 1315–1486.

—, DAVIS, L., GREENSTONE, M. and WALKER, R. (2015). Environmental health risks and housing values: evidence from 1,600 toxic plant openings and closings. *American Economic Review*, **105** (2), 678–709.

— and NEIDELL, M. (2005). Air pollution and infant health: what can we learn from California's recent experience? *The Quarterly Journal of Economics*, **120** (3), 1003–1030.

— and SCHMIEDER, J. F. (2009). Fetal exposures to toxic releases and infant health. *American Economic Review*, **99** (2), 177–83.

— and SCHWANDT, H. (2016). The 9/11 dust cloud and pregnancy outcomes: A reconsideration. *Journal of Human Resources*, **51** (4), 805–831.

— and WALKER, R. (2011). Traffic congestion and infant health: Evidence from E-ZPass. *American Economic Journal: Applied Economics*, **3** (1), 65–90.

CUTLER, D. M., LLERAS-MUNEY, A. and VOGL, T. (2008). Socioeconomic status and health: dimensions and mechanisms.

DATAR, A., KILBURN, M. R. and LOUGHRAN, D. S. (2010). Endowments and parental investments in infancy and early childhood. *Demography*, **47** (1), 145–162.

DEL BONO, E., ERMISCH, J. and FRANCESCONI, M. (2012). Intrafamily resource allocations: a dynamic structural model of birth weight. *Journal of Labor Economics*, **30** (3), 657–706.

DICKERSON, A. and MORRIS, D. (2019). *The changing demand for skills in the UK*. Tech. rep., Centre for Vocational Educational Research (CVER).

ERIKSSON, J. G., KAJANTIE, E., OSMOND, C., THORNBURG, K. and BARKER, D. J. (2010). Boys live dangerously in the womb. *American Journal of Human Biology*, **22** (3), 330–335.

EVANS, R. W., HU, Y. and ZHAO, Z. (2010). The fertility effect of catastrophe: US hurricane births. *Journal of Population Economics*, **23** (1), 1–36.

FARMER, S. A., NELIN, T. D., FALVO, M. J. and WOLD, L. E. (2014). Ambient and household air pollution: complex triggers of disease. *American Journal of Physiology-Heart and Circulatory Physiology*, **307** (4), H467–H476.

FERRIE, J. P., ROLF, K. and TROESKEN, W. (2011). *Cognitive disparities, lead plumbing, and water chemistry: intelligence test scores and exposure to water-borne lead among World War Two US Army enlistees*. Tech. rep., National Bureau of Economic Research.

FOSTER, A., GUTIERREZ, E. and KUMAR, N. (2009). Voluntary compliance, pollution levels, and infant mortality in Mexico. *American Economic Review*, **99** (2), 191–97.

GEHRING, U., WIJGA, A. H., HOEK, G., BELLANDER, T., BERDEL, D., BRÜSKE, I., FUERTES, E., GRUZIEVA, O., HEINRICH, J., HOFFMANN, B. *et al.* (2015). Exposure to air pollution and development of asthma and rhinoconjunctivitis throughout childhood and adolescence: a population-based birth cohort study. *The Lancet Respiratory Medicine*, **3** (12), 933–942.

GRENNFELT, P., ENGLERYD, A., FORSIUS, M., HOV, Ø., RODHE, H. and COWLING, E. (2020). Acid rain and air pollution: 50 years of progress in environmental science and policy. *Ambio*, **49** (4), 849–864.

GUPTA, R. P. and STRACHAN, D. P. (2017). Ventilatory function as a predictor of mortality in lifelong non-smokers: evidence from large british cohort studies. *British Medical Journal open*, **7** (7), e015381.

HA, M.-H., LEE, D.-H. and JACOBS JR, D. R. (2007). Association between serum concentrations of persistent organic pollutants and self-reported cardiovascular disease prevalence: results from the National Health and Nutrition Examination Survey, 1999–2002. *Environmental Health Perspectives*, **115** (8), 1204–1209.

HAMADANI, J. D., TOFAIL, F., HUDA, S. N., ALAM, D. S., RIDOUT, D. A., ATTANASIO, O. and GRANTHAM-MCGREGOR, S. M. (2014). Cognitive deficit and poverty in the first 5 years of childhood in Bangladesh. *Pediatrics*, **134** (4), e1001–e1008.

HANLON, W. W. (2018). *London fog: A century of pollution and mortality, 1866-1965*. Tech. rep., National Bureau of Economic Research.

HARDING, R. and MARITZ, G. (2012). Maternal and fetal origins of lung disease in adulthood. In *Seminars in Fetal and Neonatal Medicine*, Elsevier, vol. 17, pp. 67–72.

HARRISON, R. G. (2006). Urban smoke concentrations at kew, london, 1898–2004. *Atmospheric Environment*, **40** (18), 3327–3332.

HE, J., LIU, H. and SALVO, A. (2019). Severe air pollution and labor productivity: Evidence from industrial towns in China. *American Economic Journal: Applied Economics*, **11** (1), 173–201.

HSIN, A. (2012). Is biology destiny? Birth weight and differential parental treatment. *Demography*, **49** (4), 1385–1405.

ILIFF, A. and LEE, V. A. (1952). Pulse rate, respiratory rate, and body temperature of children between two months and eighteen years of age. *Child Development*, pp. 237–245.

ISEN, A., ROSSIN-SLATER, M. and WALKER, W. R. (2017). Every breath you take—every dollar you'll make: The long-term consequences of the clean air act of 1970. *Journal of Political Economy*, **125** (3), 848–902.

JAMES GAUDERMANN, W., MCCONNELL, R., GILLILAND, F., LONDON, S., THOMAS, D., AVOL, E., VORA, H., BERHANE, K., RAPPAPORT, E. B., LURMANN, F. *et al.* (2000). Association between air pollution and lung function growth in southern California children. *American Journal of Respiratory and Critical Care Medicine*, **162** (4), 1383–1390.

JANKE, K., JOHNSTON, D. W., PROPPER, C. and SHIELDS, M. A. (2020). The causal effect of education on chronic health conditions in the UK. *Journal of Health Economics*, **70**, 102252.

JAYACHANDRAN, S. (2009). Air quality and early-life mortality evidence from Indonesia's wildfires. *Journal of Human Resources*, **44** (4), 916–954.

JIN, Y., ANDERSSON, H. and ZHANG, S. (2020). Do preferences to reduce health risks related to air pollution depend on illness type? Evidence from a choice experiment in Beijing, China. *Journal of Environmental Economics and Management*, **103**, 102355.

JOHNSON, J. D. and THEURER, W. M. (2014). A stepwise approach to the interpretation of pulmonary function tests. *American Family Physician*, **89** (5), 359–366.

JOHNSON, N. M., HOFFMANN, A. R., BEHLEN, J. C., LAU, C., PENDLETON, D., HARVEY, N., SHORE, R., LI, Y., CHEN, J., TIAN, Y. *et al.* (2021). Air pollution and children's health—a review of adverse effects associated with prenatal exposure from fine to ultrafine particulate matter. *Environmental Health and Preventive Medicine*, **26** (1), 1–29.

JON, E. (2015). The lethal effects of London fog. *BBC*.

KAJEKAR, R. (2007). Environmental factors and developmental outcomes in the lung. *Pharmacology & Therapeutics*, **114** (2), 129–145.

KELLY, E. (2011). The scourge of asian flu in utero exposure to pandemic influenza and the development of a cohort of british children. *Journal of Human Resources*, **46** (4), 669–694.

KHAWAND, C. *et al.* (2015). *Air Quality, Mortality, and Perinatal Health: Causal Evidence from Wildfires*. Tech. rep., Job Market Papers.

KNITTEL, C. R., MILLER, D. L. and SANDERS, N. J. (2016). Caution, drivers! Children present: Traffic, pollution, and infant health. *Review of Economics and Statistics*, **98** (2), 350–366.

KORTEN, I., RAMSEY, K. and LATZIN, P. (2017). Air pollution during pregnancy and lung development in the child. *Paediatric Respiratory Reviews*, **21**, 38–46.

KRAEMER, S. (2000). The fragile male. *British Medical Journal*, **321** (7276), 1609–1612.

KUBISZEWSKI, I., COSTANZA, R., FRANCO, C., LAWN, P., TALBERTH, J., JACKSON, T. and AYLMER, C. (2013). Beyond GDP: Measuring and achieving global genuine progress. *Ecological Economics*, **93**, 57–68.

LEKFUANGFU, W. N. and LORDAN, G. (2018). Cross cohort evidence on gendered sorting patterns in the UK: the importance of societal movements versus childhood variables.

LEON HSU, H.-H., MATHILDA CHIU, Y.-H., COULL, B. A., KLOOG, I., SCHWARTZ, J., LEE, A., WRIGHT, R. O. and WRIGHT, R. J. (2015). Prenatal particulate air pollution and asthma onset in urban children. Identifying sensitive windows and sex differences. *American Journal of Respiratory and Critical Care Medicine*, **192** (9), 1052–1059.

LIPFERT, F. (2004). Air pollution and poverty: does the sword cut both ways?

LOGAN, W. P. *et al.* (1953). Mortality in the London fog incident, 1952. *Lancet*, pp. 336–8.

LUECHINGER, S. (2014). Air pollution and infant mortality: a natural experiment from power plant desulfurization. *Journal of Health Economics*, **37**, 219–231.

LYSTER, W. (1974). Altered sex ratio after the London smog of 1952 and the brisbane flood of 1965. *BJOG: An International Journal of Obstetrics & Gynaecology*, **81** (8), 626–631.

MANJI, S., ARNOLD, C., GOWANI, S., BARTLETT, K., KAUL, V., SHARMA, S., CHAUDHARY, A. B. and SHARMA, S. (2015). How are we doing and how we get it right for children. *Evolution of the Roles of the Public and Private Sector in Early Childhood Care and Education in Efforts to Achieve EFA Goal*, **1**.

MAYOR OF LONDON (2002). 50 years on: The struggle for Air Quality in London Since the Great Smog of December 1952.

METOFFICE.GOV.UK (n.d.). The great smog of 1952.

MOLINA, T. (2021). Pollution, ability, and gender-specific investment responses to shocks. *Journal of the European Economic Association*, **19** (1), 580–619.

NANDI, A., MAZUMDAR, S. and BEHRMAN, J. R. (2018). The effect of natural disaster on fertility, birth spacing, and child sex ratio: evidence from a major earthquake in India. *Journal of Population Economics*, **31** (1), 267–293.

NANNA, F. (2020). The UK Clean Air Act, black smoke, and infant mortality. *Working Paper*.

NILSSON, J. P., HESSELIUS, P., GRÖNQVIST, H., JANKE, K., PETTERSSON-LIDBOM, P. and SKERFVING, S. (2009). The long-term effects of early childhood lead exposure: Evidence from sharp changes in local air lead levels induced by the phase-out of leaded gasoline. In *Childhood Obesity: Introducing the Issue", The Future of Children*.

PARMAN, J. *et al.* (2012). Childhood health and sibling outcomes: the shared burden of the 1918 influenza pandemic. *Work. Pap., Coll. William & Mary, Williamsburg, VA*.

PEET, E. D. (2016). Environment and human capital: The effects of early-life exposure to pollutants in the philippines. In *2016 Annual Meeting*, PAA.

PELLEGRINO, R., VIEGI, G., BRUSASCO, V., CRAPO, R., BURGOS, F., CASABURI, R., COATES, A., VAN DER GRINTEN, C., GUSTAFSSON, P., HANKINSON, J. *et al.* (2005). Interpretative strategies for lung function tests. *European Respiratory Journal*, **26** (5), 948–968.

PHILLIPS, D. I., OSMOND, C., SOUTHALL, H., AUCOTT, P., JONES, A. and HOLGATE, S. T. (2018). Evaluating the long-term consequences of air pollution in early life: geographical correlations between coal consumption in 1951/1952 and current mortality in England and Wales. *British Medical Journal open*, **8** (4).

RESTREPO, B. (2011). Who compensates and who reinforces? Parental investment responses to child endowment shocks. *Working Paper, Ohio State University, Columbus*.

RODGERS, J. L., JOHN, C. A. S. and COLEMAN, R. (2005). Did fertility go up after the Oklahoma City bombing? an analysis of births in metropolitan counties in Oklahoma, 1990–1999. *Demography*, **42** (4), 675–692.

ROSALES-RUEDA, M. and TRIYANA, M. (2019). The persistent effects of early-life exposure to air pollution evidence from the indonesian forest fires. *Journal of Human Resources*, **54** (4), 1037–1080.

ROSENZWEIG, M. R. and ZHANG, J. (2009). Do population control policies induce more human capital investment? Twins, birth weight and China's "one-child" policy. *The Review of Economic Studies*, **76** (3), 1149–1174.

ROTH, S. (2017). Air pollution, educational achievements, and human capital formation. *IZA World of Labor*.

ROYCHOWDHURY, A. and SOMVANSHI, A. (2020). Breathing space; how to track and report air pollution under the national clean air programme. *Center for Science and Environment*.

ROYER, H. (2009). Separated at girth: US twin estimates of the effects of birth weight. *American Economic Journal: Applied Economics*, **1** (1), 49–85.

SANDERS, N. J. (2012). What doesn't kill you makes you weaker prenatal pollution exposure and educational outcomes. *Journal of Human Resources*, **47** (3), 826–850.

SELGRADE, M. K., BLAIN, R. B., FEDAK, K. M. and CAWLEY, M. A. (2013). Potential risk of asthma associated with in utero exposure to xenobiotics. *Birth Defects Research Part C: Embryo Today: Reviews*, **99** (1), 1–13.

SHANKARDASS, K., MCCONNELL, R., JERRETT, M., MILAM, J., RICHARDSON, J. and BERHANE, K. (2009). Parental stress increases the effect of traffic-related air pollution on childhood asthma incidence. *Proceedings of the National Academy of Sciences*, **106** (30), 12406–12411.

SINGH, P., DEY, S., CHOWDHURY, S. and BALI, K. (2019). Early life exposure to outdoor air pollution: effect on child health in India.

SUN, C., KAHN, M. E. and ZHENG, S. (2017). Self-protection investment exacerbates air pollution exposure inequality in urban China. *Ecological Economics*, **131**, 468–474.

TANAKA, S. (2015). Environmental regulations on air pollution in China and their impact on infant mortality. *Journal of Health Economics*, **42**, 90–103.

TURIEL, J. S. and KAUFMANN, R. K. (2021). Evidence of air quality data misreporting in China: An impulse indicator saturation model comparison of local government-reported and us embassy-reported pm2. 5 concentrations (2015–2017). *Plos one*, **16** (4), e0249063.

VENKATARAMANI, A. S. (2012). Early life exposure to malaria and cognition in adulthood: evidence from Mexico. *Journal of Health Economics*, **31** (5), 767–780.

VESTBO, J., HURD, S. S., AGUSTÍ, A. G., JONES, P. W., VOGELMEIER, C., ANZUETO, A., BARNES, P. J., FABBRI, L. M., MARTINEZ, F. J., NISHIMURA, M. *et al.* (2013). Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. *American Journal of Respiratory and Critical Care Medicine*, **187** (4), 347–365.

VON HINKE KESSLER SCHOLDER, S., WEHBY, G. L., LEWIS, S. and ZUCCOLO, L. (2014). Alcohol exposure in utero and child academic achievement. *The Economic Journal*, **124** (576), 634–667.

VYAS, S. (2019). The child health impacts of coal: evidence from India's coal expansion. *Available at SSRN 3507883*.

WANG, G., ZHANG, R., GOMEZ, M. E., YANG, L., ZAMORA, M. L., HU, M., LIN, Y., PENG, J., GUO, S., MENG, J. *et al.* (2016). Persistent sulfate formation from London fog to chinese haze. *Proceedings of the National Academy of Sciences*, **113** (48), 13630–13635.

WETTER, D. W., COFTA-GUNN, L., IRVIN, J. E., FOULADI, R. T., WRIGHT, K., DAZA, P., MAZAS, C., CINCIRIPINI, P. M. and GRITZ, E. R. (2005). What accounts for the association of education and smoking cessation? *Preventive Medicine*, **40** (4), 452–460.

WILKINS, E. (1954). Air pollution aspects of the London fog of December 1952. *Quarterly Journal of the Royal Meteorological Society*, **80** (344), 267–271.

WORLD HEALTH ORGANIZATION (2018). Ambient (outdoor) air pollution.

XU, Y. and XU, X. (2020). Days with both extreme heat and extreme air pollution are becoming more common – which can't be a good thing for global health. *The Conversation*.

YANG, M. and CHOU, S.-Y. (2015). *Impacts of being downwind of a coal-fired power plant on infant health at birth: Evidence from the precedent-setting portland rule*. Tech. rep., National Bureau of Economic Research.

YANG, Y., LIU, X., QU, Y., WANG, J., AN, J., ZHANG, Y. and ZHANG, F. (2015). Formation mechanism of continuous extreme haze episodes in the megacity Beijing, China, in January 2013. *Atmospheric Research*, **155**, 192–203.

Appendices

A Literature Review

Table A.1: Literature review

Paper	Objective	Data	Methodology	Conclusions
Literature GLS				
Bharadwaj et al. (2016)	Estimate the effect of having been exposed to the GLS in-utero or at early childhood on asthma prevalence during childhood and adulthood	English Longitudinal Study of Ageing (ELSA) (1945-1955)	DID exploring the GLS	Exposure to the GLS in-utero increases asthma rates (only significant for childhood asthma)
Ball (2018)	Estimate the effect of having been exposed to the GLS in-utero or at early childhood on miscarriage and childhood mortality, hours worked, educational attainment, and cancer prevalence	Office of National Statistics Longitudinal Study (1971, 1981, 1991, 2001, 2011)	DID exploring the GLS	Exposure to the GLS in-utero has a strong detrimental effects on childhood mortality, hours worked, educational attainment, and cancer outcomes
Literature other events				
Singh et al. (2019)	Estimate the effect of exposure to air pollution in-utero on child height and weight in India	Demographic and Health Survey of India (2016)	IV relying on the upwind biomass of burning events in neighbouring areas	Exposure to air pollution in-utero reduces height-for-age and weight-for-age
Sanders (2012)	Estimate the effects of exposure to total suspended particulate in-utero on high school test scores in the US	Texas Assessment of Academic Skills (TAAS) (1994-2002)	IV exploiting county-level changes in relative manufacturing employment	Lower levels of suspended particulates in-utero increase high school scores
Bharadwaj et al. (2017)	Estimate the effects of exposure to pollution at the period of gestation on high school test scores in Chile	SIMCE (2002-2010)	OLS considering in-sibling variation	CO exposure reduces fourth grade math scores and language test scores
Almond et al. (2009)	Estimate the effects of exposure to radiation at the period of gestation on health and school outcomes in Sweden	Administrative data on compulsory school records (1983-1988)	OLS exploring variation in the exposure to Chernobyl's radioactive fallout	Exposure to radiation in-utero has no effect on birth weight, hospitalizations, and neoplasms and diseases of the blood, but affects school outcomes in a detrimental manner
Nilsson et al. (2009)	Estimate the effect of lead reductions in Sweden on high school GPA and cognition test scores, high school completion, educational attainment, labour market earnings, and teenage motherhood.	Swedish moss survey sample for the years (1975, 1980, 1985); data from the Institute for Labor Market Policy Evaluation (IFAU) and military enrolment test scores in Sweden (2004)	OLS that accounts phase-out of leaded gasoline in different municipalities	Exposure to lead at childhood has a detrimental effect on educational outcomes and on adulthood labor outcomes at ages 20-32. Low SES children are more affected than high SES children.
Black et al. (2013)	Estimate the effects of exposure to radiation at the period of gestation on IQ scores, education, earnings, and adult height in Norway	Norwegian Registry Data and Norwegian military records (up to 2009)	OLS exploring variation in variation driven by nuclear weapon testing during the 50s and 60s	Exposure to radiation in-utero reduces IQ score at age 18. This lower IQ is transmitted to the next generation.
Isen et al. (2017)	Estimate effect of early childhood exposure to pollution on labour force participation and wages at age 30	US Census Bureau's Longitudinal Employer Household Dynamics (1998-2007) and air pollution monitoring data from the EPA	IV based on changes in air pollution driven by the 1970 Clean Air Act	Reducing exposure to lead at childhood increses waves at age 30

Source: Own elabortation

B Factor analysis

Table B.1: Outcomes

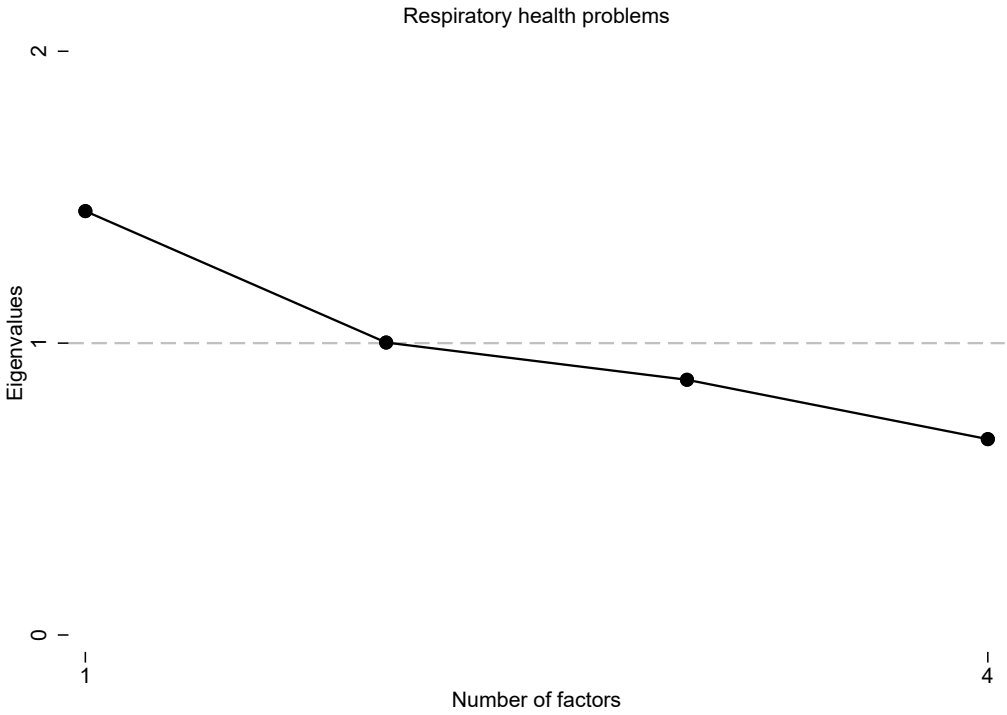
Health Outcomes	Description	Mean	SD
Indicator respiratory health	Standardized factor of *Diag. Dr. (ever)* , *Hospi. respi. from 1997-2020* , *Current self-reported drugs respi.* , and *5th pct. spiro z-score FEV1/FVC at interview date*	0	1
Diag. Dr. (ever)	Self-reported information about ever having been diagnosed by a doctor to suffer respiratory problems	0.66	0.47
Hospi. respi. from 1997-2020	Administrative data of having been hospitalized due to respiratory causes from 1997 to 2020	0.22	0.42
Current self-reported drugs respi.	Self-reported information about current consumption of respiratory prescribed medications	0.53	0.23
5th pct. spiro z-score FEV1/FVC at interview date	Data on breath spirometry is measured using a Vitalograph Pneumotrac 6800. The Z-score of the FEV1/FVC ratio (Tiffeneau-Pinelli index) is calculated. Takes into account the gender, age, and height of individuals and is restricted to whites. A dummy variable is generated, capturing if individuals are at the 5th percentile or lower of this ratio	0.51	0.22

Note: UK Biobank

Table B.2: Timeline outcome variable

Outcome variables	Before 1997	1997	2006–2010	2011–2020
Ever diagnosed of respiratory health problems	✓	✓	✓	✗
Hospitalization due to respiratory causes	✗	✓	✓	✓
Current consumption of respiratory health medication	✗	✗	✓	✗
Current results of spirometry test	✗	✗	✓	✗

Figure B.1: Screeplot factors



Source: UK Biobank.

Table B.3: Rotated loadings of factor analysis

	Factor1	Factor2	Uniqueness
Hospi. respi., from 1997-2020	0.76	-0.05	0.43
Current self-reported, drugs respi.	0.75	-0.05	0.43
Ever self-reported, respi.	0.56	0.14	0.67
5th pct. spiro, z-score FEV1/FVC, at interview date	-0.02	0.99	0.02

Source: UK Biobank.

C Extended Results

Table C.1: Linear regressions estimated through OLS – Income and pollution level as outcome and SES (age completed education) as main variable of interest

VARIABLES	(1) Income level
SES (age completed education)	0.40***
	(0.050)
Observations	123
Mean	2.679

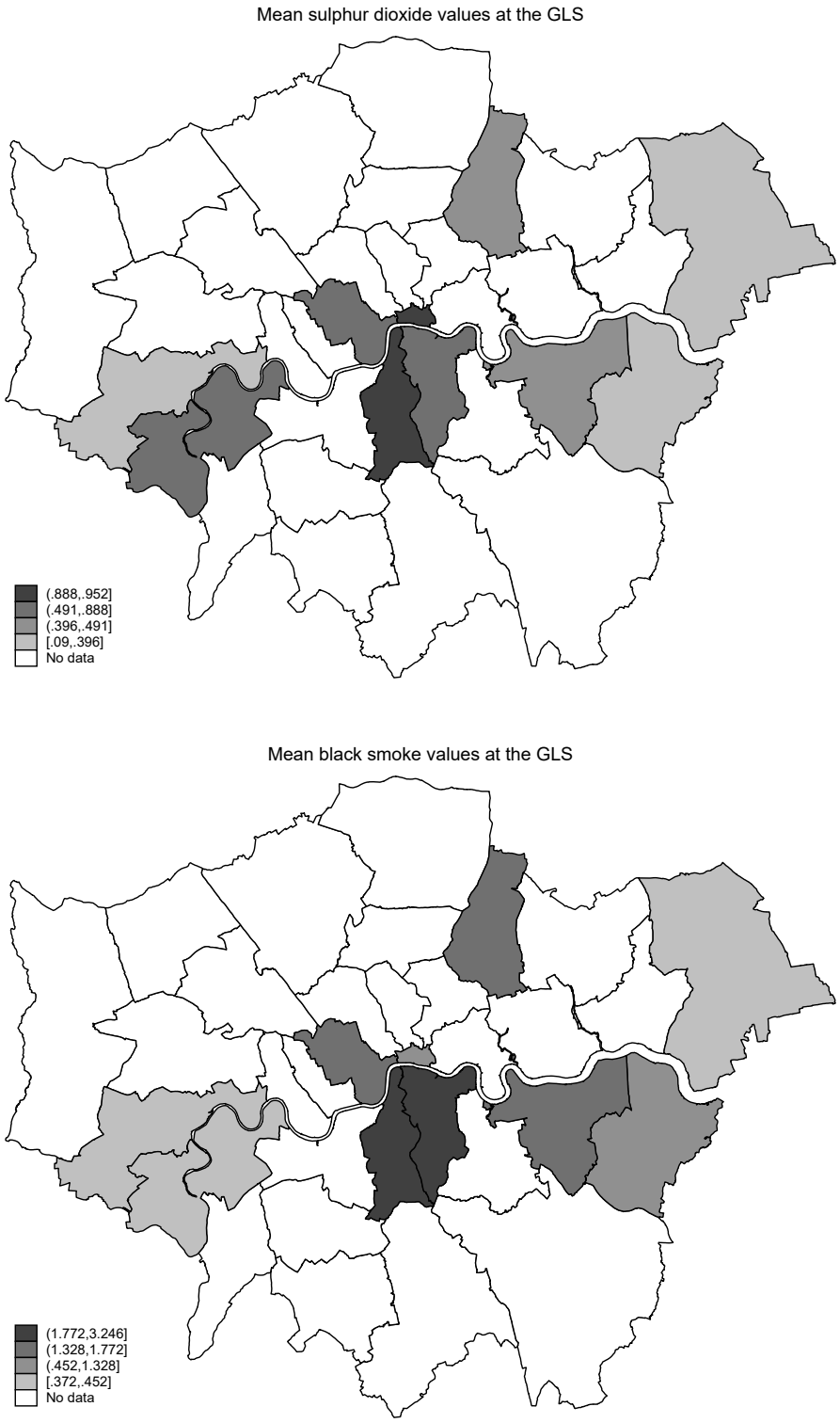
Note: The average age defines SES status completed formal education by county level for UK Biobank sample individuals born from August 1956 to August 1966. SES is considered separately for London and the rest of England. Lower values in age completed formal education proxy lower SES levels of counties. Income considers five categories: (1) Less than, 18,000; (2) 18,000 to 30,999; (3) 31,000 to 51,999, (4) 52,000 to 100,000, (5) Greater than 100,000. *Source*: UK Biobank.

Table C.2: Number of individuals born in London before and after the GLS

	In-utero or age 0-1 at GLS		
Born in London	No	Yes	Total
No	16,955	21,223	38,178
Yes	2,418	3,227	5,645
Total	19,373	24,450	43,823

Note: Sample restricted to non-missing observations for all outcomes, white ethnicity and to December 1950 to August 1953 and March 1954 to July 1956. Self-reported information of year and place of birth. *Source*: UK Biobank.

Figure C.1: Map mean air pollution by borough



Source: Wilkins (1954)

Table C.3: Multivariate linear regressions estimated through OLS – Respiratory outcomes and being born in London during GLS as interest event – Different time windows

	(1)	(2)	(3)	(4)	(5)
		1950m12–1953m8	1950m12–1953m8	1950m12–1953m8	1950m12–1953m8
VARIABLES	1950m12–1956m7	& 1954m8–1956m7	& 1954m8–1957m1	& 1955m8–1956m7	& 1955m8–1958m1
Born in London*1st trim. preg. at GLS	0.16**	0.19**	0.18**	0.17***	0.14**
	(0.065)	(0.074)	(0.072)	(0.061)	(0.062)
Born in London*2nd trim. preg. at GLS	0.03	0.06	0.05	0.04	0.01
	(0.063)	(0.068)	(0.065)	(0.068)	(0.065)
Born in London*3rd trim. preg. at GLS	-0.06	-0.02	-0.03	-0.04	-0.07
	(0.049)	(0.052)	(0.050)	(0.047)	(0.051)
Observations	47,879	40,177	43,806	32,192	43,342
Baseline covariates	✓	✓	✓	✓	✓

Note: Standard errors clustered at postal district level in parenthesis, *** p<0.01, ** p<0.05, and * p<0.10. Sample restricted to non-missing observations for all outcomes, white ethnicity and different time windows. The outcome is a standardised factor of having been hospitalised due to respiratory problems from 1997 to 2020, having ever been diagnosed by a doctor as suffering respiratory health problems, presenting spirometry values in the lowest fifth percentile of the sample at the time of the interview, and consuming prescription drugs for respiratory health problems at the time of the interview. Higher values indicate worse respiratory health and vice-versa. Baseline covariates control for being born in London or the rest of England, year-month birth FE, gender and year and month of the interview fixed effects. *Source*: UK Biobank.

Table C.4: Multivariate linear regressions estimated through OLS – Collapsed number of observations as outcome and being born in London during the GLS as interest event

	(1)	(2)	(3)
	OLS	OLS	Poisson
VARIABLES	Nr. Observations	ln(Nr. Observations)	Nr. Observations
Born in London*1st trim. preg. at GLS	-21.79	-0.10**	-0.12***
	(16.460)	(0.041)	(0.036)
Born in London*2nd trim. preg. at GLS	-38.29**	-0.03	-0.05
	(18.905)	(0.046)	(0.040)
Born in London*3rd trim. preg. at GLS	3.38	0.08	0.07*
	(13.873)	(0.050)	(0.043)
Observations	244	244	244
Baseline covariates	✓	✓	✓

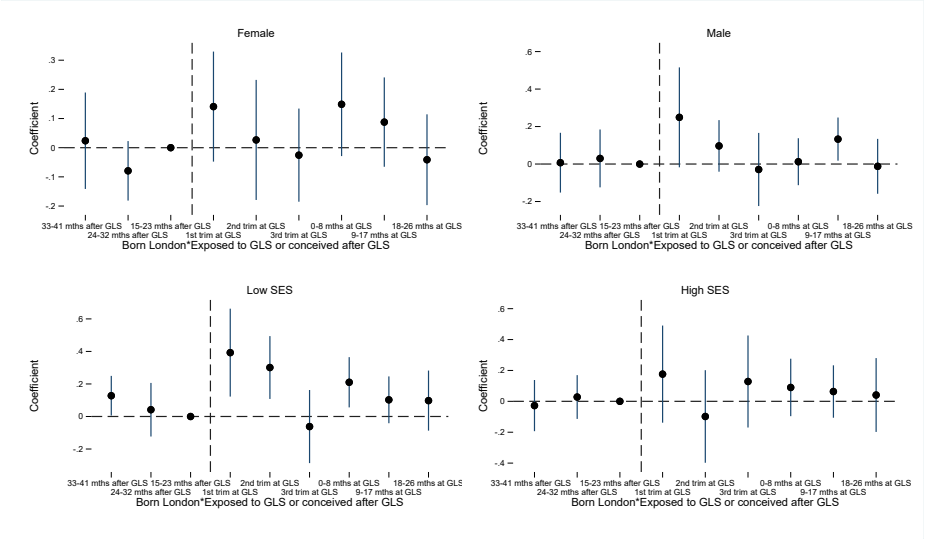
Note: Robust standard errors in parenthesis, *** p<0.01, ** p<0.05, and * p<0.10. Sample restricted to non-missing observations for all outcomes, white ethnicity and from December 1952 to August 1953 and from March 1954 to July 1956. The outcome is the collapsed number of observations at the treatment and cohort levels. *Source*: UK Biobank.

Table C.5: Multivariate linear regressions estimated through OLS – Individual respiratory health outcomes and being born in London during the GLS as interest variable with the largest sample for each outcome

VARIABLES	(1) Diag. Dr. respi (ever)	(2) Hospi. respi. from 1997-2020	(3) Current self-reported drugs respi.	(4) 5th pct. spiro z-score FEV1/FVC at interview date
Born in London*1st trim. preg. at GLS	5.08**	5.08**	1.96**	0.88
	(2.485)	(2.545)	(0.947)	(1.413)
Born in London*2nd trim. preg. at GLS	1.81	2.36	-1.41	1.94
	(2.378)	(2.443)	(1.022)	(1.286)
Born in London*3rd trim. preg. at GLS	0.16	1.86	-1.24	0.27
	(1.748)	(2.502)	(0.825)	(1.232)
Observations	37,743	31,303	37,743	30,868
Baseline covariates	✓	✓	✓	✓
Mean	64.89	23.26	5.17	5.06

Note: Standard errors clustered at the postal district level in parenthesis, *** p<0.01, ** p<0.05, and * p<0.10. Sample restricted to non-missing observations for all outcomes, white ethnicity and from December 1952 to August 1953 and from March 1954 to July 1956. Baseline covariates control for being born in London or the rest of England, year-month birth FE, gender and year and month of the interview fixed effects. *Source*: UK Biobank.

Figure C.2: Parallel trend assumption and heterogeneity



Note: Standard errors clustered at the postal district level and 95% confidence intervals. Sample restricted to non-missing observations for all outcomes, white ethnicity and from December 1952 to August 1953 and from March 1954 to July 1956. The outcome is a standardised factor of having been hospitalised due to respiratory problems from 1997 to 2020, having ever been diagnosed by a doctor as suffering respiratory health problems, presenting spirometry values in the lowest fifth percentile of the sample at the time of the interview, and consuming prescription drugs for respiratory health problems at the time of the interview. Higher values indicate worse respiratory health and vice-versa. Baseline covariates control for being born in London or the rest of England, year-month birth FE, gender and year and month of the interview fixed effects. *Source*: UK Biobank.

Table C.6: Multivariate linear regressions estimated through OLS – Respiratory outcomes and being born in London during GLS as interest event – Controlling for UK Biobank assessment centre and county of birth

VARIABLES	(1) Total	(2) Total	(3) Total
Born in London*1st trim. preg. at GLS	0.20***	0.19**	0.19**
	(0.072)	(0.076)	(0.076)
Born in London*2nd trim. preg. at GLS	0.05	0.07	0.06
	(0.065)	(0.062)	(0.064)
Born in London*3rd trim. preg. at GLS	-0.01	-0.02	-0.01
	(0.055)	(0.057)	(0.057)
Observations	25,436	25,436	25,436
Baseline covariates	✓	✓	✓
Controlling for assessment centre	✓	✗	✓
County of birth FE	✗	✓	✓

Note: Standard errors clustered at postal district level in parenthesis, *** p<0.01, ** p<0.05, and * p<0.10. Sample restricted to non-missing observations for all outcomes, white ethnicity and from December 1952 to August 1953 and from March 1954 to July 1956. The outcome is a standardised factor of having been hospitalised due to respiratory problems from 1997 to 2020, having ever been diagnosed by a doctor as suffering respiratory health problems, presenting spirometry values in the lowest fifth percentile of the sample at the time of the interview, and consuming prescription drugs for respiratory health problems at the time of the interview. Higher values indicate worse respiratory health and vice-versa. Baseline covariates control for being born in London or the rest of England, year-month birth FE, gender and year and month of the interview fixed effects. *Source*: UK Biobank.

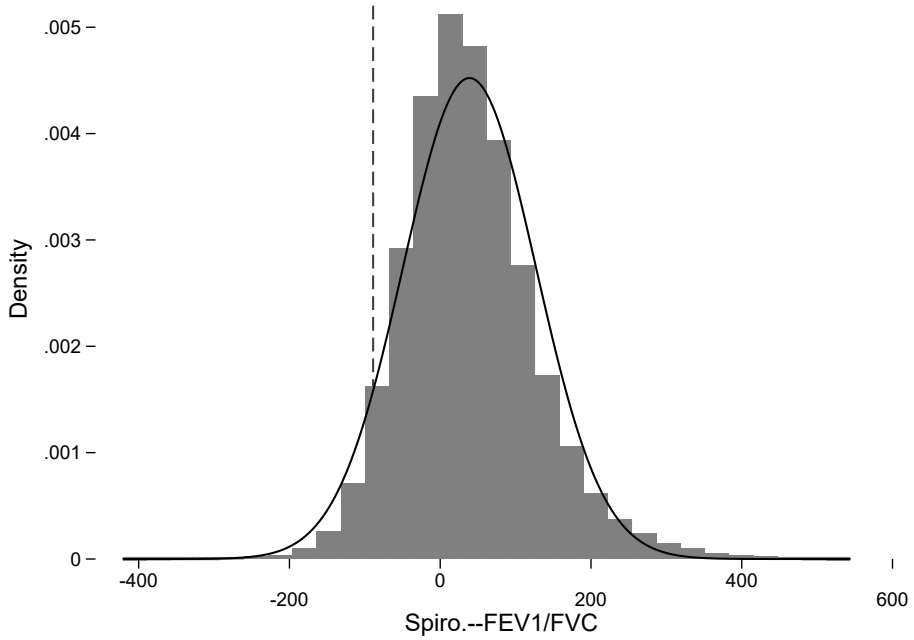
D Spirometry

According to Johnson and Theurer (2014) the first step to diagnosing obstructive or restrictive respiratory problems is the analysis of the FEV1/FVC ratio. Then, in a second step, FVC is used to determine if these respiratory problems are obstructive, such as COPD or asthma; restrictive such as fibrosis or interstitial lung diseases; or mixed. This second step depends on the FVC values.

FEV1 measures expiratory volume in one second: what is the amount of air a person can exhale in one second. FVC is forced vital capacity, which is the amount of air a person can forcefully and quickly exhale after taking a deep breath. In a first step, the FEV1/FVC considers if the amount exhaled in one second relative to the total capacity is abnormally low. In the second step, the denominator FVC is examined to explore what is driving the abnormality in the ratio. This chapter explores overall respiratory problems and not a specific condition. Therefore the ratio FEV1/FVC is considered.

The ratio FEV1/FVC predicts an obstructive defect if it is below 0.7 (Vestbo *et al.*, 2013). Another form of predicting respiratory problems is focusing on the lower limit of normal (LLN), with individuals scoring below the fift percentile in a sample with normally distributed respiratory outcomes being classified as suffering respiratory problems (Pellegrino *et al.*, 2005). This second definition is used in this chapter to use results from z-score that controls for age, race and height. The distribution of this variable and the lower LLN can be seen in Figure D.1 in the appendix. Individuals scoring below that threshold are assigned a value of one and zero otherwise.

Figure D.1: Distribution z-score FEV1/FVC



Note: Sample restricted to non-missing observations for all outcomes, white ethnicity and to individuals born from December 1950 to August 1953 and from March 1954 to July 1956. *Source*: UK Biobank.

Essay 2: Cognitive activity at work at main lifetime occupation and dementia

1 Introduction

The rising prevalence of dementia is likely to become one of the biggest health challenges in the near future due to the unprecedented increase in life expectancy over the past century. Globally, in 2017 there were 962 million individuals aged 60 or older, and this is predicted to more than double by 2050 and more than triple by 2100 (United Nations, Department of Economic and Social Affairs, Population Division, 2017). In the US, it was estimated that the elderly population will almost double from 45 million in 2017 to 70 million by 2030 (Ortman *et al.*, 2014). The main concern with an ageing population is the rise in age-related health problems, such as dementia. In 2020 50 million individuals were suffering from dementia worldwide, with around 10 million new cases every year. Predictions suggest around 82 million individuals will suffer dementia by 2030 and 152 million by 2050 (World Health Organization, 2020). A large share of these cases will be driven by the rise in life expectancies in low- and middle-income countries. Alzheimer's disease (AD) is the most common form of dementia (60-80% of all cases) (Alzheimer's Association, 2020). In 2020 five million individuals suffered AD in the US, and it was the sixth cause of death. Even more worrying than the high prevalence of AD is its exponential growing trend over time, which is expected to triple by 2050, affecting 14 million individuals in the US (Chandra *et al.*, 2020; Center of Disease, 2020). Furthermore, AD has an enormous economic cost that in 2010 in the US was of $157-$236 billion and is predicted to increase sharply to $379-$500 billion by 2040 (Hurd *et al.*, 2013). Given that currently there is no cure for dementia, the best way to tackle this problem is prevention. Thus, it is critical to improving the understanding of the determinants of dementia, and economists play a crucial role in this research field. According to Chandra *et al.* (2020) "The prospects for economists working in many different fields to contribute to our understanding of AD appear to us to be very bright.".

This essay asks if cognitive activity developed at the most prolonged lifetime occupation of individuals can reduce individuals probability of suffering dementia at an older age. The primary hypothesis is that cognitive activity at work could improve individuals' cognitive reserve and protect them from suffering dementia at an older age.

An extensive database of older adults with information about their mental health and the work activities performed at their most prolonged lifetime occupation is analysed to test this hypothesis. This database is composed of the Health and Retirement Study (HRS) and the O*NET. The HRS is a longitudinal and representative sample of approximately 20,000 individuals aged 50 and above in the US. The restricted version of the HRS is used, which gives access to detailed information on individuals' most prolonged lifetime occupations. The O*NET data provides detailed information on work activities performed in different occupations. Both databases are combined, and the results of a range of econometric models show that cognitive activities at work are protective towards suffering dementia at an older age.

The analysis focuses on understanding whether the association between cognitive activity at work and predicted dementia are robust to the inclusion of an extensive set of demographic, education, occupation, industry, and genetic controls. Individual fixed-effects are also included in the regression specification and estimate how the effects of cognitive activity at work vary with age. The results from these analyses suggest a robust association between cognitive activity at work and predicted dementia. Using a conservative regression specification, which includes measures of polygenic risk scores for general cognition and Alzheimer's disease (AD), no evidence is found of genetic impacts on the level of cognitive activity at work in a person's most prolonged lifetime occupation (no genetic related endogenous sorting). Even though the estimated association between cognitive activity at work and predicted dementia is reduced by 70% when all controls are added (relative to the raw correlation), the remaining association is still economically and statistically significant. Furthermore, the individual fixed-effects specification demonstrates that people with low levels of cognitive activity at work have a significantly steeper age-dementia gradient.

This essay makes essential contributions to the literature, adding several dimensions of workplace characteristics. It considers the variability in cognitive activity at work within 11 main occupations and 13 primary industries, occupations involving similar loads in non-cognitive and physical work activities and with similar work context, and considering individuals with a similar genetic predisposition of suffering dementia. It shows that there is genetic selection into occupations with high levels of cognitive activity at work and controls for it. Controlling for intra-individual fixed characteristics also shows that individuals with higher loads in cognitive activities at work have lower decline rates towards dementia. Therefore, the rates of dementia are affected by cognitive activities at work and the decline towards dementia within individuals.

2 Mechanism – Cognitive Reserve

According to the 'use it or lose it' hypothesis, activities performed in one's occupation may affect one's likelihood of suffering from dementia in old age. This theory suggests that individuals performing cognitively demanding activities regularly develop higher cognition levels and present lower dementia rates (Livingston *et al.*, 2020). It could explain that even if the absolute number of individuals suffering dementia is currently increasing dramatically, age and sex-specific rates have declined in Europe and North America by 13% per decade over the past 25 years (Wolters *et al.*, 2020). A suggested explanation for this is younger cohorts having access to better education and more cognitive activities at work, protecting them from dementia through the cognitive reserve.

Cognitive reserve refers to the brain's resilience to physiological neuronal changes related to the ageing process (Stern, 2002,0). A higher cognitive reserve leads to more efficient use of the networks in the brain and improved ability to use alternative neural networks. In addition, previous medical literature shows robust evidence that a high cognitive reserve reduces the risk of dementia (Valenzuela and Sachdev, 2006). It has been shown that cognitive stimulation can induce the brain to change by forming new connections (synapses) between brain cells (neurons), called neuron-plasticity. A large group of trials have studied the effects of cognitive training on cognitive performance. They conclude that for healthy adults, training improves cognition levels[1] (Butler *et al.*, 2018; Kivipelto *et al.*, 2018). Similarly, physical activity and stimulating cognitive abilities have been suggested to be protective for dementia. In a review of the literature, Cheng (2016) concludes that physical activity during the life-cycle preserved the neuronal structure of the brain, while cognitive activity strengthens the functioning and plasticity of neural circuits. Thus, through neuron-plasticity, cognitive stimulation may increase individuals' cognitive reserve, protecting them against developing dementia at all or shifting the onset of dementia to a later age.

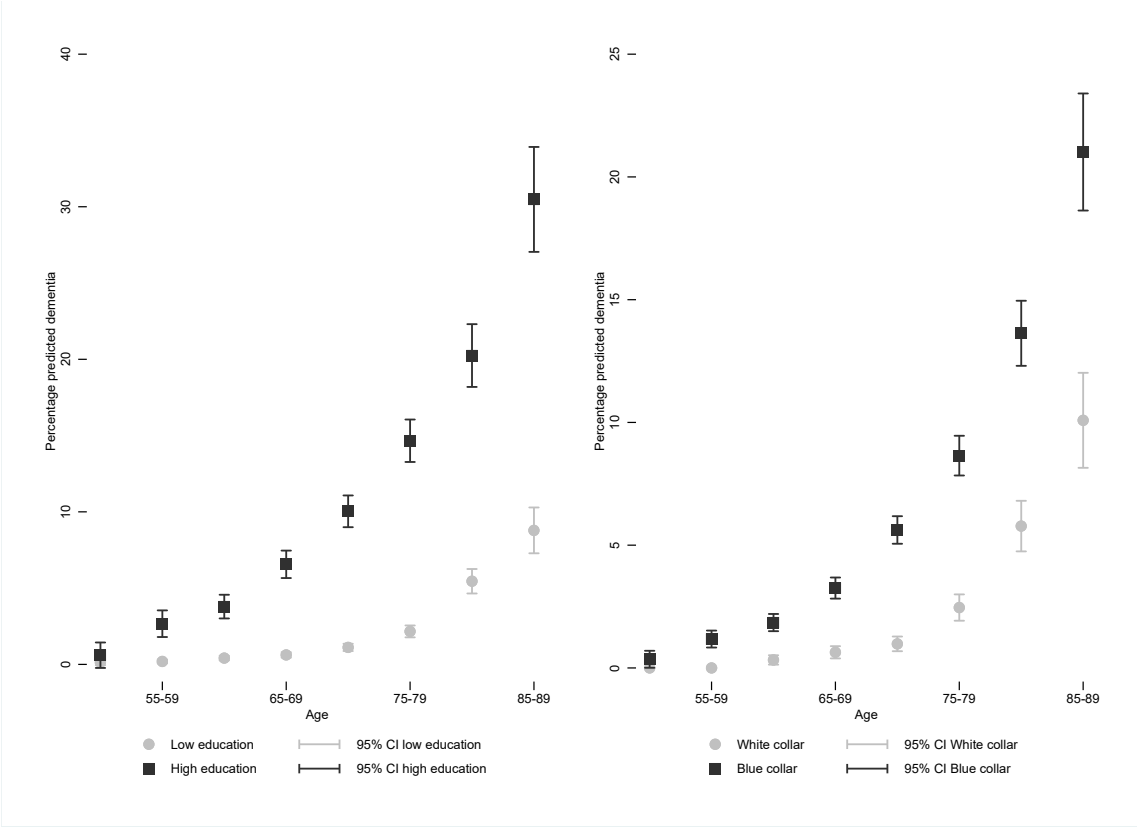
It is well known that there are large differences in the prevalence of dementia across occupation and education levels (Gurland *et al.*, 1999; Shadlen *et al.*, 2006; Andersen *et al.*, 1999; Ruitenberg *et al.*, 2001; Berr *et al.*, 2005). These differences can be seen in Figure 1, which presents a striking gradient in the prevalence of dementia by education and occupation[2]. While at ages 50 to 54, the proportion of individuals with dementia is close to zero for all occupation and education groups, it raises exponentially with age. At ages 85 and above, there is almost 23 percentage points difference between individuals with low and high levels of education and almost 11 percentage points difference between individuals in white and blue occupations. These figures are uncontrolled, and selection into education and occupations probably explains a big part of this variability. Still, the gradient is extraordinarily large, and it is reasonable to assume that

[1] The effect of cognitive training on cognition for non-healthy individuals (e.g. mild cognitive impairment, dementia) is less clear.

[2] Descriptive results of own elaboration. Details of the data composition can be found in the Data section. Similar results are found for females, white-collar and non-white-collar occupations in Figures A.1, A.2, and A.3 in the Appendix.

workplace activities drive a part of it. Given that people in different occupations are exposed to various degrees of cognitive stimulation at the workplace, it is worth considering to what extent cognitive activities at work may contribute to the educational and occupational gradient in dementia rates. This essay tries to wipe out as much 'noise' as possible from this gradient to capture the cleanest effect of cognitive activities at work on dementia.

Figure 1: Proportion of individuals predicted to suffer dementia for education levels and collar colour occupations



Note: Sample restricted to males aged 60 to 89. *Predicted dementia:* Dementia is defined as scoring below a threshold in a psychometric test validated by Crimmins *et al.* (2011). More details of this variable can be found in appendix C. *Education:* Low education considers no high school and GED. High education considers high school and college or above. *Collar colour:* Blue-collar occupations involve skilled or unskilled manual labour occupations. White-collar occupations involve professional, managerial, or administrative occupations. *Source*: HRS.

3 Literature Review

This essay uses a similar strategy to the labour and health economics literature that has examined the effects of work activities and work context on different health and non-health related outcomes. Several outcomes have been considered, such as self-reported health outcomes (Ravesteijn *et al.*, 2018; Fletcher *et al.*, 2011; Schmitz, 2016), body mass index (Dang *et al.*, 2019; Ishizaki *et al.*, 2004), mental health (Cottini and Lucifora, 2013), cognition (Schmitz, 2016), reincorporation to the labour market after suffering cancer

(Heinesen *et al.*, 2018) and wages (Abraham and Spletzer, 2009). An example is the work of Lakdawalla and Philipson (2007), who explore the relationship between physically fitness-demanding workplace activities on body mass index (BMI). This essay uses an equivalent approach, substituting the physically fitness-demanding workplace activities with cognitive activities and BMI with dementia. Therefore, the mechanism considered in this essay is well-accepted in the economics literature and adapted to the specific scenario of dementia and workplace activities.

Considering outcomes that are more relevant to this essay, the relationship between education and dementia has received a lot of attention so far (Beard *et al.*, 1992; Stern *et al.*, 1992,9; Cobb *et al.*, 1995; Evans *et al.*, 1997; Gatz *et al.*, 2001; Karp *et al.*, 2004; Almeida *et al.*, 2015; Kemppainen *et al.*, 2008; Perneczky *et al.*, 2006; Wang *et al.*, 2017; McDowell *et al.*, 2007; Orrell and Sahakian, 1995; Ngandu *et al.*, 2007; Sattler *et al.*, 2012; Bosma *et al.*, 2003a). Lower levels of educational achievement are highly correlated with suffering dementia at old age and with neuronal structures related to dementia. It is hypothesised that individuals with higher levels of education have a greater cognitive reserve, as they engage more often in cognitively demanding activities.

Other authors consider psycho-social, and work environment correlates and study the detrimental consequences of psycho-social work environment on dementia (Wang *et al.*, 2012; Andel *et al.*, 2012; Crowe *et al.*, 2007; Sindi *et al.*, 2017). They find a significant detrimental effect of work-related stress on dementia, which suggest that specific work environments are harmful to mental health in the long run. Individuals whose most prolonged lifetime occupation involves much stress are more likely to suffer dementia.

The relationship between workplace activities and overall cognition in older adulthood has also been considered (Andel *et al.*, 2007,0; Jonaitis *et al.*, 2013; Smart *et al.*, 2014; Fisher *et al.*, 2014; Elovainio *et al.*, 2009; Potter *et al.*, 2006,0; Finkel *et al.*, 2009; Wang *et al.*, 2017; Marquié *et al.*, 2010; Jorm *et al.*, 1998; Bosma *et al.*, 2003b). Occupations involving high levels of cognitive and non-cognitive activities are associated with better cognitive ageing. There is consensus that intellectually demanding or socially engaging occupations are associated with higher levels of cognition, while physically demanding occupations are associated with lower levels of cognitive functioning and a steeper rate of cognitive decline.

A last group of papers, most similar to this essay, explores the relationship between work-related cognitive domains and dementia (Dartigues *et al.*, 1992; Stern *et al.*, 1995; Smyth *et al.*, 2004; Andel *et al.*, 2005; Kröger *et al.*, 2008; Boots *et al.*, 2015; Hasselgren *et al.*, 2018; Stern *et al.*, 1995; Qiu *et al.*, 2003; Rovio *et al.*, 2007; Seidler *et al.*, 2004; Then *et al.*, 2017; Karp *et al.*, 2009). Occupations with high levels of cognitive and non-cognitive workplace activities are associated with a lower prevalence of dementia. Some of these papers use similar survey data, variables and identification strategies to this essay and therefore are described in more detail.

Bosma *et al.* 2003b examine whether individuals with cognitive demanding occupations are protected from cognitive impairment, analysing the Maastricht Aging Study (MAAS). Cognitive impairment is considered, not dementia, and subjective measures of cognitive complexity at the workplace are used. Individuals with cognitive demanding occupations have lower risks of suffering cognitive impairment.

Andel *et al.* 2005 explore if working with data or social interactions is correlated with AD, analysing the Swedish Panel Study of Living Conditions of the Oldest Old. They use an objective measure of complexity in workplace activities, similar to the one in this essay, and a medically validated measure of AD. A co-twin control design is also run, considering a small sample of twins and controlling for genetics and similar environmental conditions. Their results show that working with data and social interactions is related to a reduced prevalence of AD.

Kröger *et al.* (2008) consider the relation between working with data, people and machines with dementia, analysing the Canadian Study of Health and Ageing. They use an objective measure of workplace activities, a medically validated dementia outcome, and analyse panel data considering the decline towards dementia within individuals. The high complexity of work with data and people is associated with a reduced risk of dementia.

Hasselgren *et al.* (2018) study if exposure to the work environment factors of work control, support, psychological demands, physical demands and job hazards is related to AD, analysing the H70 Birth Cohort Study and the Prospective Populations Study of Women. They use an objective measure of workplace characteristics, a medically validated measure of AD and consider the genetic predisposition towards AD, measured with the APOE $\epsilon 4$ protein. Work control reduces the risk of suffering AD, with the most substantial effect on individuals with a high genetic predisposition.

Fisher *et al.* (2014) do not consider dementia, but examine a research question related to this essay, using the same data. They estimate the correlation between work activities and cognition before and after retirement using the HRS and the O*NET database. Cognitive demanding occupations are associated with higher levels of cognitive functioning before retirement and a slower rate of cognitive decline after retirement.

This essay makes essential contributions to this literature. Compared to the work of Fisher *et al.* (2014), this essay examines a much more extreme outcome, dementia, while they considered general cognition. Work activities can affect both outcomes differently, with cognition being highly dependent on crystallised intelligence, while dementia being a medical condition related to genetic and organic factors. Compared to the rest of the literature, a much richer set of work-activities and work context controls are included. The variability in cognitive workplace activities is considered within main occupations and industries, occupations with similar loads in non-cognitive and physical work activities, and a similar work context. Previous work has captured genetic information with co-twin designs and with the APOE $\epsilon 4$ protein, while this essay

uses the rich poly-genetic risk scores of the HRS, which capture the genetic predisposition of suffering several conditions related to dementia. It obtains robust estimates and can make strong inferences about genetic selection into occupations with specific work activities. The decline towards dementia within individuals is estimated by making use of the panel structure of the HRS. Models with individual fixed-effects are run, capturing intra-individual fixed characteristics such as genetics and childhood circumstances.

4 Data

Two different data sources are used for the data analysis: the Health and Retirement Study (HRS) and the Occupational Information Network database (O*NET). Both are described below.

4.1 Health and Retirement Study

The HRS is a longitudinal study of older Americans' economic, health, marital, and retirement decisions and is a valuable resource for studying an ageing population. It initially surveyed a nationally representative sample of US adults aged 50 and above and their spouses regardless of their age. The first wave of interviews was undertaken in 1992, respondents have been re-interviewed on a biennial basis ever since, and refresher cohorts were added in 1998, 2004, and 2010.

This essay uses the RAND HRS Longitudinal File 2014, a version of the HRS that contains clean and homogeneous variables across survey years. The data contains detailed information covering various measures, including demographics, health, income, and employment history for 12 HRS survey years (1992 to 2014). Wave 1 contains variables that cannot be compared across waves, and therefore waves 2 to 12 are included, with data spanning more than 20 years (1993-2014).

An important feature of the RAND HRS Longitudinal File 2014 is that it can be merged with other HRS data products. Some of the outcomes in the general release of the HRS are aggregated to maintain confidentiality, such as occupation. This outcome is summarised into 11 categories. Detailed occupation information is available in the HRS's restricted industry and occupation database, which provides detailed occupational histories for each respondent, with over 1,000 different Standard Occupation System (SOC) codes. Linking this to the RAND HRS Longitudinal File 2014, individuals' main lifetime occupations are identified with a great level of detail.

The initial sample is composed of 203,219 observations and is restricted to individuals with non-missing information about their longest lifetime occupation and cognitive status, which reduces the sample to 125,819 observations. It is further restricted to males, which reduces the sample to 53,241. Only males are considered to minimise the reporting error related to individuals having different occupations during their life-cycle, which is assumed to be much larger for the female of that cohort. More details about this

are given in the methodology section. It is also restricted to individuals aged 60 to 85, which reduces the sample to 43,018 observations[3]. This age window is selected since individuals older than 60 start retiring and individuals older than 85 start presenting other age-related problems that can interfere with the main results. The final sample consists of 7,928 individuals and 43,018 observations.

4.2 Key variables

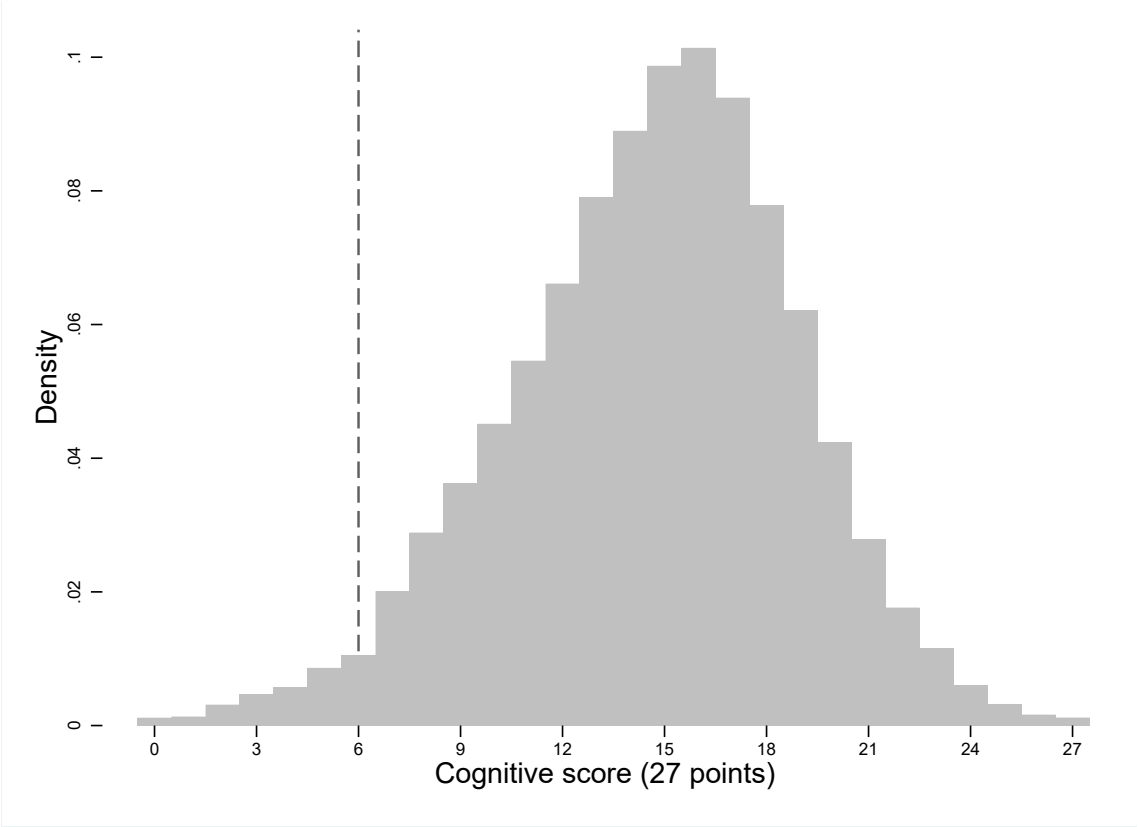
Dementia

The outcome variable is derived from a cognitive test administered to HRS respondents that were developed by Brandt *et al.* (1988) and validated by Welsh *et al.* (1993). Each survey wave respondents undergo a cognitive screening test, making it possible to measure their cognitive evolution over time. The test contains three sections: (1) 10-words immediate and delayed memory test (0-20 points). (2) 7s serial test (0-5 points). (3) A backward counting test (0-2 points)[4]. Scores on these three sections are added to produce a scale ranging from zero to 27, with higher scores capturing better levels of cognition. Following Crimmins *et al.* (2011), individuals with scores ranging from zero to six are classified as suffering from dementia. Figure 2 presents the distribution of this cognitive test and highlights that individuals predicted to suffer dementia are only at the lowest tail.

[3] For more information regarding the sample selection see Appendix B.

[4] (1) 10-words immediate and delayed memory: individuals are read a list of 10 words and have to recall it immediately and after a while. They get one point for each correctly recalled word. (2) 7s serial test: asks the respondents to subtract seven from the last number, beginning with 100 for five trials. Correct subtractions are based on the last number given so that even if one subtraction is incorrect, subsequent trials are evaluated on the given (perhaps wrong) answer. (3) Backward counting test: examines whether the respondent could successfully count backwards for ten serial numbers from 20 and 86, respectively. Two points are given if successful on the first try, one if successful on the second, and zero if not successful on either try.

Figure 2: Distribution of 27 points cognitive test



Note: Sample restricted to males aged 60 to 85. *Predicted dementia:* Dementia is defined as scoring below a threshold in a psychometric test validated by Crimmins *et al.* (2011). More details of this variable can be found in appendix C. The vertical dotted line defines a threshold for predicted dementia and the left lower tail presents the proportion of individuals with dementia. *Source*: HRS.

Information is collected through proxy respondents for individuals with communication problems due to ill health, communication disorders, or psychiatric disorders. It is crucial to include these respondents since individuals who have dementia may not respond to the survey interview themselves. Proxy respondents can not be directly used as a predictor of dementia since there are reasons other than cognitive impairment that may lead individuals to need assistance. However, based on Crimmins *et al.* (2011), it can be predicted if individuals using proxy respondents have dementia combining three tests. (1) Memory assessment, in which the proxy respondent rates the primary respondents' memory from excellent to poor (0-4 points). (2) An assessment of activities, in which the interviewer asks the proxy respondent if the main respondent has problems doing the groceries, preparing hot meals, using the phone, handling money and taking their medication (0-5 points). (3) The interviewer assessed the respondents' difficulty in answering the survey (0-2 points). These three items are added to generate a scale that ranges from zero to 11, with individuals being classified as suffering dementia if they score six to 11 points.

The threshold used to classify respondents with dementia is validated by Crimmins *et al.* (2011), using the Ageing, Demographics and Memory Study (ADAMS), which consists of a stratified random subsample

of the HRS respondents (856 individuals) receiving a detailed cognitive assessment. Respondents in the ADAMS were selected based on the score on the self- or proxy-cognitive assessment measure and undergo an intensive examination by a nurse and neuropsychology technician who runs a very detailed cognitive assessment[5]. The cut-off points of Crimmins *et al.* (2011) are very consistent with the results of ADAMS, and many papers have used them to predict dementia (Hsu and Willis, 2013; Sutin *et al.*, 2018a,0; Crimmins *et al.*, 2018; Basu, 2013; Clouston *et al.*, 2015; Lièvre *et al.*, 2008; Garcia *et al.*, 2017).

Because respondents take the cognitive tests for each survey wave, it is possible for their prediction of dementia to change from wave to wave. In other words, they can score below the threshold one wave and above the threshold the next one. Therefore, the measure of predicted dementia is modified to use a more conservative approach, accounting for this variation at an individual level. Respondents are classified as suffering dementia if, after the first wave, they are predicted to suffer dementia, the average of their subsequent scores are below the threshold. This method should help reduce miss-predictors of dementia[6].

Gianattasio *et al.* (2020) use a machine learning approach to construct alternative predictors of dementia in the HRS. Table F.1 presents estimations with their predictions of dementia as outcomes and show that there are no significant differences compared to the estimations using the predictions of Crimmins *et al.* (2011). The predictions of Crimmins *et al.* (2011) are preferred since in the results, there are no significant differences compared to the ones of Gianattasio *et al.* (2020), and these are only available for a sub-sample of the HRS. This smaller sub-sample is disadvantageous when running stratifications of the results.

Lifetime occupation

Respondents' main lifetime occupations are the occupations they hold for most of their working life and can be found in the HRS's restricted industry and occupation database. However, this data set is not consistently coded and questions regarding respondents' lifetime occupation vary across survey waves. The approach of Bugliari *et al.* (2018) is used to correct for this and to identify a consistent main lifetime occupation of individuals while minimising the loss of observations[7].

There is also a problem with occupations in the O*NET database being slightly different from those in the HRS. David and Dorn (2013) provide a solution to this issue by generating comparable occupational codes for the HRS and O*NET. They produce 330 unique and comparable occupations codes that link the HRS data with the O*NET.

[5] ADAMS respondents undertake the following psychometric tests: MMSE, Boston naming test, digit span, Symbol Digit Modality Test, animal fluency, word list three trial learning, construction praxis copying, Trail Making Test, Wechsler Memory Scale, Fuld Object Memory Test, Shipley vocabulary test and the Wrat 3 reading test. Diagnosis is determined based on detailed neuropsychological assessment and a consensus diagnosis.

[6] Further details can be found in Appendix C.

[7] Further details can be followed in Appendix D

Work activities

The O*NET database contains detailed information on the work activities undertaken in a large number of occupations[8]. A factor analysis is run on work activities with the specific variables and their loadings on the main factors are presented in Table E.1[9]. Three factors are retained based on eigenvalues larger than one and the scree-plots in Figure E.1. These factors are standardised with mean zero and standard deviation one. Factor 1 loads on non-cognitive work activities such as developing teams, motivating people, and resolving conflicts. These activities are relevant for occupations such as managers, chief executives, public administrators, and similar ones. Factor 2 loads on cognitive processing work activities such as processing information, analysing data, and thinking creatively. These activities are relevant to occupations such as mathematicians and statisticians, air traffic controllers, and similar ones. Factor 3 loads on physical demanding work activities such as performing general physical activities, handling and moving objects, and repairing and maintaining mechanical equipment. These activities are relevant to occupations such as such as air conditioning mechanics, automobile mechanics, repairers, and similar ones. These factors are labelled as: 'Non-cognitive work activities', 'Cognitive work activities', and 'Physical work activities'. This essay examines cognitive work activities, and therefore, the primary analysis is based on the second factor, while the other factors are used as controls.

Table 1 presents occupations with high and low cognitive activity scores, within six broad occupation groups that cover 86% of the sample. The table demonstrates that the generated cognitive work activities factor provides a sensible ranking of occupations: mathematicians and statisticians (mean cognitive activity score of 2.57), and biological scientists (1.95) are classified as high cognitive activity occupations, while operators of construction equipment (-2.05) and miners (-2.02) are classified as low cognitive activity occupations. It also demonstrates that there is considerable variation in predicted cognitive activity levels within broad occupation groups. For example, within the 'managers' group, accountants and auditors have a score of 1.70, while retail buyers and wholesalers have -1.24. This considerable variation highlights the need for an extensive set of covariates to be included in regressions to control non-random selection into occupations.

[8] Variables related to the work context and knowledge are used in separate factor analysis. Five work contexts and six knowledge factors are found, which are included as controls for some robustness specifications.

[9] Details for the factor analysis can be found in Appendix E.

Table 1: Ten occupations with best and worse scores for cognitive work activities factor

Occupation Groups	Three Highest Scores	Three Lowest Scores
Managers	Accountants and auditors (1.70); inspectors and compliance officers (1.46); other financial specialists (1.18)	Buyers, wholesale, and retail trade (-1.24); office supervisors (-0.93) business and promotion agents (0.79)
Professionals	Mathematicians and statisticians (2.57); biological scientist (1.95); physicists and astronomers (1.91)	Athletes, sport instructors, and officials (-1.37); musicians and composers (-0.79); clergy and religious workers (-0.75)
Sales	Sales engineers (1.10); financial services sales occupations (1.01); insurance sales occupations (0.77)	Sales supervisors and proprietors (-1.69); door-to-door sales, street sales, and news vendors (-0.98); retail salespersons and clerks (-0.37)
Office and administration	Statistical clerks (1.87); computer and peripheral equipment operators (1.63); insurance adjusters, examiners, and investigators (1.54)	Office machine operators (-1.03); mail carriers for postal service (-0.84); mail clerks outside of post office
Production, craft, and repair	Power plant operators; repairers of data processing equipment (1.19); elevators installers and repairers (1.08)	Other mining occupations (-2.02); painters, construction, and maintenance (-1.93); glaziers (-1.89)
Operators, fabricators, and laborers	Production checkers, graders, and sorters (1.93) packers, fillers, and wrappers (0.52); forge and hammer operators (0.39)	Operations engineers of construction equipment (-2.05); excavating and loading machine operators (-1.86); parking lots attendants (-1.75)

Source: HRS and O*NET.

A factor analysis is also conducted using the variables in the O*NET relative to work context and knowledge. Work context refers to physical and social factors that influence the nature of work. Knowledge refers to the knowledge of principles and facts required at work, and is organised into domains such as mathematics, medicine, and biology. Five factors are generated for the work context domain and six for the knowledge domain. These are included as covariates in the main regression models.

Genetics

Genetic data of HRS is linked to respondents and measures their propensity to suffer dementia. This data is a random subset of 26,000 participants selected to participate in enhanced face-to-face interviews and saliva specimen collection (for DNA) in 2006, 2008, 2010, and 2012. Genotyping was conducted from 2011 to 2014 using the Illumina Human Omni-2.5 Quad beadchip (HumanOmni2.5-4v1 array), with approximately 2.5 million SNPs coverage. In addition, best-guess genotypic data is used, which was imputed using approximately 21 million DNA variants from the 1,000 Genomes Project, phase I, to increase SNPs coverage. This method provides individual genetic risk scores for suffering from different health conditions. These are standardised poly-genetic risk scores (PGS) with higher values for a higher propensity of suffering specific health outcomes and vice-versa.

These weights are constructed through extensive GWAS meta-analysis studies related to the selected phenotype. When possible, meta-analyses that did not include the HRS in the discovery analysis were selected to be independent of the HRS data and avoid potential endogeneity problems. If the HRS was

included in the meta-analyses, it was requested from the consortia for the analysis to be repeated with the HRS removed.

For the analysis, samples using HRS information are restricted to 22,461 males with non-missing genetic, cognition and work activities information and who are genetical of European descent. It follows accepted practices of restricting the genetic diversity to match the genetic prole of the discovery sample as closely as possible.

Three main PGS that are closely related to dementia are used in this essay. (1) The PGS for AD, because Alzheimer's disease is the first cause of dementia. (2) The PGS for cognition, because the prediction of dementia is constructed from cognitive information. (3) The PGS for coronary arterial disease, because cardiac pathologies are one of the leading causes of dementia. The variables are standardised with mean zero, and standard deviation one, and higher values refer higher genetic predisposition[10].

5 Methodology

5.1 Identification

A range of econometric models are estimated with predicted dementia as an outcome variable and cognitive work activities in the main lifetime occupation of individuals as the main explanatory variable. It is analysed if cognitive work activities are related to the prevalence of predicted dementia at old age and if this relationship is robust to the inclusion of different controls. Multivariate linear regression models are estimated by OLS[11]. The main model is specified as follows.

$$Dem_i = \beta_0 + \beta_1 CognitiveWorkActivities_i + \beta_2 Demographics_i' + \beta_3 Education_i' + \beta_4 O^*NETFactors_i' + \beta_5 OccupationIndustry_i' + \beta_6 WealthEmployment_i' + \beta_7 Genetics_i' + \epsilon_i. \quad (1)$$

The dependent variable Dem is a categorical variable equal to one if individual i is predicted to suffer dementia and zero otherwise. $CognitiveWorkActivities$ is a standardised variable measuring the level of cognitive work activities involved in the most prolonged lifetime occupations of individuals. β_1 is the primary coefficient of interest, and it can be interpreted as having a main lifetime occupation with an additional standard deviation in cognitive work activities, changing the probability of suffering dementia at old age in β_1 percentage points.

[10] An example of the distribution of these variables can be found in Figure G.1 in Appendix G.

[11] Given the binary nature of the dependent variable, sensitivity of the results is tested estimating non-linear, probit and logit models which can be found in Table F.2 in Appendix F. These non-linear models produce similar results.

Once the unconditional correlation between cognitive work activities and predicted dementia is examined, different controls are included in a step-wise manner to examine how robust the relation of cognitive work activities and predicted dementia is.

$Demographics$ is a vector of demographic controls including race, proxy respondent, parental education, and dummy variables for age, survey year, and place of birth. Race, age, and country of birth are included because the literature has found these variables to be strongly associated with dementia (Plassman *et al.*, 2007; Lawton *et al.*, 2015; Parlevliet *et al.*, 2016). Having a proxy respondent captures potential impairments related to dementia. Parental education controls for having lived in a cognitively stimulating environment during childhood. Survey year accounts for time contextual variables that could have affected the psychometric tests.

$Educ$ is a vector of five dummy variables that measure the level of education of individuals. These are included because, as shown in Figure 1, education is a good predictor of dementia.

O^*NET is a vector of other O*NET factors capturing different work activities and work contexts. Work activities are divided into two factors representing non-cognitive and physical work activities. Work contexts are divided into five factors, representing physical and social factors intrinsic to the nature of the work in different occupations (interpersonal relations, physical conditions, and others). These controls are included to explore variability in cognitive work activities within occupations with similar loads in non-cognitive and physical work activities and with similar work contexts. For example, jobs requiring high levels of cognitive work activities are less likely to require high levels of physical activities and more likely to be conducted indoors with low environmental hazard risk[12].

$OccInd$ is a vector of dummy variables with 11 main occupations and 13 primary industries where individuals worked during their most prolonged lifetime occupation and are included to capture variability in cognitive work activities within main occupations and industries. These partially control for unobserved determinants of occupational choice.

$WealthEmployment$ is a vector of contemporary wealth and employment variables including retirement status, household wealth, retirement status, the total number of years worked. These variables are endogenous because they could affect the outcome but also be affected by it. Moreover, they are partly determined by the nature of occupational choice and by cognitive decline. Therefore, they have to be interpreted with caution.

$Genetics$ is a vector of controls for the genetic predisposition of suffering dementia-related conditions, such as AD, cognition level, and coronary arterial problems; this last one having been related to vascular

[12] Six factors controlling for knowledge required in specific occupations are also generated. These capture organised principles and facts necessary for different occupations (administration, biology, construction, chemistry, clerical, and others). The controls of knowledge are highly correlated with cognitive work activities; they over-control the results. For example, the fifth work context factor has a correlation with cognitive work activities of 0.77, which is significant at a 99% confidence level. Therefore these factors are not included in the main specification.

dementia (Paciaroni and Bogousslavsky, 2013). Ten ancestry specific principal components are included to control for confounding from population stratification or to account for any ancestry differences in genetic structures within populations that could bias estimates. In addition, this specification controls for the genetic predisposition of suffering dementia and for genetic selection into occupations with more or less cognitive work activities. Including genetic controls is essential because if there is selection of individuals with a genetic predisposition to suffer dementia into occupations with high levels of cognitive work activities, the main specification would capture the effect of genetics and present it as the effect of cognitive work activities.

The term ϵ is an idiosyncratic error term.

The panel structure of the HRS is also used to explore if cognitive work activities lead to an earlier or later onset of dementia. Multivariate linear and non-linear regression models estimated by OLS and including individual-level fixed-effects are run to explore the decline towards dementia within individuals. These models control all time-invariant unobserved determinants of dementia such as genetics and baseline cognition (cognition, health, wealth). They explore differences in rates of decline towards dementia due to cognitive work activities. The main model is specified as follows.

$$\begin{aligned} Dem_{it} = \alpha_i + \beta_0 + \beta_1 Age_{it} + \beta_2 CognitiveWorkActivities'_i * Age_{it} + \beta_3 X'_{it} * Age_{it} \\ + \beta_4 Age^2_{it} + \beta_5 CognitiveWorkActivities'_i * Age^2_{it} + \beta_6 X'_{it} * Age^2_{it} + \epsilon_{it} \end{aligned} \tag{2}$$

The dependent variable Dem is equivalent to Model (1), with the only difference of including the subscript t to consider changes in dementia status within individuals over time. Age_{it} is a persons i age at time t, and for the ease of interpretation, this variable is demeaned. $CognitiveWorkActivities_i * Age_{it}$ is an interaction term of the time-fixed cognitive work activities and a persons age. It measures the effect of cognitive work activities on dementia over different age ranges. X' is a vector that includes race, the education level of individuals, the collar colour of their most prolonged lifetime occupation, and a measure of non-cognitive and physical work activities. These variables are also interacted with individuals' ages. It is included to capture the effect of these controls on dementia over different age ranges. The squared term of age accounts for non-linear effects of cognitive work activities for different ages, and it interacts with $CognitiveWorkActivities_i$ and X'. α_i is an individual-specific effect that controls for unobserved, time-invariant heterogeneity across individuals such as genetic, childhood environment, baseline cognition, and similar cofounders. The main effects of the time fixed variables are not included since they drop out with the individual-level fixed-effects. The coefficients of interest are β_2 and β_5. Their interpretation is difficult since

it requires a combination of age dependent interaction terms. For this reason, marginal effects at different combinations of age and cognitive work activities are estimated.

5.2 Biases

Some potential biases could have affected the identification strategy. In most cases, these biases can be solved, and if this is not possible, it is hypothesized how they could be affecting the results.

Individuals with lower levels of cognitive work activities could have a shorter life expectancy than individuals with higher levels of cognitive work activities. This shorter live expectancy for specific occupational groups would lead to an unbalanced sample at the oldest age groups. The sample is restricted to age 85 since it is reasonable to assume that the survival bias increases with age to minimize this bias. However, a part of this bias would remain, and it can be hypothesized which direction it would take. If individuals with low levels of cognitive work activities die at a younger age than individuals with high levels of cognitive work activities, the sample of older individuals, who are more likely to suffer dementia, would be composed of a higher proportion of individuals with higher levels of cognitive work activities. Therefore, the results would be capturing a lower bound of the effect of cognitive work activities on dementia.

There could also be self-selection of individuals into occupations with high levels of cognitive work activities due to some characteristics that could affect their risk of suffering dementia. However, even if it is impossible to rule this out completely, the primary analysis tries to limit this problem considering the variability in cognitive work activities within main occupations and industries, occupations with similar levels in non-cognitive and physical work activities and with similar work context, and individuals with similar genetic predisposition of suffering dementia.

Individuals who have dementia could also drop out of the sample due to their mental health condition. The HRS addresses this issue, including proxy respondents and performing interviews in aged care centres.

Individuals could also have held multiple occupations throughout their life cycle, while only their main lifetime occupation is considered. These multiple occupations could have involved a different range of work activities, while the estimations only consider the work activities of the main lifetime occupation. This problem cannot be solved due to data limitations since the HRS only collects information about an individuals' most prolonged lifetime occupation. This error is minimized by restricting the sample to males, for whom this problem should be smaller than females. This error is likely to be more significant for females of this generation since many worked in unpaid occupations, as housewives, and could report some short-term paid occupations. Therefore, restricting the sample to males should reduce the size of this error.

6 Results

6.1 Main Regression Results

This section explores the association between predicted dementia and cognitive work activities by estimating a series of regressions with increasingly larger covariate sets. Larger covariate sets will ideally control a more significant proportion of the non-random selection into different occupations and later life outcomes. Occupation selection is likely a function of individual characteristics such as genetic factors, parental socioeconomic status, and educational attainment. It is also essential to control for other job characteristics associated with cognitive work activities.

Table 2 includes the estimated regression coefficients on cognitive work activities. Column 1 presents the estimated association from a univariate regression, which equals -2.23. This coefficient implies that a one standard deviation increase in cognitive work activities decreases the probability of predicted dementia at time t by 2.23 percentage points. In Column 2, the regression includes survey year fixed-effects and control variables representing exogenous demographic characteristics (age, race, place of birth, and parental education). As a result, the estimated association reduces in magnitude by 33% to -1.49. Additionally, controlling for own educational attainment, which often precedes occupational choice, further reduces the association to -0.73 Column 3. The substantial reduction in magnitude indicates that educational attainment is strongly associated with cognitive work activities levels required at work and the likelihood of dementia in later life.

Table 2: Multivariate linear regressions estimated through pooled OLS – Predicted dementia as dependent variable and cognitive work activity as main interest independent variable

VARIABLES	(1)	(2)	(3)	(4)	(5)
Cognitive work activity	-2.23***	-1.49***	-0.73***	-0.69***	-0.75***
	(0.505)	(0.323)	(0.246)	(0.255)	(0.239)
Observations	43,018	43,018	43,018	43,018	43,018
Demographics	✗	✓	✓	✓	✓
Educational attainment	✗	✗	✓	✓	✓
O*NET factors and Occupation and industry	✗	✗	✗	✓	✓
Wealth and employment	✗	✗	✗	✗	✓

Note: The outcome in all regressions is predicted dementia, defined in Appendix C. Cognitive work activity level is standardised to have mean zero and standard deviation one. *Demographics*: Age, race, parents' education, place of birth, being a proxy respondent and survey year. *Educational achievement*: No high school, GED, high school, some college, and college and above. *O*NET job factor and occupation and industry covariates*: Non-cognitive and physical work activities and five 'work context' factors. 11 main occupations and 13 main industries. *Wealth and employment*: retirement status, last year worked, years employed, years employed in longest life-time occupation, and household wealth. Standard errors clustered at the occupation level shown in parentheses. *Source*: HRS and O*NET.

Also controlling for job, occupation and industry factors has little effect on the estimated association. In Column 4, the regression includes additional predicted job factors from the O*NET database and 11 occupation fixed-effects and 13 industry fixed-effects, which partially control for unobserved determinants

of occupational choice. In this regression, the estimated association between cognitive work activities and predicted dementia is identified by variation in job work content within the broad occupation and industry categories. For example, there are considerable differences between insurance salespersons (cognitive work activities equal to 0.77) and retail salespersons (cognitive work activities equal to -0.37). The estimated association in Column 4 drops slightly in magnitude to -0.69, suggesting that the difference in required cognitive work activities between insurance and retail sales occupations (1.14 standard deviations) reduces the likelihood of dementia by around 0.8 percentage points. A significant reduction considering that the sample mean equals 4.1%. The regression in Column 4, with demographic, education, occupation, and industry covariates, is the baseline specification.

In the last column of Table 2 (Column 5), endogenous controls are added (retirement status, last year worked, years employed in paid occupation, years employed in most prolonged lifetime occupation, household wealth). These characteristics will be partly determined by occupational choice and may also be determined by cognitive decline, and therefore should be interpreted with caution. Nevertheless, these regression results demonstrate that the significant association remains even when controlling for differences in the economic status between occupations. Moreover, the association becomes slightly more prominent.

This large negative association is robust to alternative modelling choices. First, in Table F.2 a logit or probit estimator is used. The estimated marginal effects for cognitive work activities are -0.68 and -0.67, compared with -0.69 in Column 4 of Table 2[13]. Second, a modelling assumption inherent in the Table 2 regressions is that the association between cognitive work activities and predicted dementia is (approximately) linear. This assumption is supported by the results shown in Appendix Table F.3, with added quadratic and squared root terms statistically insignificant. Third, the estimates are even larger if alternative approaches are used to predict dementia. Appendix Table F.1 provides estimates using the main approach, alongside the approaches from Hurd *et al.* (2013), the 'expert' model in Gianattasio *et al.* (2020), and the 'Lasso' model in Gianattasio *et al.* (2020)[14].

6.2 Genetics Confounders

The results presented in Table 2 demonstrate that the estimated association between cognitive work activities levels and predicted dementia is intense and cannot be 'washed away' with the inclusion of demographic, education and occupation factors. However, it is still possible that omitted covariates jointly influence job type and dementia risk. One candidate is genetic factors that are not adequately controlled with variables such as own and parental educational attainment. For example, genetic factors associated with high intelligence may increase cognitive work activities level (through occupation choice) and reduce the likelihood of

[13] The marginal effects have been estimated with the Stata command margins
[14] The sample the outcomes of Hurd *et al.* (2013) and Gianattasio *et al.* (2020) is smaller, since fewwer HRS sample waves are used.

dementia, even conditional on observed characteristics. The potential for genetic confounding is explored by using the HRS data on polygenic risk scores (PGSs) available for a reduced sample of 22,461 individuals with European ancestry.

To begin with, predicted dementia is regressed on three poly-genetic risk scores, representing individuals' genotype profiles for Alzheimer's disease (AD), general cognition, and coronary arterial disease (CAD). The CAD score is included because stroke and heart attack are associated with dementia (Paciaroni and Bogousslavsky, 2013). Column 1 in Table 3 demonstrates that the AD score is positively associated with predicted dementia, while the cognition and CAD scores are not. After adding the baseline set of covariates, corresponding to column (5) in Table 2 the estimated association with the AD score reduces in magnitude but is still significantly above zero (at the 10% level).

Table 3: Multivariate linear regressions estimated through OLS – Predicted dementia as dependent variable and work activity as main interest independent variable controlling for genetics

	(1)	(2)	(3)	(4)	(5)
VARIABLES	Dem	Dem	Cognitive work activity	Cognitive work activity	Dem
AD gen.	0.28**	0.19*	-0.02	0.00	0.19*
	(0.121)	(0.109)	(0.018)	(0.011)	(0.107)
Cog. gen.	-0.07	0.05	0.06**	0.00	0.06
	(0.168)	(0.155)	(0.023)	(0.010)	(0.156)
Cor. art. gen.	-0.06	-0.01	-0.03	-0.01	-0.01
	(0.143)	(0.132)	(0.017)	(0.010)	(0.131)
Cognitive work activity					-0.77***
					(0.275)
Observations	22,461	22,461	22,461	22,461	22,461
R-squared	0.00	0.07	0.02	0.58	0.07
10 PCA	✓	✓	✓	✓	✓
Baseline covariates	✗	✓	✗	✓	✓
Mean	4.13	4.13	-0.25	-0.25	4.13

Note: Standard errors clustered at occupational level in parentheses, *** p<0.01; ** p<0.05; * p<0.10. Sample restricted to males aged 60 to 85. *Predicted dementia:* Dementia is defined as scoring below a threshold in a psychometric test validated by Crimmins *et al.* (2011). More details of this variable can be found in appendix C. *Cognitive work activity:* Cognitive load of longest lifetime occupation, standardized with mean zero and standard deviation one. *Demographics:* Race, being a proxy respondent and parents education, age, survey year, place of birth. *Education:* Five education levels. *Occupation and industry:* 11 main occupations and 20 main industries. *Non-cognitive and physical work activities:* Two work activities factors. *Context:* Five Workplace context factors. *AD gen:* The poly-genetic risk score of suffering AD is a standardized variable with mean zero and standard deviation one (higher values increase risk). *Cog gen:* The poly-genetic risk score of cognition level is a standardized variable with mean zero and standard deviation one (higher values increase risk). *Cor. art. gen:* Poly-genetic risk score of suffering coronary arterial diseases (a risk factor for dementia) is a standardized variable with mean zero and standard deviation one (higher values increase risk). *10 PCA:* Ancestry specific principal components 1-10 are included for each group to control for confounding from population stratification, or to account for any ancestry differences in genetic structures within populations that could bias estimates (Price *et al.*, 2006). *Source*: HRS and O*NET.

Similarly, Columns 3 and 4 present estimated associations between the poly-genetic risk scores and cognitive work activities. Again, the AD score is not associated with cognitive work activities, with or without control variables added; however, a positive association with general cognition in Column 3 is reduced to near-zero once education, occupation, and industry covariates are added.

The results in Columns 1 to 4 in Table 3 indicate that genetic factors are unlikely to be confounding the estimated associations presented in Table 2. There is no apparent relationship between a person's genotype (AD, general cognition, CAD) and occupational choice and dementia, particularly when added to an

extensive set of covariates. The results in Column 5 confirm this. The poly-genetic risk scores are added to the baseline dementia regression, using the smaller sample for whom this information is available. The estimated coefficient on cognitive work activities is similar to the baseline estimate.

6.3 Placebo

The potential for omitted covariates that jointly influence job type and dementia risk is further explored by estimating regressions with indicators of non-neurological disease as the outcomes. If omitted variables, such as lifestyle factors and socioeconomic status (SES), are confounding the associations presented above, cognitive work activities might also be associated with health problems not generally linked with cognitive reserve and dementia. Precisely, the association between cognitive work activities and three common health problems, arthritis, cancer, and lung disease, is estimated while including all the control variables used in Column 4 of Table 2. These conditions have a strong socioeconomic gradient (i.e. more common among disadvantaged populations) and are impacted by lifestyle choices.

In Column 1 of Table 4 the baseline dementia regression from Table 2 is repeated, using the sub-sample with non-missing information on the incidence of arthritis, cancer, and lung disease. This sub-sample is slightly older (mean of 73 years versus 71 years) and has a lower percentage of Black and Hispanic people who worked in blue-collar occupations (mean of 44% individuals working in blue-collar occupations versus 38%) as can be seen in Table F.4. Consequently, the coefficient estimate on work cognitive activity is more prominent in magnitude (-0.80) than the total estimation sample (-0.69). Columns 2 to 5 show the estimated associations with the three 'placebo' health outcomes. The estimates for arthritis and cancer are near zero, indicating that cognitive work activities are not associated with these conditions. The estimate for lung disease is imprecise, and the point estimate, while non-trivial in magnitude, is the opposite sign to the dementia estimate: a one standard deviation increase in cognitive work activities is associated with a 0.78 percentage point increase in the likelihood of lung disease (8% increase relative to the sample mean). Overall, these findings suggest that the association between work cognitive work activities and predicted dementia is unlikely to be reflecting general SES or health lifestyle decisions.

Table 4: Multivariate linear regressions estimated through pooled OLS – Predicted dementia and other non-cognition related health outcomes as dependent variable and cognitive work activity as main interest independent variable

VARIABLES	(1) Dementia	(2) Arthritis	(3) Cancer	(4) Lung disease
Cognitive work activity	-0.80***	0.15	0.14	0.78
	(0.246)	(0.158)	(0.153)	(0.786)
Observations	40,688	40,688	40,688	40,688
R-squared	0.11	0.01	0.00	0.03
Baseline covariates	✓	✓	✓	✓
Mean	4.09	4.94	3.23	9.95

Note: Standard errors clustered at occupational level in parentheses, *** p<0.01; ** p<0.05; * p<0.10. Sample restricted to males aged 60 to 85. *Predicted dementia:* Dementia is defined as scoring below a threshold in a psychometric test validated by Crimmins *et al.* (2011). More details of this variable can be found in appendix C. *Arthritis:* Ever having been diagnosed from arthritis (self-reported). *Cancer:* Ever having been diagnosed from cancer (self-reported). *Lung disease:* Ever having been diagnosed from lung disease (self-reported). *Cognitive work activity:* Cognitive load of longest lifetime occupation, standardized with mean zero and standard deviation one. *Demographics:* Race, being a proxy respondent and parents education, age, survey year, place of birth. *Education:* Five education levels. *Occupation and industry:* 11 main occupations and 20 main industries. *Non-cognitive and physical work activities:* Two work activities factors. *Context:* Five Workplace context factors. *Source*: HRS and O*NET.

6.4 Further test for robustness

One of the recognised modifiable risk factors identified by the 2020 Lancet Commission on dementia prevention is physical inactivity. This is partly controlled for with physical activity at work through the work activity factors and the five physical and social work context factors. However, a further model is run including additional physical activity at work variables (the respondents reported performing general physical activities), and physical activity at home variables (walking frequently and undertaking sports and exercise). In addition, being cognitively active at work could be a complement or substitute for cognitive activity at home. Therefore, additionally this model controls for the frequency of cognitive activity at home, captured by educational activities, reading, writing, doing crosswords, playing cards or chess, or using the computer. When all these additional variables are included, the main work cognitive activity estimate increases to -1.11 (at a 99% confidence level) compared to the main estimate of -0.69. This implies that a one standard deviation increase in work cognitive activity reduces predicted dementia by 1.1 percentage points (or 27% relative to the mean). Most of this increase appears to be driven by further controlling for physical activities at work. In addition to physical inactivity, the Lancet Commission highlighted another 11 recognised risk factors for dementia: low education, hypertension, hearing impairment, smoking, obesity, depression, low social contact, excessive alcohol consumption, traumatic brain injury, and air pollution. The main regression specification already controls for education, but it is tested if the positive estimated association holds even when we control for these risk factors. Interestingly, it is found that these additional covariates leave the main estimate unchanged at -0.69.

6.5 Within-Individual Variation Over Time

Previous results demonstrate that the association between age and predicted dementia is smaller for occupations with high cognitive work activities. This analysis is extended by estimating individual fixed-effects regressions, which control for all time-invariant unobserved determinants of dementia. The main effect of cognitive work activities is not identified in within-individual regressions (because it is time-invariant), but it is possible to estimate how it influences the association between predicted dementia and age. Results from this exercise are shown in Table 5.

The within-individual estimated association between age and predicted dementia equals 0.48, indicating that a ten-year increase in age raises the probability of having dementia by 4.8 percentage points. In Column 2 of Table 5 the interaction between age and cognitive work activities is included. The coefficient suggests that the age gradient is 0.08 lower (7% relative to the age coefficient in Column 1) for people with occupations with a cognitive work activities level one standard deviation higher than the average.

Table 5: Multivariate linear and non-linear regressions estimated through OLS with individual fixed effects – Predicted dementia as dependent variable and interaction of cognitive work activity and age as main interest independent variable

VARIABLES	(1)	(2)	(3)
Age	0.48***	1.09***	-5.98***
	(0.043)	(0.104)	(1.311)
Cognitive work activity x Age		-0.08**	0.92
		(0.041)	(0.555)
Age^2			0.05***
			(0.009)
Cognitive work activity x Age^2			-0.01*
			(0.004)
Individual level fixed effects	✓	✓	✓
Covariates	✗	✓	✓
Observations	43,018	43,018	43,018

Note: Standard errors clustered at occupational level in parentheses, *** p<0.01; ** p<0.05; * p<0.10. Sample restricted to males aged 60 to 85. *Predicted dementia:* Dementia is defined as scoring below a threshold in a psychometric test validated by Crimmins *et al.* (2011). More details of this variable can be found in appendix C. Joint hypothesis $\beta_2 CognitiveWorkActivities * Age = CognitiveWorkActivities * Age^2 = 0$ is 0.034. *X':* Includes race, the education level of individuals, the collar colour of their longest lifetime occupation and non-cognitive and physical work activities are also interacted with individuals' ages. *Source*: HRS.

As Figure 1 makes clear, the association between age and dementia is highly nonlinear. Column 3 allows for this possibility by including age-squared terms. Thus, it is difficult to interpret the combination of age terms, but they allow for calculating estimated marginal effects at different combinations of age and cognitive work activities. Specifically, the estimated age marginal effects (gradient) at ages 70, 75 and 80, for someone with mean cognitive work activities levels, equal 1.02, 1.52 and 2.02, respectively. In comparison, the estimated marginal effects for someone with cognitive work activities levels two standard deviations higher than the mean equal 0.06, 0.36 and 0.66, respectively. Therefore, it is clear from the

individual fixed-effects regression estimates that the age-dementia association is substantially reduced for people who had occupations with high levels of cognitive work activities.

7 Discussion

This essay explored the effect of cognitive work activities performed during individuals' most prolonged lifetime occupation on their probability of suffering dementia at old age. The HRS has provided information about individuals' dementia status over time and their most prolonged lifetime occupations, while the O*NET has provided information about work activities in individuals' most prolonged lifetime occupations. The results point out that higher loads in cognitive work activities are strongly related to a lower probability of dementia at old age and a slower decline towards it.

A much richer set of work activities, work context and knowledge required for specific occupations are included compared to the previous literature (Andel *et al.*, 2005; Kröger *et al.*, 2008; Bosma *et al.*, 2003b; Hasselgren *et al.*, 2018). With this, variability in cognitive activities is considered within individuals in main occupations and industries, with similar non-cognitive and physical work activities, and a similar work context.

Using PGS information as a genetic predictor of dementia, it has been shown that there is genetic selection into occupations with different levels of cognitive work activities, but that the main specification controls for this. It has also been shown that there is no gene-environment interplay in the effect of cognitive work activities on dementia. Using the APOE $\epsilon 4$ protein as a genetic predictor of dementia, Hasselgren *et al.* (2018) suggest that work control is more protective for individuals with a higher genetic predisposition of suffering dementia compared to individuals with a lower genetic load. This essay finds that the effect of cognitive work activities does not differ regardless of the genetic load of suffering dementia. These differences could be driven by Hasselgren *et al.* (2018) using the APOE $\epsilon 4$ protein to predict dementia, while this essay using PGS.

This essay makes essential contributions to Fisher *et al.* (2014). It considers a much more extreme outcome, dementia, instead of general cognition. Furthermore, models with individual fixed-effects are run, which explore the decline towards dementia. These capture baseline cognition and intra-individual fixed characteristics such as genetics and childhood circumstances.

It is also explored if there are differences in the decline towards dementia within individuals due to the cognitive work activities hold in their longest lifetime occupations. Controlling for intra-individual fixed characteristics (genetics, childhood circumstances, baseline cognition) and estimating nonlinear models, it is shown that there are significant differences in the decline towards dementia within individuals, driven by

cognitive work activities. Furthermore, starting at age 70, rates of decline towards dementia differ significantly among individuals with different levels of cognitive work activities.

Two main conclusions can be drawn from this essay. First, in terms of basic research, it corroborates that dementia can be prevented through cognitive stimulating activities. It presents further evidence of the long-term effects of cognitive reserve on mental health. Prevention in the form of cognitive activities during the whole cycle-life of individuals has to be promoted to avoid the high rates of dementia predicted in the near future. Labour-life deserves much attention due to its enormous variability in cognitive activities and the wide of time individuals spend in it. It is well acknowledged that continuous physical activity during the life cycle prevents many hazardous physical health outcomes at old age. Similarly, cognitive activities train cognitive reserve and prevent hazardous mental health outcomes, such as dementia.

Second, in terms of applied research and policymaking, this essay points out a labour health hazard that has received little attention so far. The results show that cognitive work activities prevent the onset and decline towards dementia. However, it is well understood that certain physical work activities can cause detrimental physical outcomes. To address these problems, the US's Occupational Safety and Health Administration has established rigid legislation on operating with occupations that involve work activities considered a health hazard. The results of this essay suggest that this legislation should be expanded to consider occupations with low levels of cognitive work activities. Similarly to physical strenuous work activities having detrimental effects on individuals physical health, activities that do not involve many cognitive work activities can cause chronic mental health problems, such as dementia. Therefore, it is necessary to protect workers in these risky positions. This protection could greatly affect individuals' mental health and public health systems, smoothing the transition towards an ageing population.

References

ABRAHAM, K. G. and SPLETZER, J. R. (2009). New evidence on the returns to job skills. *American Economic Review*, **99** (2), 52–57.

ALMEIDA, R. P., SCHULTZ, S. A., AUSTIN, B. P., BOOTS, E. A., DOWLING, N. M., GLEASON, C. E., BENDLIN, B. B., SAGER, M. A., HERMANN, B. P., ZETTERBERG, H. *et al.* (2015). Effect of cognitive reserve on age-related changes in cerebrospinal fluid biomarkers of Alzheimer disease. *JAMA Neurology*, **72** (6), 699–706.

ALZHEIMER'S ASSOCIATION (2020). What is Alzheimer's? https://www.alz.org/alzheimers-dementia/what-is-alzheimers, accessed: 2020-03-09.

ANDEL, R., CROWE, M., HAHN, E. A., MORTIMER, J. A., PEDERSEN, N. L., FRATIGLIONI, L., JOHANSSON, B. and GATZ, M. (2012). Work-related stress may increase the risk of vascular dementia. *Journal of the American Geriatrics Society*, **60** (1), 60–67.

—, —, KÅREHOLT, I., WASTESSON, J. and PARKER, M. G. (2011). Indicators of job strain at midlife and cognitive functioning in advanced old age. *Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, **66** (3), 287–291.

—, —, PEDERSEN, N. L., MORTIMER, J., CRIMMINS, E., JOHANSSON, B. and GATZ, M. (2005). Complexity of work and risk of Alzheimer's disease: a population-based study of Swedish twins. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, **60** (5), P251–P258.

—, KÅREHOLT, I., PARKER, M. G., THORSLUND, M. and GATZ, M. (2007). Complexity of primary lifetime occupation and cognition in advanced old age. *Journal of Aging and Health*, **19** (3), 397–415.

ANDERSEN, K., LAUNER, L. J., DEWEY, M. E., LETENNEUR, L., OTT, A., COPELAND, J., DARTIGUES, J.-F., KRAGH-SORENSEN, P., BALDERESCHI, M., BRAYNE, C. *et al.* (1999). Gender differences in the incidence of AD and vascular dementia The EURODEM Studies. *Neurology*, **53** (9), 1992–1992.

BASU, R. (2013). Education and dementia risk: results from the Aging Demographics and Memory Study. *Research on Aging*, **35** (1), 7–31.

BEARD, C. M., KOKMEN, E., OFFORD, K. P. and KURLAND, L. T. (1992). Lack of association between alzheimer's disease and education, occupation, marital status, or living arrangement. *Neurology*, **42** (11), 2063–2063.

BERR, C., WANCATA, J. and RITCHIE, K. (2005). Prevalence of dementia in the elderly in Europe. *European Neuropsychopharmacology*, **15** (4), 463–471.

BOOTS, E. A., SCHULTZ, S. A., ALMEIDA, R. P., OH, J. M., KOSCIK, R. L., DOWLING, M. N., GALLAGHER, C. L., CARLSSON, C. M., ROWLEY, H. A., BENDLIN, B. B. *et al.* (2015). Occupational complexity and cognitive reserve in a middle-aged cohort at risk for Alzheimer's disease. *Archives of Clinical Neuropsychology*, **30** (7), 634–642.

BOSMA, H., VAN BOXTEL, M., PONDS, R., HOUX, P. and JOLLES, J. (2003a). Education and age-related cognitive decline: the contribution of mental workload. *Educational Gerontology*, **29** (2), 165–173.

—, VAN BOXTEL, M. P., PONDS, R. W., HOUX, P. J., BURDORF, A. and JOLLES, J. (2003b). Mental work demands protect against cognitive impairment: MAAS prospective cohort study. *Experimental Aging Research*, **29** (1), 33–45.

BRANDT, J., SPENCER, M. and FOLSTEIN, M. (1988). The telephone interview for cognitive status. *Neuropsychology, Neuropsychiatry, and Behavioral Neurology*, **1** (2), 111–117.

BUGLIARI, D., CAMPBELL, N., CHAN, C., HAYDEN, O., HAYES, J., HURD, M., MAIN, R., MALLETT, J., MCCULLOUGH, C., MEIJER, E. *et al.* (2018). RAND HRS Longitudinal File 2014 (V2) Documentation.

BUTLER, M., MCCREEDY, E., NELSON, V. A., DESAI, P., RATNER, E., FINK, H. A., HEMMY, L. S., MCCARTEN, J. R., BARCLAY, T. R., BRASURE, M. *et al.* (2018). Does cognitive training prevent cognitive decline? A systematic review. *Annals of Internal Medicine*, **168** (1), 63–68.

CENTER OF DISEASE (2020). What is Alzheimer's disease? https://www.cdc.gov/aging/aginginfo/alzheimers.html, accessed: 2020-03-09.

CHANDRA, A., COILE, C. and MOMMAERTS, C. (2020). What can economics say about alzheimer's disease? *NBER*, **Working Paper 27760**.

CHENG, S.-T. (2016). Cognitive reserve and the prevention of dementia: the role of physical and cognitive activities. *Current Psychiatry Reports*, **18** (9), 85.

CLOUSTON, S. A., GLYMOUR, M. M. and TERRERA, G. M. (2015). Educational inequalities in aging-related declines in fluid cognition and the onset of cognitive pathology. *Alzheimer's & Dementia: Diagnosis, Assessment & Disease Monitoring*, **1** (3), 303–310.

COBB, J., WOLF, P. A., AU, R., WHITE, R. and D'AGOSTINO, R. (1995). The effect of education on the incidence of dementia and Alzheimer's disease in the Framingham Study. *Neurology*, **45** (9), 1707–1712.

COTTINI, E. and LUCIFORA, C. (2013). Mental health and working conditions in Europe. *ILR Review*, **66** (4), 958–988.

CRIMMINS, E. M., KIM, J. K., LANGA, K. M. and WEIR, D. R. (2011). Assessment of cognition using surveys and neuropsychological assessment: the Health and Retirement Study and the Aging, Demographics, and Memory Study. *Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, **66** (suppl_1), i162–i171.

—, SAITO, Y., KIM, J. K., ZHANG, Y. S., SASSON, I. and HAYWARD, M. D. (2018). Educational differences in the prevalence of dementia and life expectancy with dementia: Changes from 2000 to 2010. *The Journals of Gerontology: Series B*, **73** (suppl_1), S20–S28.

CROWE, M., ANDEL, R., PEDERSEN, N. L. and GATZ, M. (2007). Do work-related stress and reactivity to stress predict dementia more than 30 years later? *Alzheimer Disease & Associated Disorders*, **21** (3), 205–209.

DANG, A., MAITRA, P. and MENON, N. (2019). Labor market engagement and the body mass index of working adults: evidence from India. *Economics & Human Biology*, **33**, 58–77.

DARTIGUES, J.-F., GAGNON, M., LETENNEUR, L., BARBERGER-GATEAU, P., COMMENGES, D., EVALDRE, M. and SALAMON, R. (1992). Principal lifetime occupation and cognitive impairment in a french elderly cohort (Paquid). *American Journal of Epidemiology*, **135** (9), 981–988.

DAVID, H. and DORN, D. (2013). The growth of low-skill service jobs and the polarization of the US labor market. *American Economic Review*, **103** (5), 1553–97.

ELOVAINIO, M., FERRIE, J. E., SINGH-MANOUX, A., GIMENO, D., DE VOGLI, R., SHIPLEY, M. J., VAHTERA, J., BRUNNER, E. J., MARMOT, M. G. and KIVIMÄKI, M. (2009). Cumulative exposure to high-strain and active jobs as predictors of cognitive function: the Whitehall II study. *Occupational and Environmental Medicine*, **66** (1), 32–37.

EVANS, D. A., HEBERT, L. E., BECKETT, L. A., SCHERR, P. A., ALBERT, M. S., CHOWN, M. J., PILGRIM, D. M. and TAYLOR, J. O. (1997). Education and other measures of socioeconomic status and risk of incident Alzheimer disease in a defined population of older persons. *Archives of Neurology*, **54** (11), 1399–1405.

FINKEL, D., ANDEL, R., GATZ, M. and PEDERSEN, N. L. (2009). The role of occupational complexity in trajectories of cognitive aging before and after retirement. *Psychology and Aging*, **24** (3), 563.

FISHER, G. G., STACHOWSKI, A., INFURNA, F. J., FAUL, J. D., GROSCH, J. and TETRICK, L. E. (2014). Mental work demands, retirement, and longitudinal trajectories of cognitive functioning. *Journal of Occupational Health Psychology*, **19** (2), 231.

FLETCHER, J. M., SINDELAR, J. L. and YAMAGUCHI, S. (2011). Cumulative effects of job characteristics on health. *Health Economics*, **20** (5), 553–570.

GARCIA, M. A., DOWNER, B., CHIU, C.-T., SAENZ, J. L., ROTE, S. and WONG, R. (2017). Racial/ethnic and nativity differences in cognitive life expectancies among older adults in the United States. *The Gerontologist*.

GATZ, M., SVEDBERG, P., PEDERSEN, N. L., MORTIMER, J. A., BERG, S. and JOHANSSON, B. (2001). Education and the risk of Alzheimer's disease: findings from the study of dementia in Swedish twins. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, **56** (5), P292–P300.

GIANATTASIO, K. Z., CIARLEGLIO, A. and POWER, M. C. (2020). Development of algorithmic dementia ascertainment for racial/ethnic disparities research in the US Health and Retirement Study. *Epidemiology*, **31** (1), 126–133.

GURLAND, B. J., WILDER, D. E., LANTIGUA, R., STERN, Y., CHEN, J., KILLEFFER, E. H. and MAYEUX, R. (1999). Rates of dementia in three ethnoracial groups. *International Journal of Geriatric Psychiatry*, **14** (6), 481–493.

HASSELGREN, C., DELLVE, L., EKBRAND, H., ZETTERGREN, A., ZETTERBERG, H., BLENNOW, K., SKOOG, I. and HALLERÖD, B. (2018). Socioeconomic status, gender and dementia: the influence of work environment exposures and their interactions with APOE ϵ4. *Social Science and Medicine-Population Health*, **5**, 171–179.

HEINESEN, E., IMAI, S. and MARUYAMA, S. (2018). Employment, job skills and occupational mobility of cancer survivors. *Journal of Health Economics*, **58**, 151–175.

HSU, J. W. and WILLIS, R. (2013). Dementia risk and financial decision making by older households: The impact of information. *Journal of Human Capital*, **7** (4), 340–377.

HURD, M. D., MARTORELL, P., DELAVANDE, A., MULLEN, K. J. and LANGA, K. M. (2013). Monetary costs of dementia in the United States. *New England Journal of Medicine*, **368** (14), 1326–1334.

ISHIZAKI, M., MORIKAWA, Y., NAKAGAWA, H., HONDA, R., KAWAKAMI, N., HARATANI, T., KOBAYASHI, F., ARAKI, S. and YAMADA, Y. (2004). The influence of work characteristics on body mass index and waist to hip ratio in Japanese employees. *Industrial Health*, **42** (1), 41–49.

JONAITIS, E., LA RUE, A., MUELLER, K. D., KOSCIK, R. L., HERMANN, B. and SAGER, M. A. (2013). Cognitive activities and cognitive performance in middle-aged adults at risk for Alzheimer's disease. *Psychology and Aging*, **28** (4), 1004.

JORM, A. F., RODGERS, B., HENDERSON, A. S., KORTEN, A. E., JACOMB, P. A., CHRISTENSEN, H. and MACKINNON, A. (1998). Occupation type as a predictor of cognitive decline and dementia in old age. *Age and Ageing*, **27** (4), 477–483.

KARP, A., ANDEL, R., PARKER, M. G., WANG, H.-X., WINBLAD, B. and FRATIGLIONI, L. (2009). Mentally stimulating activities at work during midlife and dementia risk after age 75: follow-up study from the Kungsholmen Project. *The American Journal of Geriatric Psychiatry*, **17** (3), 227–236.

—, KÅREHOLT, I., QIU, C., BELLANDER, T., WINBLAD, B. and FRATIGLIONI, L. (2004). Relation of education and occupation-based socioeconomic status to incident Alzheimer's disease. *American Journal of Epidemiology*, **159** (2), 175–183.

KEMPPAINEN, N. M., AALTO, S., KARRASCH, M., NÅGREN, K., SAVISTO, N., OIKONEN, V., VIITANEN, M., PARKKOLA, R. and RINNE, J. O. (2008). Cognitive reserve hypothesis: Pittsburgh Compound B and fluorodeoxyglucose positron emission tomography in relation to education in mild Alzheimer's disease. *Annals of Neurology: Official Journal of the American Neurological Association and the Child Neurology Society*, **63** (1), 112–118.

KIVIPELTO, M., MANGIALASCHE, F. and NGANDU, T. (2018). Lifestyle interventions to prevent cognitive impairment, dementia and Alzheimer disease. *Nature Reviews Neurology*, **14** (11), 653–666.

KRÖGER, E., ANDEL, R., LINDSAY, J., BENOUNISSA, Z., VERREAULT, R. and LAURIN, D. (2008). Is complexity of work associated with risk of dementia? The Canadian Study of Health and Aging. *American Journal of Epidemiology*, **167** (7), 820–830.

LAKDAWALLA, D. and PHILIPSON, T. (2007). Labor supply and weight. *Journal of Human Resources*, **42** (1), 85–116.

LAWTON, D. M., GASQUOINE, P. G. and WEIMER, A. A. (2015). Age of dementia diagnosis in community dwelling bilingual and monolingual Hispanic Americans. *Cortex*, **66**, 141–145.

LIÈVRE, A., ALLEY, D. and CRIMMINS, E. M. (2008). Educational differentials in life expectancy with cognitive impairment among the elderly in the United States. *Journal of Aging and Health*, **20** (4), 456–477.

LIVINGSTON, G., HUNTLEY, J., SOMMERLAD, A., AMES, D., BALLARD, C., BANERJEE, S. and COSTAFREDA, S. (2020). Dementia prevention, intervention, and care: 2020 report of the Lancet Commission. *Lancet*, **396**, 413–446.

MARQUIÉ, J.-C., DUARTE, L. R., BESSIÈRES, P., DALM, C., GENTIL, C. and RUIDAVETS, J. (2010). Higher mental stimulation at work is associated with improved cognitive functioning in both young and older workers. *Ergonomics*, **53** (11), 1287–1301.

MCDOWELL, I., XI, G., LINDSAY, J. and TIERNEY, M. (2007). Mapping the connections between education and dementia. *Journal of Clinical and Experimental Neuropsychology*, **29** (2), 127–141.

NGANDU, T., VON STRAUSS, E., HELKALA, E.-L., WINBLAD, B., NISSINEN, A., TUOMILEHTO, J., SOININEN, H. and KIVIPELTO, M. (2007). Education and dementia: what lies behind the association? *Neurology*, **69** (14), 1442–1450.

ORRELL, M. and SAHAKIAN, B. (1995). Education and dementia. *British Journal of Medicine*, **310**, 951–952.

ORTMAN, J. M., VELKOFF, V. A., HOGAN, H. *et al.* (2014). *An aging nation: the older population in the United States*. United States Census Bureau, Economics and Statistics Administration, US Department of Commerce.

PACIARONI, M. and BOGOUSSLAVSKY, J. (2013). Connecting cardiovascular disease and dementia: further evidence.

PARLEVLIET, J. L., UYSAL-BOZKIR, Ö., GOUDSMIT, M., VAN CAMPEN, J. P., KOK, R. M., TER RIET, G., SCHMAND, B. and DE ROOIJ, S. E. (2016). Prevalence of mild cognitive impairment and dementia in older non-western immigrants in the Netherlands: a cross-sectional study. *International Journal of Geriatric Psychiatry*, **31** (9), 1040–1049.

PERNECZKY, R., DRZEZGA, A., DIEHL-SCHMID, J., SCHMID, G., WOHLSCHLÄGER, A., KARS, S., GRIMMER, T., WAGENPFEIL, S., MONSCH, A. and KURZ, A. (2006). Schooling mediates brain reserve in Alzheimer's disease: findings of fluoro-deoxy-glucose-positron emission tomography. *Journal of Neurology, Neurosurgery & Psychiatry*, **77** (9), 1060–1063.

PLASSMAN, B. L., LANGA, K. M., FISHER, G. G., HEERINGA, S. G., WEIR, D. R., OFSTEDAL, M. B., BURKE, J. R., HURD, M. D., POTTER, G. G., RODGERS, W. L. *et al.* (2007). Prevalence of dementia in the United States: the aging, demographics, and memory study. *Neuroepidemiology*, **29** (1-2), 125–132.

POTTER, G., PLASSMAN, B., HELMS, M., FOSTER, S. and EDWARDS, N. (2006). Occupational characteristics and cognitive performance among elderly male twins. *Neurology*, **67** (8), 1377–1382.

POTTER, G. G., HELMS, M. J. and PLASSMAN, B. L. (2008). Associations of job demands and intelligence with cognitive performance among men in late life. *Neurology*, **70** (19 Part 2), 1803–1808.

PRICE, A. L., PATTERSON, N. J., PLENGE, R. M., WEINBLATT, M. E., SHADICK, N. A. and REICH, D. (2006). Principal components analysis corrects for stratification in genome-wide association studies. *Nature Genetics*, **38** (8), 904–909.

QIU, C., KARP, A., VON STRAUSS, E., WINBLAD, B., FRATIGLIONI, L. and BELLANDER, T. (2003). Lifetime principal occupation and risk of Alzheimer's disease in the Kungsholmen project. *American Journal of Industrial Medicine*, **43** (2), 204–211.

RAVESTEIJN, B., KIPPERSLUIS, H. V. and DOORSLAER, E. V. (2018). The wear and tear on health: What is the role of occupation? *Health Economics*, **27** (2), e69–e86.

ROVIO, S., KÅREHOLT, I., VIITANEN, M., WINBLAD, B., TUOMILEHTO, J., SOININEN, H., NISSINEN, A. and KIVIPELTO, M. (2007). Work-related physical activity and the risk of dementia and Alzheimer's disease. *International Journal of Geriatric Psychiatry: A journal of the psychiatry of late life and allied sciences*, **22** (9), 874–882.

RUITENBERG, A., OTT, A., VAN SWIETEN, J. C., HOFMAN, A. and BRETELER, M. M. (2001). Incidence of dementia: does gender make a difference? *Neurobiology of Aging*, **22** (4), 575–580.

SATTLER, C., TORO, P., SCHÖNKNECHT, P. and SCHRÖDER, J. (2012). Cognitive activity, education and socioeconomic status as preventive factors for mild cognitive impairment and Alzheimer's disease. *Psychiatry Research*, **196** (1), 90–95.

SCHMITZ, L. L. (2016). Do working conditions at older ages shape the health gradient? *Journal of Health Economics*, **50**, 183–197.

SEIDLER, A., NIENHAUS, A., BERNHARDT, T., KAUPPINEN, T., ELO, A. and FRÖLICH, L. (2004). Psychosocial work factors and dementia. *Occupational and Environmental Medicine*, **61** (12), 962–971.

SHADLEN, M.-F., SISCOVICK, D., FITZPATRICK, A. L., DULBERG, C., KULLER, L. H. and JACKSON, S. (2006). Education, cognitive test scores, and black-white differences in dementia risk. *Journal of the American Geriatrics Society*, **54** (6), 898–905.

SINDI, S., HAGMAN, G., HÅKANSSON, K., KULMALA, J., NILSEN, C., KÅREHOLT, I., SOININEN, H., SOLOMON, A. and KIVIPELTO, M. (2017). Midlife work-related stress increases dementia risk in later life: the CAIDE 30-year study. *Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, **72** (6), 1044–1053.

SMART, E. L., GOW, A. J. and DEARY, I. J. (2014). Occupational complexity and lifetime cognitive abilities. *Neurology*, **83** (24), 2285–2291.

SMYTH, K., FRITSCH, T., COOK, T., MCCLENDON, M., SANTILLAN, C. and FRIEDLAND, R. (2004). Worker functions and traits associated with occupations and the development of AD. *Neurology*, **63** (3), 498–503.

STERN, Y. (2002). What is cognitive reserve? Theory and research application of the reserve concept. *Journal of the International Neuropsychological Society*, **8** (3), 448–460.

— (2012). Cognitive reserve in ageing and Alzheimer's disease. *The Lancet Neurology*, **11** (11), 1006–1012.

—, ALEXANDER, G. E., PROHOVNIK, I. and MAYEUX, R. (1992). Inverse relationship between education and parietotemporal perfusion deficit in Alzheimer's disease. *Annals of Neurology: Official Journal of the American Neurological Association and the Child Neurology Society*, **32** (3), 371–375.

—, —, —, STRICKS, L., LINK, B., LENNON, M. C. and MAYEUX, R. (1995). Relationship between lifetime occupation and parietal flow implications for a reserve against Alzheimer's disease pathology. *Neurology*, **45** (1), 55–60.

—, GURLAND, B., TATEMICHI, T. K., TANG, M. X., WILDER, D. and MAYEUX, R. (1994). Influence of education and occupation on the incidence of Alzheimer's disease. *JAMA*, **271** (13), 1004–1010.

SUTIN, A. R., STEPHAN, Y., LUCHETTI, M. and TERRACCIANO, A. (2018a). Self-reported personality traits are prospectively associated with proxy-reported behavioral and psychological symptoms of dementia at the end of life. *International Journal of Geriatric Psychiatry*, **33** (3), 489–494.

—, — and TERRACCIANO, A. (2018b). Facets of conscientiousness and risk of dementia. *Psychological Medicine*, **48** (6), 974–982.

THEN, F. S., LUCK, T., HESER, K., ERNST, A., POSSELT, T., WIESE, B., MAMONE, S., BRETTSCHNEIDER, C., KÖNIG, H.-H., WEYERER, S. *et al.* (2017). Which types of mental work demands may be associated with reduced risk of dementia? *Alzheimer's & Dementia*, **13** (4), 431–440.

UNITED NATIONS, DEPARTMENT OF ECONOMIC AND SOCIAL AFFAIRS, POPULATION DIVISION (2017). World population ageing 2017 - highlights.

VALENZUELA, M. J. and SACHDEV, P. (2006). Brain reserve and dementia: a systematic review. *Psychological Medicine*, **36** (4), 441–454.

WANG, H.-X., MACDONALD, S. W., DEKHTYAR, S. and FRATIGLIONI, L. (2017). Association of life-long exposure to cognitive reserve-enhancing factors with dementia risk: a community-based cohort study. *PLoS medicine*, **14** (3), e1002251.

—, WAHLBERG, M., KARP, A., WINBLAD, B. and FRATIGLIONI, L. (2012). Psychosocial stress at work is associated with increased dementia risk in late life. *Alzheimer's & Dementia*, **8** (2), 114–120.

WELSH, K. A., BREITNER, J. C. and MAGRUDER-HABIB, K. M. (1993). Detection of dementia in the elderly using telephone screening of cognitive status. *Neuropsychiatry, Neuropsychology, & Behavioral Neurology*.

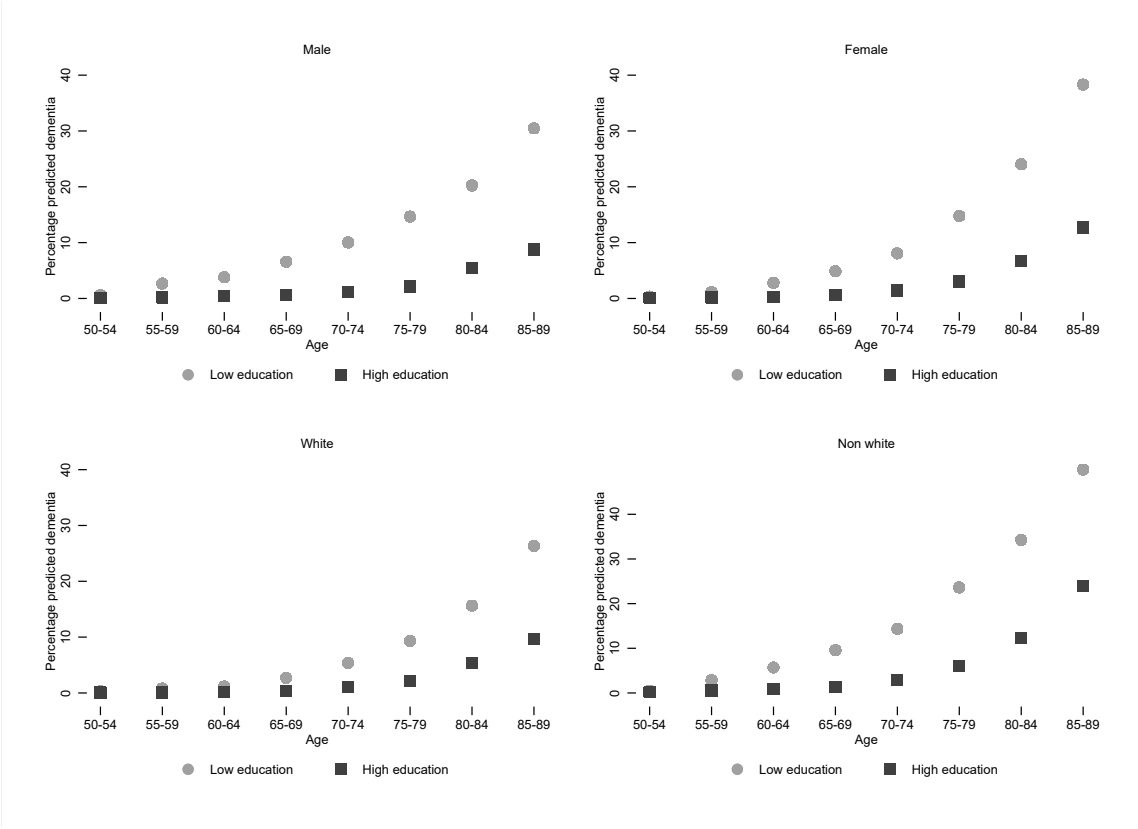
WOLTERS, F. J., CHIBNIK, L. B., WAZIRY, R., ANDERSON, R., BERR, C., BEISER, A., BIS, J. C., BLACKER, D., BOS, D., BRAYNE, C. *et al.* (2020). 27-year time trends in dementia incidence in Europe and the US: the Alzheimer Cohorts Consortium. *Neurology*.

WORLD HEALTH ORGANIZATION (2020). Dementia. https://www.who.int/news-room/fact-sheets/detail/dementia, accessed: 2020-03-09.

Appendices

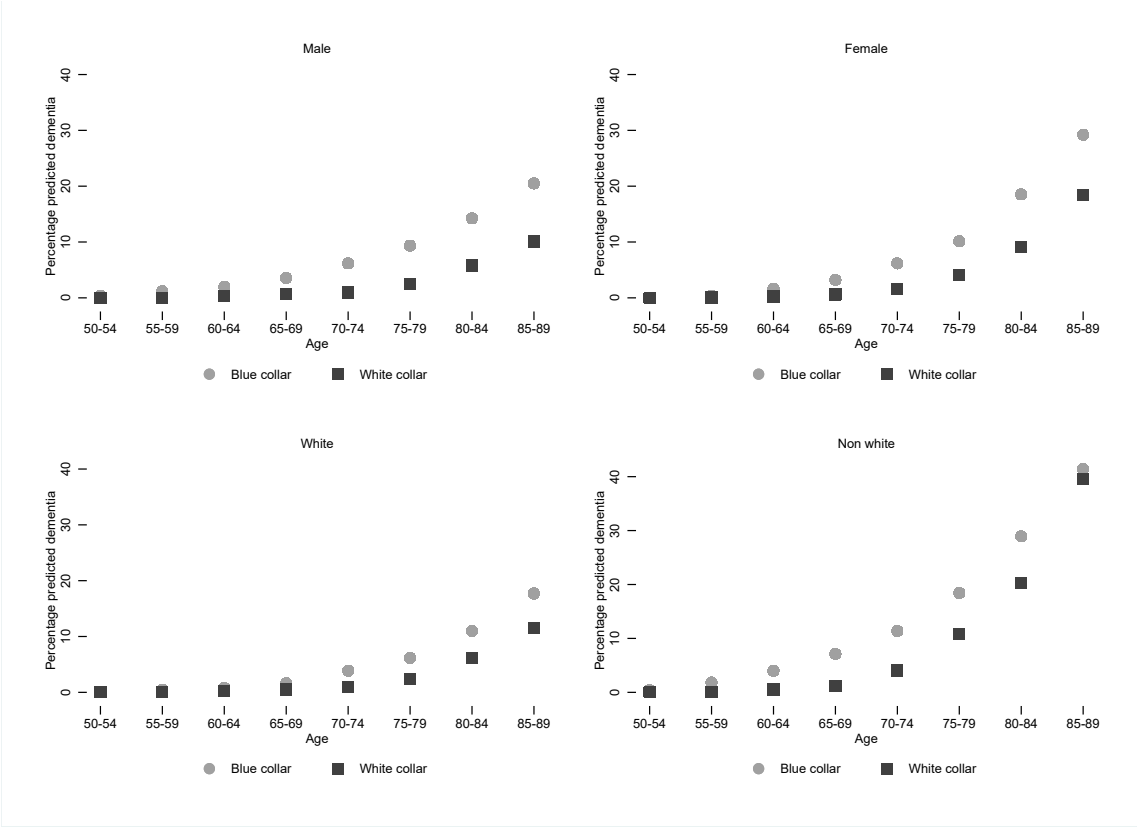
A Extended descriptives

Figure A.1: Proportion of individuals predicted to suffer dementia for education levels



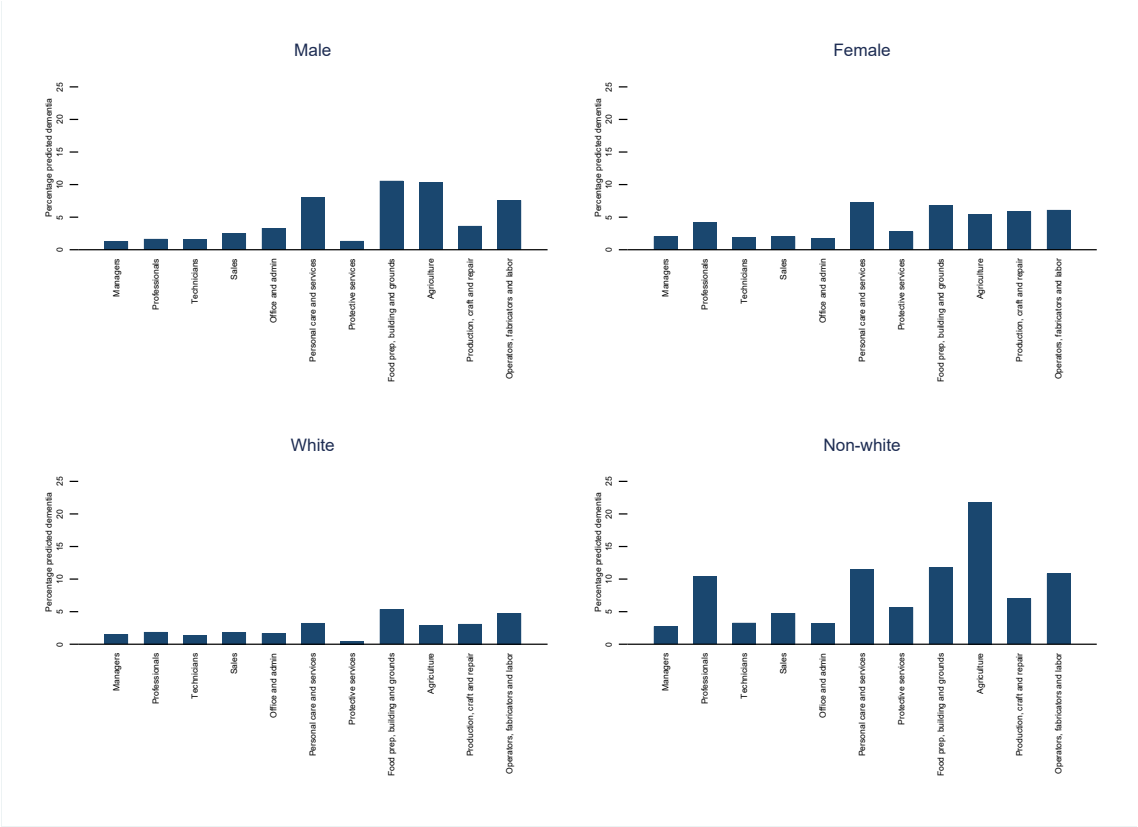
Note: Sample restricted to males aged 60 to 85. *Predicted dementia:* Dementia is defined as scoring below a threshold in a psychometric test validated by Crimmins *et al.* (2011). More details of this variable can be found in appendix C. *Education:* Low education considers no high school and GED. High education considers high school and college or above. *Source*: HRS.

Figure A.2: Proportion of individuals predicted to suffer dementia for education levels



Note: Sample restricted to males aged 60 to 85. *Predicted dementia:* Dementia is defined as scoring below a threshold in a psychometric test validated by Crimmins *et al.* (2011). More details of this variable can be found in appendix C. *Collar colour:* Blue-collar occupations involve skilled or unskilled manual labour occupations. White-collar occupations involve professional, managerial, or administrative occupations. *Source*: HRS.

Figure A.3: Proportion of individuals predicted to suffer dementia for main occupations



Note: Sample restricted to males aged 60 to 85. *Predicted dementia:* Dementia is defined as scoring below a threshold in a psychometric test validated by Crimmins *et al.* (2011). More details of this variable can be found in appendix C. *Source*: HRS.

B Sample Construction

The main sample consists of male aged 60 to 85 with non-missing information about their longest lifetime occupation and the score in the cognitive test[15]. Some robustness also include information for the main respondents' spouses. The RAND HRS Longitudinal File 2014 is a sample of 37,495 individuals who participated in different survey years of the HRS (from one to 12 survey years). These individuals can be part of the original HRS sample or any of its refresher cohorts. Each of them can be observed a different number of times (from one to 12), depending on the cohort when they first participated (more recent cohorts can only be observed a lower number of times) and attrition. This database is transformed into a long format and the number of observations increases to 203,219 observations, which consider the multiple answers of the same individual through the different survey years. From this point on, the sample gets restricted.

Individuals with non-missing information regarding their longest lifetime occupation and cognition are considered, which reduces the sample to 125,819 observations. The reasons for the missing observations

[15] Table B.1 presents summary statistics for the full sample and for the sample with no missing observations in cognition and longest lifetime occupation. Both samples are very balanced in terms of race and education level. Still, the sample with no missing observations is three years younger than the sample used for the main estimations.

at the longest lifetime occupation are: (1) not having worked at all or not having reported longest lifetime occupation; (2) individuals reporting longest lifetime occupations with census codes (SOC1980) 900 and 990. These codes do not get transformed to occ1990dd; (3) individuals reporting longest lifetime occupations with census codes (SOC2000) 123, 650, 691, 692, 693, 884, 950, 980 and 981. These codes do not get transformed to occ1990dd. Although for (2) and (3) there is available information about the longest lifetime occupation of the individuals, they are excluded when considering homogeneous labour codes for all survey years with the occ1990dd codification. Nevertheless, these are the smallest part of these lost observations.

The sample is further restricted to male aged 60 to 85, leading to 43,018 observations and 7,928 individuals.

Table B.1: Descriptive variables of the full sample and the sample of non-missing observations in cognition and longest lifetime occupations

	(1)		(2)	
	Full sample		Sample – non-missing cognition or occupation	
	mean	sd	mean	sd
White	0.79	0.41	0.80	0.38
Black	0.12	0.32	0.14	0.35
Hispanic	0.08	0.26	0.05	0.22
No high-school	0.24	0.43	0.25	0.43
GED	0.05	0.22	0.05	0.22
High-school	0.28	0.45	0.27	0.44
Some college	0.18	0.39	0.20	0.40
College and above	0.24	0.43	0.23	0.42
Age	70.79	6.61	67.06	10.44

Note: Sample restricted to males aged 60 to 85. Non-cognition related health outcomes are arthritis, cancer, and lung disease. *Source*: HRS and O*NET.

C Recoveries from dementia

Individuals run the cognitive test each survey year making it possible for them to 'recover' from their dementia status. In other words, they can score at one survey year below the defined threshold that categorize them as having dementia and at the next survey year above. This happens quite a lot. Figure C.1 presents the number of individuals that according to the instrument used, never suffer from dementia, suffer from dementia and eventually recover at some further survey year and suffer from dementia and do not recover at any further survey year. 3.085 individuals are diagnosed with dementia at some survey year and of these, 1,235 individuals do not present any recovery after first having being been predicted to suffer dementia. That is, after having scored under the threshold that defines them as having dementia, they consistently score below the threshold at the subsequent survey years. On the contrary, 1,850 individuals experience some recovery after first having been diagnosed with dementia. After having scored under the threshold that predicts dementia, they score above it at some subsequent wave.

This raises the question if these individuals truly suffer from dementia, as part of them could have a cognitive status above the one that predicts them to have dementia, but after several survey, years running

the psychometric test, score below the threshold. This could be caused by factors impossible to control for, such as fatigue, emotional state or simply chance. Therefore, it is possible for individuals experiencing recoveries after first having been diagnosed with dementia to have a different cognitive status to the ones never recovering. This is confirmed in Figure C.2, which presents the average cognitive score of individuals before and after first being diagnosed with dementia. Individuals recovering, tend to score consistently above the threshold after first having been diagnosed with dementia, which suggests that individuals recovering from dementia could have normal cognition and an only score below the score because of the previously suggested error. For this reason, it seems reasonable to examine more in detail the definition of dementia and to generate a cleaner predictor.

It is examined if the problem lies in the different definitions for having dementia. As explained in the methodology section, individuals can be diagnosed with dementia according to their scores in the cognitive test or a composed score formed by indicators taken from the proxy respondents and the interviewers. The proportion of individuals diagnosed with dementia with recoveries and without could differ due to the fact of being a normal or a proxy respondent. Figure C.1 is replicated only for proxy respondents and Figure C.3 shows that of the 1.850 individuals having dementia with no recovery, 410 (33%) are proxy respondents and of the 1,235 individuals having dementia with recovery, 990 (53%) are proxy respondents.

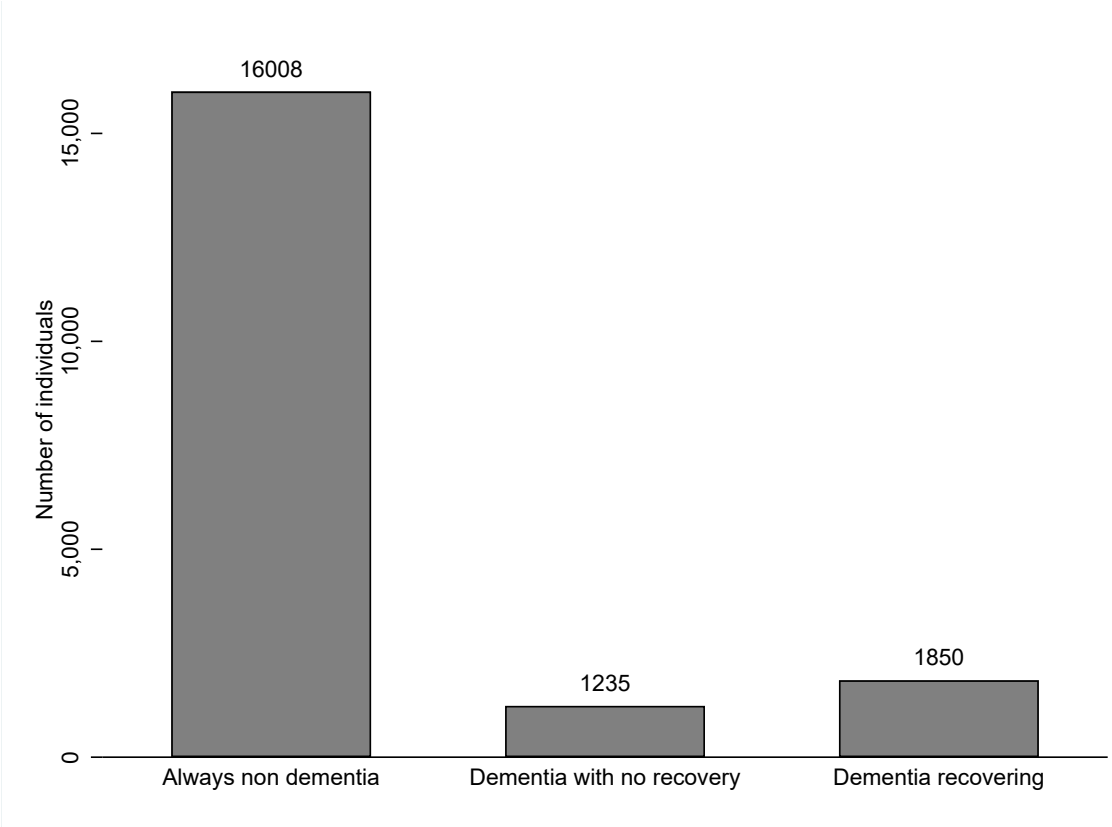
It is also examined if there exist big differences in the number of recoveries that individuals experience. Some individuals might have consistently elevated cognition, score below the cut-off score of dementia at one wave and then persistently score above in the subsequent waves. Other individuals might score below the cut-off of dementia, then score above that in one wave and after that score consistently below in the subsequent waves. Figure C.4 presents the number of individuals that experience a different number of recoveries after first being diagnosed with dementia. Most individuals (62%) only present one or two recoveries and for the rest, there exists high variability in the number of recoveries. This indicates that the problem might lie only in a part of this sub-sample.

To examine these differences in the number of recovery periods, the average cognitive score before and after first having been diagnosed with dementia is calculated, for individuals experiencing a different number of recovery periods. Figure C.5 shows that on average individuals having only one recovery period score well below the threshold at the subsequent survey years, while individuals having two or more recovery periods on the average score above or very close to the threshold at the subsequent survey years.

This shows that there exist great differences in the number of recoveries for the different individuals and that there also exist great differences in their average scores after having first been diagnosed with dementia. This suggests that a part of the individuals recovering after first being diagnosed with dementia should be considered to have a normal condition, while the other truly suffer from dementia. To account for this error, the screening instrument for dementia of Crimmins *et al.* (2011) is slightly modified, and individuals are

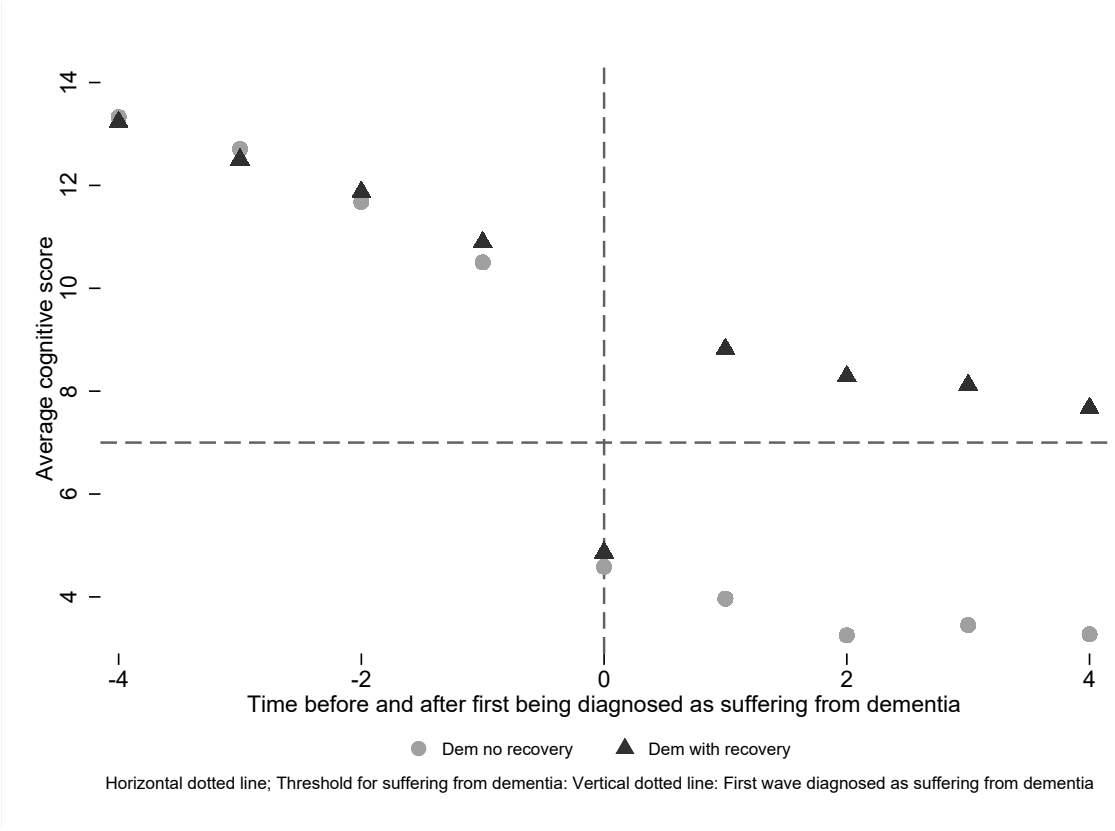
only predicted to suffer dementia if their average score in the subsequent periods after first having been diagnosed with dementia is below the cut-off. This makes it possible to keep the largest possible number of observations but clean a part of the dubious cases of dementia.

Figure C.1: Number of individuals predicted to suffer dementia, never predicted to suffer dementia and predicted to suffer dementia and to recover



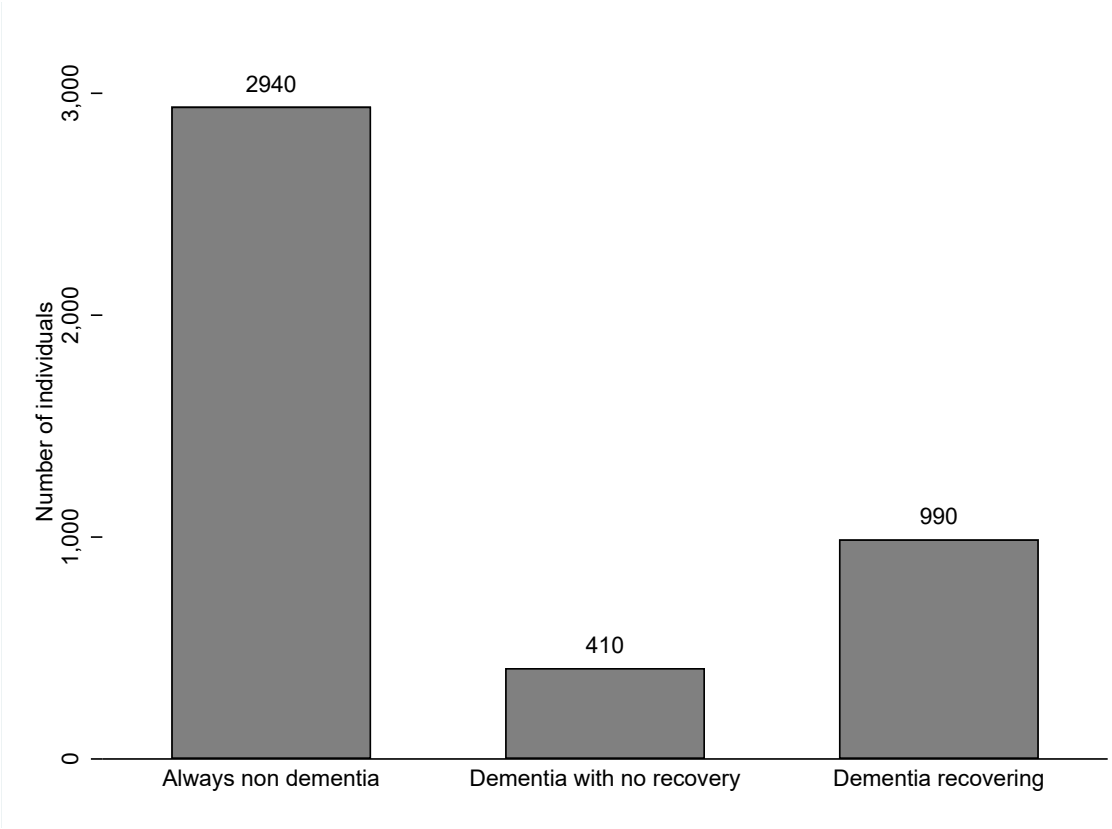
Note: Sample restricted to males aged 60 to 85. *Predicted dementia:* Dementia is defined as scoring below a threshold in a psychometric test validated by Crimmins *et al.* (2011). More details of this variable can be found in appendix C. Considers number of individuals never being predicted to suffer dementia, being predicted to suffer dementia at one wave and not being predicted to suffer dementia at some subsequent wave, and being predicted to suffer dementia at one wave and all other subsequent waves. *Source*: HRS.

Figure C.2: Average cognitive scores before and after being predicted to suffer dementia, for recoveries and non-recoveries



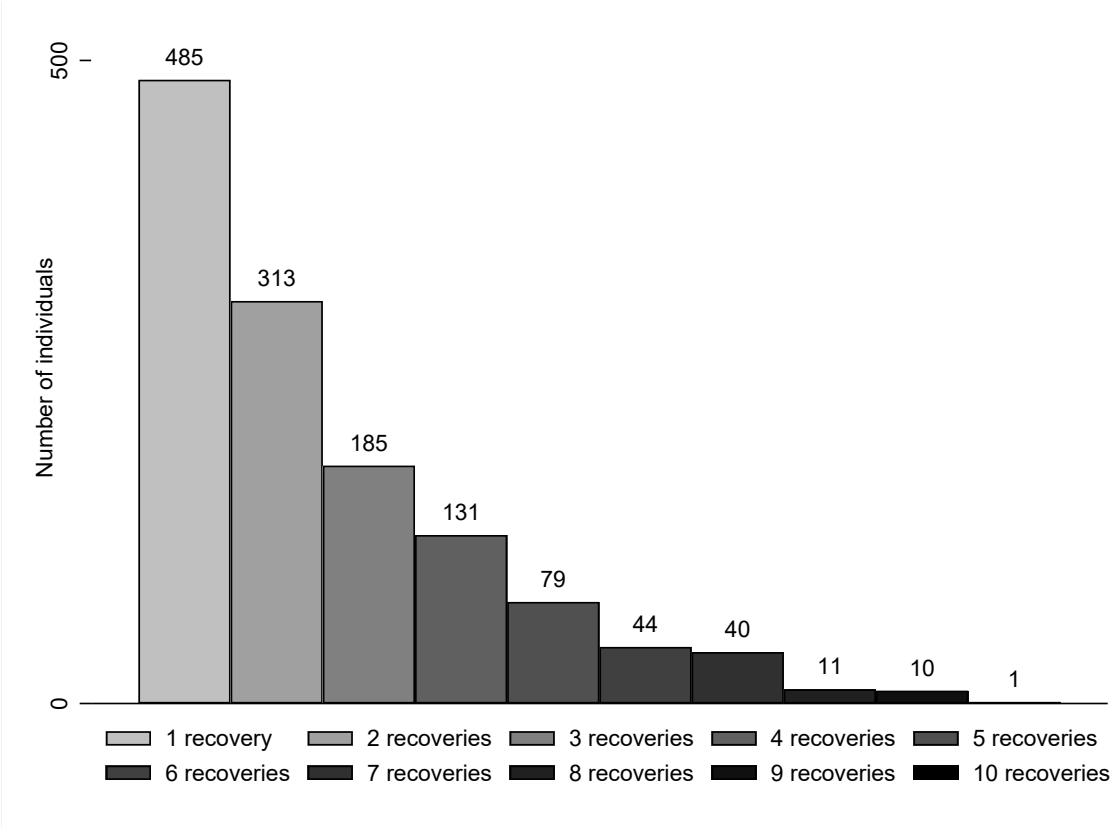
Note: Sample restricted to males aged 60 to 85. *Predicted dementia:* Dementia is defined as scoring below a threshold in a psychometric test validated by Crimmins *et al.* (2011). More details of this variable can be found in appendix C. Does not include proxy respondents, but the results have been replicated for these and are very similar. *Source*: HRS.

Figure C.3: Number of proxy individuals predicted to suffer dementia, never predicted to suffer dementia and predicted to suffer dementia and to recover



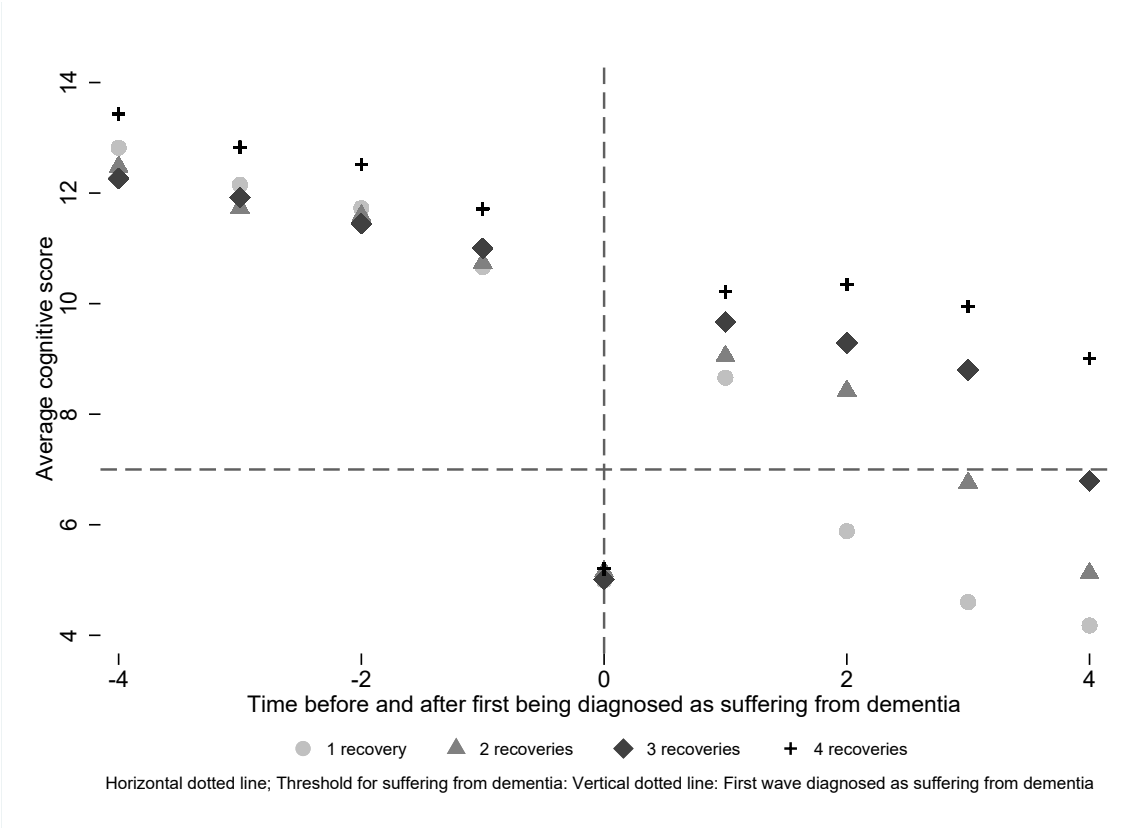
Note: Sample restricted to males aged 60 to 85. *Predicted dementia:* Dementia is defined as scoring below a threshold in a psychometric test validated by Crimmins *et al.* (2011). More details of this variable can be found in appendix C. Considers number of proxy individuals never being predicted to suffer dementia, being predicted to suffer dementia at one wave and not being predicted to suffer dementia at some subsequent wave, and being predicted to suffer dementia at one wave and all other subsequent waves. *Source*: HRS.

Figure C.4: Number of individuals experiencing 1, 2, ..., 10 recoveries after first being diagnosed of dementia



Note: Sample restricted to males aged 60 to 85. *Predicted dementia:* Dementia is defined as scoring below a threshold in a psychometric test validated by Crimmins *et al.* (2011). More details of this variable can be found in appendix C. Recoveries are being predicted to suffer dementia at one wave and not being predicted to suffer dementia at some subsequent wave. *Source*: HRS.

Figure C.5: Average cognitive score before and after first screened of dementia, for individuals experiencing one, two, three, and four recoveries



Note: Sample restricted to males aged 60 to 85. *Predicted dementia:* Dementia is defined as scoring below a threshold in a psychometric test validated by Crimmins *et al.* (2011). More details of this variable can be found in appendix C. Does not consider proxy respondents, but results are very similar for them. *Source*: HRS.

D Construction of independent variable

Longest lifetime occupation is the main interest, independent variable this information is found in the HRS restricted data of industry and occupation[16]. It is not homogeneously coded and questions regarding longest lifetime occupation are quite different across survey years. The approach of Bugliari *et al.* 2018 is followed to generate a measure of longest lifetime occupation across individuals, trying to lose as few observations as possible. This approach is a hierarchical combination of three different variables: (1) longest lifetime occupation; (2) last occupation; (3) current occupation. Longest lifetime occupation refers to the occupation that individuals hold for the longest period of time. Last occupation assumes that the last occupation of individuals was their longest lifetime occupation. Current occupation makes it possible to approximate the last occupation of individuals, if these retire in a further survey year. The hierarchical combination goes as follows. Longest lifetime occupation is the preferred variable, but this variable is not observed for some

[16] There is no detailed information regarding the dates when the work activities were performed, nor how many years they lasted. This data limitation makes it impossible to explore this time dimension within work activities.

individuals. Last occupation is the second best option and if neither of both variables are observed the current occupation of individuals is used.

There is information of individuals at different survey years, while the longest lifetime occupation is fixed for each of them over time. Only observing one of these variables at one survey year is enough to capture the longest lifetime occupation of individuals.

The HRS restricted data of industry and occupation leads to some 1000 SOC occupation codes for the different individuals. For each of the different occupations a score is assigned for the different work activities. The work activities scores are generated from the O*NET database, following a factor analysis that is summarized in the next section. The factor analysis leads to the three main work activities factors that are label as: (1) non-cognitive, (2) cognitive, and (3) physical work activities. Each individual at the database has a score on these factors, depending on his longest lifetime occupation.

A problem of this analysis is that the occupations considered in the O*NET are a slightly different respect to the ones of the HRS and that the occupation categories of the HRS vary over time. This is solved generating a group of homogeneous occupations for the HRS and O*NET following David and Dorn 2013. This leads to 330 occ1990cc homogeneous occupations codes for both databases. The factor analysis at the O*NET is generated for these 330 occ1990cc occupation codes and the generated factors are assigned to the occ1990cc occupation codes in the HRS.

E Factor analysis

The O*NET database offers a large number of variables with occupational information. These are classified in six different groups which consider abilities, interests, knowledge, skills, work activities and work context. This essay explores the correlation between the activities performed during the main lifetime occupation of individuals and the likelihood of having dementia at old age. Work activities approach this definition, describing job behaviours occurring on multiple jobs and are divided in four main domains: (1) information Input (where and how data required for the job is obtained, five variables); (2) interacting with others (which interactions occur during the performance of the job, 17 variables); (3) work Output (which physical activities are performed and what equipment is used for the job, nine variables); (4) mental Processes (which kind of mental processes are required during the performance of the job, 10 variables).

Orthogonally rotated factors are considered, because this allows them to correlate with each other[17]. It is possible a same occupation having a heavy work load at different work activities at once or on the contrary having a low work load in all of them. Some occupations are more intensive in general terms than others

[17] The factor analysis has been calculated with the Stata factor command.

and therefore can have higher loadings at several work activities. Because of these differences in intensities, there does not necessarily occur a trade off between work activities. As a robustness, table F.3 presents the results of the main specification using obliquely correlated factors and including a fourth factor. In general terms, the results do almost not differ respect the ones with oblique rotations and with three factors.

The number of retained factors is chosen considering the loadings of the different variables upon the factors. Table E.1, presents sorted loadings of the different orthogonally rotated variables upon the factors. Three main factors are retained with different loads. The variables from 'Guiding, directing and motivating subordinates' to 'Performing administrative activities' mainly load upon Factor 1. The variables from 'Processing information' to 'Handling objects' mainly load upon Factor 2. The variables from 'Repairing and maintaining mechanical equipment' to 'Drafting, laying out and specifying technical devices, parts and equipment' mainly load upon Factor 3. It seems reasonable not to include a fourth factor, as only one variable has a heavy load upon it. Figure E.1 shows a scree plot for this analysis and supports the choice of three factors, as the first three factors have Eigenvalues well above 1. It could be considered to retain factors 4 and 5, since the Keiser rule sets as a rule of thumb to retain factors with Eigenvalues greater then 1. Nevertheless, their values are very close to one and therefore it reasonable to exclude them. The three retained factors can be labelled considering the loadings of the different variables upon them: factor 1, labeled as non-cognitive work activities; factor 2, labeled as cognitive work activities; and factor 3, labeled as physical work activities.

The same analysis is run for work context and knowledge O*NET variables, with work context referring to physical and social factors that influence the nature of work and knowledge to organized sets of principles and fact applying in general domains such as mathematics, medicine, biology, etc. Five factors are generated for work context and six for knowledge and these are included as controls at the regression analysis. Screeplots and sorted loading can be found at Figures E.2 and E.3 and tables E.2 and E.3.

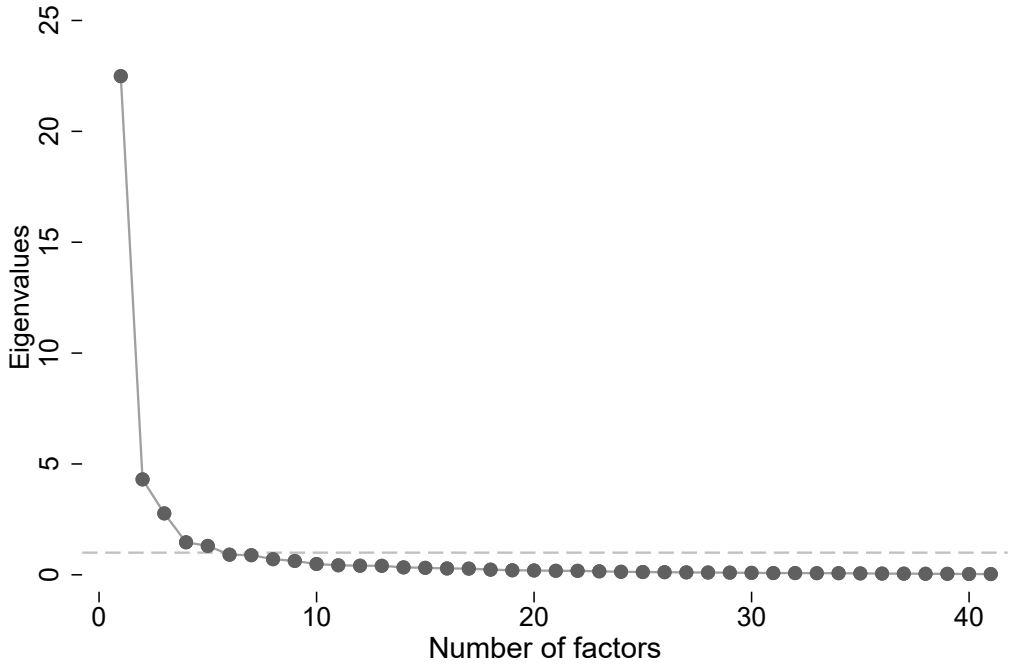
Results for alternative definitions of cognitive work activities can be found in Table F.6. It presents results for alternative estimations of the work activities factor considering oblique and orthogonal rotations. The findings of this table are highly consistent with the main specification.

Table E.1: Variables of work activities sorted according to the orthogonally rotated variables loadings over factors

	Factor1	Factor2	Factor3	Factor4	Uniqueness
Coaching and developing others	0.89	0.24	-0.01	0.08	0.14
Guiding, directing, and motivating subordinates	0.89	0.16	-0.07	0.25	0.10
Developing and building teams	0.89	0.29	-0.03	0.06	0.12
Scheduling work and activities	0.87	0.32	-0.05	0.02	0.11
Coordinating the work and activities of others	0.87	0.23	-0.05	0.21	0.14
Resolving conflicts and negotiating with others	0.86	0.30	-0.10	-0.07	0.14
Training and teaching others	0.85	0.31	0.03	0.05	0.17
Establishing and maintaining interpersonal relationships	0.84	0.42	-0.13	-0.17	0.07
Developing objectives and strategies	0.83	0.41	-0.09	0.06	0.11
Staffing organizational units	0.82	0.11	-0.14	0.24	0.23
Organizing, planning and prioritizing work	0.76	0.47	-0.14	-0.03	0.16
Monitoring and controlling resources	0.75	0.16	-0.06	0.18	0.33
Selling or influencing other	0.75	0.21	-0.11	-0.24	0.32
Communicating with supervisors, peers or subordinates	0.74	0.51	-0.00	0.04	0.18
Performing for or working directly with the publi	0.74	0.18	-0.01	-0.28	0.24
Communicating with persons outside organization	0.72	0.46	-0.14	-0.26	0.19
Assisting and caring for others	0.71	0.21	0.08	-0.07	0.24
Provide consultation and advice to others	0.70	0.54	-0.10	0.07	0.17
Making decisions and solving problems	0.70	0.58	0.06	0.15	0.12
Thinking creatively	0.65	0.40	-0.11	-0.16	0.26
Performing administrative activities	0.64	0.51	-0.20	0.02	0.28
Judging the qualities of things, services or people	0.60	0.32	-0.01	0.35	0.36
Processing information	0.40	0.82	-0.14	0.04	0.15
Analyzing data or information	0.37	0.78	-0.10	0.21	0.14
Getting information	0.31	0.75	-0.06	0.21	0.28
Interacting with computers	0.44	0.74	-0.21	-0.20	0.17
Documenting/recording information	0.49	0.70	0.01	0.10	0.22
Updating and using relevant knowledge	0.60	0.70	0.10	-0.04	0.13
Performing general physical activities	-0.13	-0.68	0.51	0.11	0.24
Interpreting the meaning of information for others	0.64	0.65	-0.13	-0.07	0.14
Identifying objects, actions and events	0.37	0.65	0.20	0.38	0.26
Evaluating information to determine compliance with standards	0.27	0.61	0.13	0.37	0.39
Handling and moving objects	-0.49	-0.59	0.46	0.16	0.17
Repairing and maintaining mechanical equipment	-0.26	-0.24	0.79	0.12	0.22
Repairing and maintaining electronic equipment	-0.02	0.24	0.77	-0.08	0.33
Inspecting equipment, structures or material	-0.11	-0.13	0.76	0.40	0.21
Operating vehicles, mechanized devices or equipment	0.25	-0.18	0.67	-0.13	0.43
Controlling machines and processes	-0.47	-0.38	0.57	0.29	0.22
Monitor processes, materials or surroundings	0.13	0.21	0.42	0.73	0.22
Drafting, laying out and specifying technical devices, parts and equipment	0.03	0.13	0.37	-0.09	0.25
Estimating the quantifiable characteristics of products, events or information	0.33	0.26	0.12	0.38	0.29

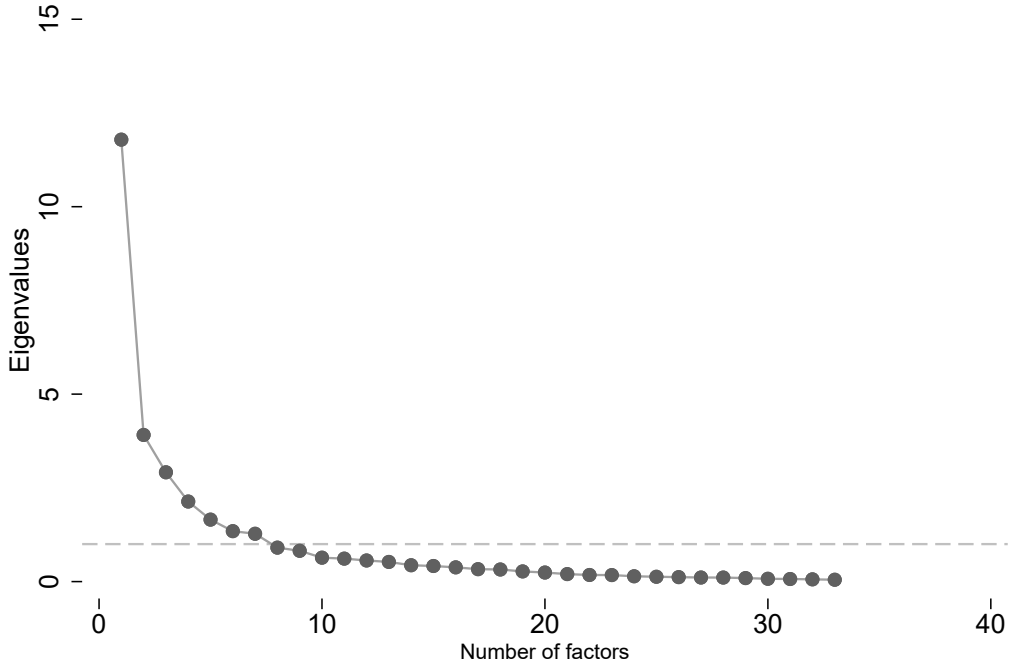
Note: Sample restricted to males aged 60 to 85. *Predicted dementia:* Dementia is defined as scoring below a threshold in a psychometric test validated by Crimmins *et al.* (2011). More details of this variable can be found in appendix C. Does not consider proxy respondents, but results are very similar for them. *Source*: HRS.

Figure E.1: Scree-plot of Eigenvalues of each factor of work activities



Source: O*NET

Figure E.2: Scree-plot of Eigenvalues of each factor of work related knowledge



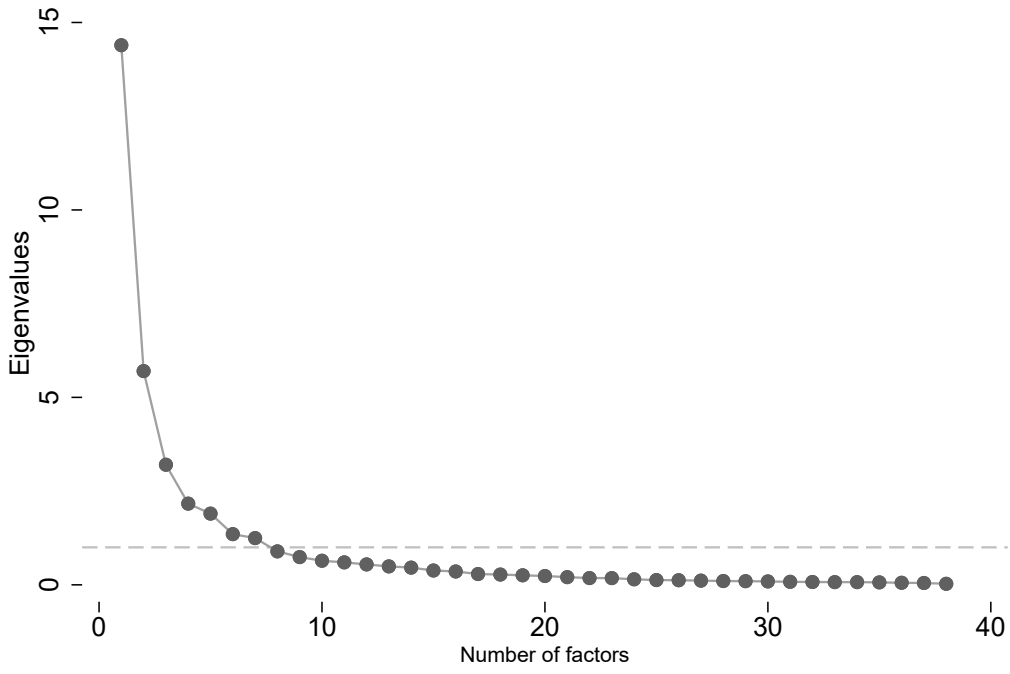
Source: O*NET

Table E.2: Sorted loadings of orthogonally rotated variables of work related knowledge over factors

	Factor1	Factor2	Factor3	Factor4	Factor5	Factor6	Uniqueness
Therapy and counseling	0.92	0.12	-0.11	-0.01	0.05	-0.02	0.13
Psychology	0.87	0.35	-0.09	0.10	0.08	0.12	0.09
Medicine and dentistry	0.84	0.02	-0.03	-0.05	0.13	-0.20	0.17
Sociology and anthropology	0.82	0.25	-0.11	0.14	0.10	0.33	0.11
Philosophy and theology	0.79	0.18	-0.07	0.13	0.07	0.31	0.21
Education and training	0.72	0.48	0.06	0.14	0.17	0.18	0.16
Foreign language	0.55	0.17	-0.18	0.19	0.20	0.24	0.50
Economics and accounting	0.10	0.88	-0.13	0.05	0.15	-0.01	0.17
Administration and management	0.38	0.81	0.01	0.16	0.11	0.05	0.15
Sales and marketing	0.12	0.75	-0.13	0.02	0.07	0.16	0.36
Personnel and human resources	0.46	0.72	-0.05	0.10	0.03	0.03	0.25
Customer and personal service	0.50	0.62	-0.22	0.18	0.09	-0.07	0.26
Clerical	0.22	0.57	-0.27	0.15	0.47	-0.08	0.29
English language	0.50	0.55	-0.08	0.19	0.48	0.18	0.14
Mathematics	0.02	0.55	0.36	-0.01	0.47	-0.07	0.32
Law and government	0.50	0.55	0.03	0.36	0.15	0.00	0.29
Engineering and technology	-0.12	-0.07	0.92	0.06	0.11	-0.06	0.11
Design	-0.09	0.11	0.83	-0.03	-0.03	0.22	0.21
Physics	0.07	-0.28	0.76	0.11	0.25	-0.13	0.19
Mechanical	-0.19	-0.36	0.65	0.02	-0.29	-0.28	0.24
Building and construction	-0.10	-0.05	0.61	0.21	-0.45	0.04	0.33
Production and processing	-0.20	0.17	0.47	-0.32	-0.18	-0.06	0.47
Transportation	0.03	0.15	0.06	0.83	-0.02	-0.10	0.27
Geography	0.09	0.08	0.01	0.75	0.29	0.36	0.20
Public safety and security	0.47	0.22	0.27	0.63	-0.10	-0.18	0.22
Computer and electronics	0.23	0.43	0.19	0.08	0.70	0.07	0.21
Telecommunications	0.22	0.29	0.02	0.49	0.52	0.02	0.32
Communications and media	0.35	0.39	-0.07	0.23	0.51	0.43	0.23
Fine arts	0.12	-0.08	-0.06	-0.18	-0.05	0.75	0.37
History and archeology	0.44	0.14	0.02	0.27	0.15	0.66	0.25
Food production	-0.05	0.21	-0.18	0.09	-0.24	0.10	0.26
Biology	0.59	0.03	0.05	0.04	0.11	-0.05	0.20
Chemistry	0.36	-0.15	0.41	-0.06	0.08	-0.17	0.29

Source: O*NET

Figure E.3: Scree-plot of Eigenvalues of each factor of work context



Source: O*NET

Table E.3: Sorted loadings of orthogonally rotated variables of work related context over factors

	Factor1	Factor2	Factor3	Factor4	Factor5	Uniqueness
Sitting	-0.86	-0.20	0.09	-0.02	-0.18	0.16
Standing	0.86	0.09	-0.00	0.08	0.09	0.23
Bending, twisting body	0.70	0.22	-0.11	0.20	0.45	0.15
Exposed to minor burns, cutes, bites	0.69	0.26	-0.29	0.30	0.17	0.24
Kneeling	0.67	0.30	-0.13	0.08	0.46	0.21
Exposed to contaminants	0.62	0.41	-0.31	0.30	0.15	0.21
Common protective equipment	0.59	0.24	-0.33	0.35	0.19	0.11
Exposed to hazardous conditions	0.48	0.31	-0.35	0.33	0.26	0.29
Indoors	-0.12	-0.92	-0.02	0.07	-0.17	0.10
Outdoors	0.19	0.89	0.11	-0.12	0.22	0.10
Extremely birght inadecuate lighting	0.35	0.72	-0.04	0.20	0.32	0.20
Hot or cold temperatures	0.49	0.71	-0.16	0.19	0.21	0.15
Exposed to whole body vibration	0.14	0.67	-0.19	0.41	0.21	0.28
Deal with unpleasent or angry people	-0.13	-0.05	0.87	-0.25	-0.10	0.14
Frequency of conflict situations	-0.22	-0.02	0.79	-0.11	-0.03	0.16
Contact with others	-0.19	-0.12	0.74	-0.32	-0.16	0.16
Deal with physically agressive people	-0.07	0.12	0.74	-0.05	-0.09	0.40
Deal with external customers	-0.12	-0.11	0.73	-0.40	-0.16	0.25
Walking	0.46	0.33	0.52	0.03	0.11	0.38
Degree of automation	-0.03	-0.07	-0.18	0.86	-0.07	0.22
Pace determined by speed of equipment	0.22	0.01	-0.28	0.84	-0.06	0.14
Exposed to hazardous equipment	0.46	0.30	-0.33	0.63	0.17	0.15
Sounds, noise levels are uncomfortable	0.44	0.46	-0.14	0.62	0.16	0.16
Using hands to handle control objects	0.47	0.07	-0.45	0.47	0.22	0.18
Exposed to high places	0.16	0.34	-0.10	0.00	0.82	0.16
Climbing	0.34	0.37	-0.18	0.02	0.77	0.12
Balance	0.49	0.36	-0.01	0.09	0.67	0.17
Cramped work space	0.51	0.37	-0.25	0.16	0.54	0.21
Exposed to radiation	0.09	0.03	-0.01	-0.07	0.00	0.33
Exposed to disease or infections	0.21	-0.14	0.24	-0.18	-0.18	0.26
Consequence of error	-0.21	0.07	0.05	0.19	0.20	0.22
Responsible for others health and safety	0.20	0.22	0.40	0.13	0.12	0.21
Importance of being exact or accurate	-0.50	-0.26	-0.15	0.06	0.16	0.34
Specialized protective equipment	0.30	0.29	-0.28	0.08	0.22	0.43
Responsible for outcomes and results	-0.10	-0.09	0.27	0.03	0.10	0.19
Coordinate or lead others	-0.17	-0.05	0.33	-0.09	0.05	0.18
Importance repeating same tasks	-0.03	-0.06	-0.04	0.55	0.25	0.26
Repetitive motions	0.29	-0.12	-0.18	0.50	0.31	0.19

Source: O*NET

F Extended results

Table F.1: Multivariate linear regressions estimated through pooled OLS – Different predictors for dementia as dependent variable and cognitive work activity as main interest independent

	(1)	(2)	(3)	(4)	(5)
VARIABLES	Dem. Crimm.	Dem. Crimm.	Dem. Hurd	Dem. Expert	Dem. Lasso
Cognitive work activity	-0.69***	-1.27***	-1.84***	-1.41***	-1.51***
	(0.255)	(0.371)	(0.520)	(0.461)	(0.466)
Observations	43,018	18,623	18,623	18,815	17,972
R-squared	0.10	0.12	0.13	0.13	0.16
Baseline covariates	✓	✓	✓	✓	✓
Mean	4.13	5.79	8.96	9.13	9.53

Note: Standard errors clustered at occupational level in parentheses, *** p<0.01; ** p<0.05; * p<0.10. Sample restricted to males aged 60 to 85. *Pred. Dem. Crimm.:* Dementia is defined as scoring below a threshold in a psychometric test validated by Crimmins *et al.* (2011). More details of this variable can be found in appendix C. *Dem. Hurd.:* Prediction of dementia of Hurd *et al.* (2013). From zero to one with zero being lower probabilities of suffering dementia and vice-versa. *Dem. Expert.:* Prediction of dementia from expert model of Gianattasio *et al.* (2020). From zero to one with zero being lower probabilities of suffering dementia and vice-versa; *Dem. Lasso.:* Prediction of dementia from Lasso model of Gianattasio *et al.* (2020). From zero to one with zero being lower probabilities of suffering dementia and vice-versa. *Cognitive work activity:* Cognitive load of longest lifetime occupation, standardized with mean zero and standard deviation one. *Demographics:* Race, being a proxy respondent and parents education, age, survey year, place of birth. *Education:* Five education levels. *Occupation and industry:* 11 main occupations and 13 main industries. *Non-cognitive and physical work activities:* Two work activities factors. *Context:* Five Workplace context factors. *Source*: HRS and O*NET.

Table F.2: Multivariate logit and probit – Predicted dementia as dependent variable and cognitive work activity as main interest independent

	(1)	(2)	(3)
VARIABLES	OLS	Logit	Probit
Cognitive work activity	-0.69***	-0.20***	-0.10***
	(0.255)	(0.077)	(0.036)
Observations	43,018	43,018	43,018
Baseline covariates	✓	✓	✓
Mean	4.13	4.13	4.13

Note: Standard errors clustered at occupational level in parentheses, *** p<0.01; ** p<0.05; * p<0.10. Sample restricted to males aged 60 to 85. *Predicted dementia:* Dementia is defined as scoring below a threshold in a psychometric test validated by Crimmins *et al.* (2011). More details of this variable can be found in appendix C. *Cognitive work activity:* Cognitive load of longest lifetime occupation, standardized with mean zero and standard deviation one. *Demographics:* Race, being a proxy respondent and parents education, age, survey year, place of birth. *Education:* Five education levels. *Occupation and industry:* 11 main occupations and 13 main industries. *Non-cognitive and physical work activities:* Two work activities factors. *Context:* Five Workplace context factors. *Source*: HRS and O*NET.

Table F.3: Multivariate linear and non-linear regressions estimated through pooled OLS – Predicted dementia as dependent variable and cognitive work activity as main interest independent variable

VARIABLES	(1)	(2)	(3)
Cognitive work activity	-0.69***	-0.73**	0.68
	(0.255)	(0.282)	(2.186)
$Cognitiveworkactivity^2$		0.08	
		(0.188)	
$\sqrt{Cognitiveworkactivity}$			-4.59
			(7.283)
Observations	43,018	43,018	43,018
R-squared	0.10	0.10	0.10
Baseline covariates	✓	✓	✓
Mean outcome	4.13	4.13	4.13

Note: Standard errors clustered at occupational level in parentheses, *** p<0.01; ** p<0.05; * p<0.10. Sample restricted to males aged 60 to 85. *Predicted dementia:* Dementia is defined as scoring below a threshold in a psychometric test validated by Crimmins *et al.* (2011). More details of this variable can be found in appendix C. *Cognitive work activity:* Cognitive load of longest lifetime occupation, standardized with mean zero and standard deviation one. *Demographics:* Race, being a proxy respondent and parents education, age, survey year, place of birth. *Education:* Five education levels. *Occupation and industry:* 11 main occupations and 13 main industries. *Non-cognitive and physical work activities:* Two work activities factors. *Context:* Five Workplace context factors. *Source*: HRS and O*NET.

Table F.4: Descriptive variables of the full sample and the sample of non-missing non-cognition related health outcomes

	(1) Full sample		(2) Sample – non-missing health out.	
	mean	sd	mean	sd
White	0.79	0.41	0.84	0.37
Black	0.12	0.32	0.10	0.30
Hispanic	0.08	0.26	0.04	0.21
No high-school	0.24	0.43	0.30	0.46
GED	0.05	0.22	0.05	0.22
High-school	0.28	0.45	0.25	0.43
Some college	0.18	0.39	0.19	0.39
College and above	0.24	0.43	0.22	0.41
White collar	0.39	0.49	0.46	0.50
Blue collar	0.44	0.50	0.38	0.49
Pink collar	0.17	0.37	0.16	0.37
Age	70.79	6.61	72.71	4.64

Note: Sample restricted to males aged 60 to 85. Non-cognition related health outcomes are arthritis, cancer, and lung disease. *Source*: HRS and O*NET.

Table F.5: Multivariate linear regressions estimated through pooled OLS – Predicted dementia as dependent variable and cognitive work activity as main interest independent variable for age groups

VARIABLES	(1) Age<65	(2) Age 65-75	(3) Age>75
Cognitive work activity	-0.07	-0.61*	-1.39**
	(0.189)	(0.357)	(0.550)
Observations	17,130	22,884	13,227
R-squared	0.04	0.08	0.19
Baseline covariates	✓	✓	✓
Mean	0.95	3.23	9.90

Note: Standard errors clustered at occupational level in parentheses, *** p<0.01; ** p<0.05; * p<0.10. Sample restricted to males aged 60 to 85. *Predicted dementia:* Dementia is defined as scoring below a threshold in a psychometric test validated by Crimmins *et al.* (2011). More details of this variable can be found in appendix C. *Cognitive work activity:* Cognitive load of longest lifetime occupation, standardized with mean zero and standard deviation one. *Demographics:* Race, being a proxy respondent and parents education, age, survey year, place of birth. *Education:* Five education levels. *Occupation and industry:* 11 main occupations and 13 main industries. *Non-cognitive and physical work activities:* Two work activities factors. *Context:* Five Workplace context factors. *Source*: HRS and O*NET.

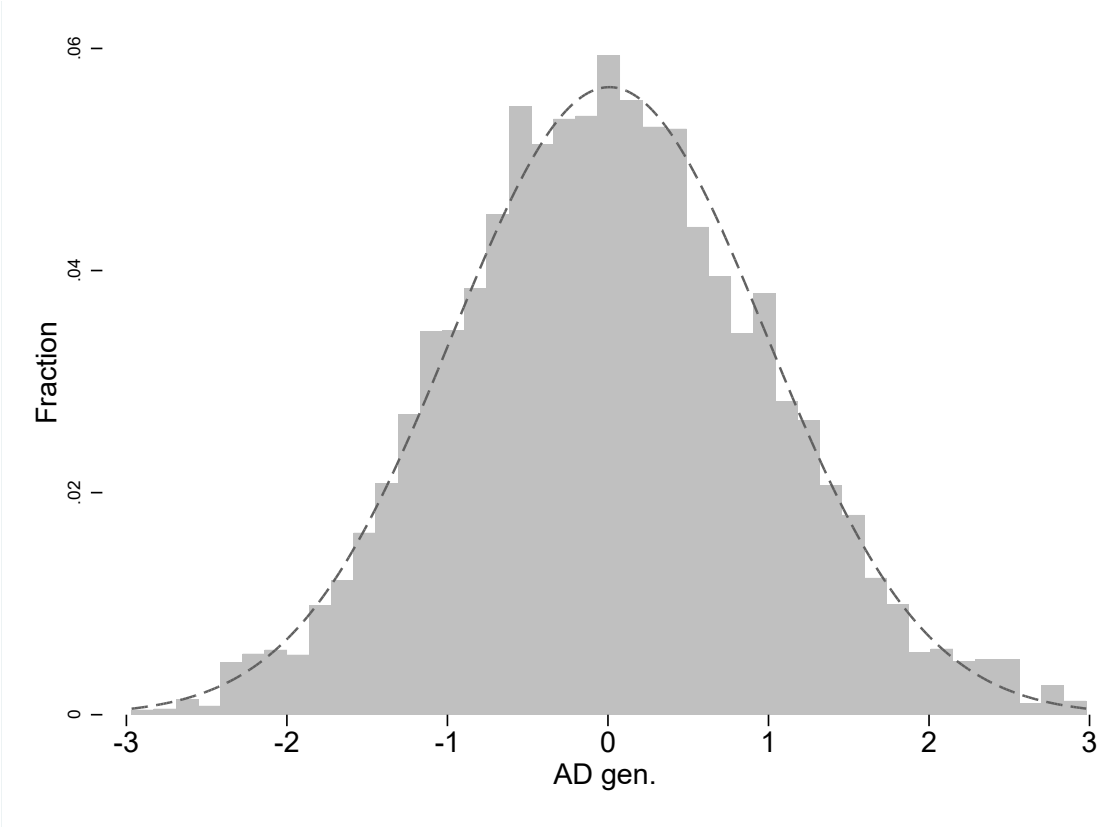
Table F.6: Multivariate linear regressions estimated through pooled OLS – Predicted dementia as dependent variable and orthogonal and obliquely rotated cognitive work activity as main interest independent

VARIABLES	(1) Otrhogonal rot.	(2) Otrhogonal rot.	(3) Oblique rot.	(4) Oblique rot.
Work cognitive activity	-0.69***	-0.62**	-0.54**	-0.50*
	(0.255)	(0.265)	(0.268)	(0.258)
Observations	43,018	43,018	43,018	43,018
R-squared	0.10	0.10	0.10	0.10
Baseline covariates	✓	✓	✓	✓
4th Work activity factor	✗	✓	✗	✓
Mean	1.57	1.57	1.57	1.57

Note: Standard errors clustered at occupational level in parentheses, *** p<0.01; ** p<0.05; * p<0.10. Sample restricted to males aged 60 to 85. *Predicted dementia:* Dementia is defined as scoring below a threshold in a psychometric test validated by Crimmins *et al.* (2011). More details of this variable can be found in appendix C. *Cognitive work activity:* Cognitive load of longest lifetime occupation, standardized with mean zero and standard deviation one. *Demographics:* Race, being a proxy respondent and parents education, age, survey year, place of birth. *Education:* Five education levels. *Occupation and industry:* 11 main occupations and 13 main industries. *Non-cognitive and physical work activities:* Two work activities factors. *Context:* Five Workplace context factors. *4th Work activity factor:* It is the fourth work activities factor. *Source*: HRS and O*NET.

G Genetics

Figure G.1: Histogram of poly-genetic risk score for dementia



Note: Sample size 25,411 observations and normal distribution fitted. *Source*: HRS

Essay 3: Risky and non-risky financial investments and cognition

1 Introduction

Age-related cognitive deterioration is one of the biggest challenges worldwide, and it is estimated to become an even more significant problem in the near future. For example, in the United Kingdom (UK), around 850,000 individuals had dementia in 2019, and this is estimated to rise to 1.5 million individuals by 2040 (Alzheimer's Society, 2019). This form of extreme cognitive deterioration is not exclusive to the oldest individuals, up to 42,000 individuals aged 65 or younger have dementia in the UK.

One of the most problematic consequences of age-related cognitive decline is that it can significantly impact an individual's decision making, and good financial decision-making is particularly relevant for older adults. In 2018, in the UK, there were 12 million individuals aged 65 or above, most of them retired, who strongly relied on savings and financial decisions taken over their lifetime (Age UK, 2019). Financial decisions can be highly complex and sophisticated and depend on an individual's age and wealth. Optimal decisions about risky and non-risky financial investments can maximise wealth at old age and significantly improve quality of life. On the contrary, suboptimal financial decisions can lead to deprivation at old age. For example, the average female in the UK is predicted to outlive her savings by 12.6 years and the average male by 10.3 years (Amishi and Han, 2019). This risk of financial deprivation at older age highlights the importance of optimising financial investments throughout life.

This essay explores how overall cognitive level and cognitive deterioration affect the financial decisions of households' composed of older adults. Three research questions are analysed. Firstly, does risky and non-risky investment behaviour differ by level of cognition? To answer this question, differences in investment behaviour between households by their level of cognition are compared. Secondly, does cognitive deterioration affect risky and non-risky investment behaviour? In answering this question, it is analysed

how household changes in cognition affect their investment behaviour over time. Thirdly, do reactions to fluctuations of stock market prices differ by levels of cognition? To answer this question, it is explored if households react to such fluctuations by changing their investment in risky and non-risky financial investments and whether these reactions differ by levels of cognition.

Data from the English Longitudinal Study of Ageing (ELSA) is analysed to answer these questions. This data is a nationally representative sample of older adults in the UK containing information about the cognitive level and ownership of financial investments over almost two decades. The panel structure of the ELSA allows estimation of the individual-level fixed-effects models to analyse the associations between cognitive decline within households and changes in financial investment. The analysis is based on eight ELSA survey waves and includes several time-varying controls for health, wealth, and labour-force status. In addition, potential misreporting of financial investments, driven by individuals with lower levels of cognition systematically misreporting their financial outcomes, is addressed by considering if individuals used technical documentation to provide their financial information.

It is found that investing in financial assets is strongly associated with cognition. One additional standard deviation in cognition increases the likelihood of holding risky financial investments by 10% and non-risky ones by 11%. Cognitive deterioration is associated with both the probability of holding risky financial investments and their relative value.

Suffering a reduction in cognition from the highest cognition group (15–20 correctly recalled words in memory test) to the lowest one (0–5 correctly recalled words) from one survey wave to another reduces the probability of holding risky financial investments by 2.8 percentage points and the value of risky financial investments by 38%. The results further show that non-risky financial investments remain unaffected.

It is also shown that cognition does not moderate households' financial reactions to stock market fluctuations. Individuals react to fluctuations in the stock market, but this reaction does not differ by level of cognition.

Most work has focused on the first research question, examining differences in financial investments between households related to cognition (McArdle *et al.*, 2009; Kézdi and Willis, 2003; Kim *et al.*, 2012; Cole and Shastry, 2009; Grinblatt *et al.*, 2011; Delavande *et al.*, 2008; Almenberg and Widmark, 2011; Banks and Oldfield, 2007; Christelis *et al.*, 2010). Comparatively little work has examined the association between changes in cognition and changes in financial investments over time. Further, there is a need to address cohort effects, unobserved confounding factors, and potential biases not considered so far, such as systematic misreporting of financial investments by the level of cognition. A purely cross-sectional analysis makes it difficult to distinguish the extent to which differences in investment behaviour are due to household differences in cognitive performance or due to other confounding factors.

Only a few papers have considered the second research question, examining the relationship of cognitive deterioration within individuals with investment behaviour (Pak and Babiarz, 2018; Mazzonna *et al.*, 2018; Bogan, 2008). This paper contributes to this literature by addressing potential sources of endogeneity and reverse causality.

The third research question builds on to the paper of Browning and Finke (2015) who explored whether individuals reacted differently to the Great Financial Crisis (GFC) due to their level of cognition. However, Browning and Finke (2015) do not consider the reaction to financial fluctuations but the extreme event of the GFC. Considering overall financial fluctuations makes it possible to identify if individuals react differently to financial information due to their level of cognition.

2 Literature Review

2.1 Reasons why cognition may impact financial investments

Two potential mechanisms may explain the relationship between cognition and financial decision making. The first mechanism argues that myopic risk aversion is negatively associated with cognition (Lilleholt, 2019). Individuals with low cognition levels are usually more risk-averse than individuals with high cognition levels. Thus, cognition reshapes the utility function of individuals, weighting risk perception. This would mean that two hypothetical individuals, identical apart from their cognition levels, have different utility levels from the same amount of risky financial investments due to their differences in risk aversion. As such, it is likely they hold different amounts of risky financial investments. This mechanism has been empirically validated since willingness to take risks decreases with age (Dohmen *et al.*, 2011,0; Bonsang and Dohmen, 2015; Tymula *et al.*, 2013; Mata *et al.*, 2016; Koscielniak *et al.*, 2016), and about 85% of the association between biological ageing and the willingness to take risk can be attributed to cognitive ageing (Bonsang and Dohmen, 2015).

The second mechanism suggests that there are costs associated with making risky financial investments, and these costs are negatively correlated with cognition. Cognitive load is required to process information relative to decision making because individuals need to analyse their odds of succeeding or failing. This cost is considered to be higher for individuals with lower levels of cognition (Dohmen *et al.*, 2018,0; Beauchamp *et al.*, 2017). Thus, two hypothetical individuals, identical apart from their cognition levels, would differ in their optimisation of life cycle financial investments, leading to differences in risky financial investments.

Cognitive deterioration can affect both mechanisms, and given the neuron-degenerative nature of dementia, individuals' cognition can deteriorate for a long time before this can be categorised as a clinical

condition (Crimmins *et al.*, 2011). While still being considered healthy, individuals are held fully responsible for their decisions. This can lead to sub-optimal decisions in many dimensions of individuals' lives, including financial decision making.

2.2 Does risky and non-risky investment behaviour differ due to cognition?

The first research question explores if risky and non-risky investment behaviour differ by level of cognition[1]. It builds on previous research by similarly concluding that households with high cognition levels hold more risky financial investments than those with lower cognition levels (McArdle *et al.*, 2009; Kézdi and Willis, 2003; Kim *et al.*, 2012; Cole and Shastry, 2009; Grinblatt *et al.*, 2011; Delavande *et al.*, 2008; Almenberg and Widmark, 2011; Banks and Oldfield, 2007; Christelis *et al.*, 2010). In this essay similar data and methodologies to Kim *et al.* (2012); Christelis *et al.* (2010); Banks and Oldfield (2007) are used.

Kim *et al.* (2012) and Christelis *et al.* (2010) study the association between cognitive abilities and the probability of holding stocks using the 2004 survey wave of the US Health and Retirement Study (HRS) and the 2004/2005 survey wave of the Survey of Health, Ageing and Retirement in Europe (SHARE) respectively. Both papers show that cognitive performance is positively associated with investments in stocks for all risk profiles.

Banks and Oldfield (2007) scrutinise the relationship between numerical ability and wealth accumulation at old age, using the 2002 survey wave of the ELSA. Their outcome variables are financial wealth[2] and holdings in a spread of assets[3], and the results show that households of less-numerate individuals are less likely to hold riskier assets.

This paper builds on Banks and Oldfield (2007), addressing potential limitations they and other authors highlighted, and that newly available data allows solving. Banks and Oldfield (2007) consider only one survey wave, making it impossible to rule out that cohort effects drive their results. Pak and Babiarz (2018) also suggest the need to account for unobserved factors not considered so far. This essay explores all eight ELSA survey waves, comparing observations of individuals of the same age at different times. It also controls for potential confounding factors such as health, wealth, and labour-force status. Health shocks may impair cognition and, at the same time, reduce the probability of holding risky financial investments. Working may preserve an individual's cognition due to having enough cognitively stimulating activities. However, it may also increase individuals' probability of holding risky financial investments. Contrary to previous papers that only looked at the extensive margin, this work also considers the intensive and

[1] Three research questions are explored that examine whether the relation between ageing, cognition, and willingness to take risk hold for real-life behaviours such as those concerned with risky and non-risky financial investments. Table A.1 summarises objectives, methodologies, and outcomes of the literature concerning these questions.

[2] Financial wealth includes savings (interest-bearing deposit accounts), plus investments (other investment products such as shares and unit trusts), less any outstanding debts(excluding mortgage debts).

[3] Assets considered for the categorical outcome are accounts, shares (defined as riskier assets), and private pensions.

extensive margin of financial investments. Furthermore, potential misreporting of financial investments is addressed, which could be driven by individuals with lower levels of cognition systematically misreporting their financial outcomes.

2.3 Does old-age cognitive deterioration affect risky and non-risky investment behaviour?

The second research question, which analyses if age-related cognitive deterioration affects financial investments, builds on the work of Bogan and Fertig (2013); Pak and Babiarz (2018); Mazzonna *et al.* (2018). These papers also use data from the HRS.

Bogan and Fertig (2013) investigate whether changes in mental health and cognition affect portfolio choice. They find that if households are affected by mental health problems, memory issues or cognitive limitations, they decrease risky investments.

Mazzonna *et al.* (2018) show that individuals who are unaware of their cognitive deterioration face wealth and financial investments losses. Furthermore, all individuals experiencing cognitive decline, regardless of being aware of it, disinvest in risky financial investments but not in non-risky ones.

Pak and Babiarz (2018) show a positive association between cognitive ageing and stock market holdings using individual fixed-effects models and an IV approach. They argue that past studies analysing similar research questions did not address the endogeneity problem of reverse causality and unobserved confounding factors. Reverse causality may come from individuals holding more risky investments being more likely to receive higher returns in the long run and reinvest this surplus in their health. This health reinvestment is likely to prevent cognitive ageing and increase their probability of remaining in the workforce longer, preventing cognitive ageing. Pak and Babiarz (2018) try to address these problems with an IV strategy based on exogenous variation in individuals cognitive levels at certain seasons of the year (Kent *et al.*, 2009). However, their instrument captures seasonal variation in cognitive levels, not cognitive-ageing. Seasonal changes in cognitive levels affect all age groups and are not chronic; individuals suffer mild cognitive decline during some seasons of the year and mild cognitive recovery during other seasons. This seasonal drop in cognition differs greatly from cognitive-ageing (Aartsen *et al.*, 2002).

This is a crucial point when considering economies such as the US, which have private healthcare systems (Pak and Babiarz, 2018), while its less relevant in the UK scenario, which has an universal health care system. Still, other health investments not directly related with health care, such as physical activity and diet, could lead to a similar problem of reverse causality. In this essay, the endogeneity problem of the unobserved confounding factors suggested by Pak and Babiarz (2018) is addressed by including detailed controls for labour force status, wealth and health. It is also important to note that the endogeneity problem

is also less severe in the UK, where education and health access depend less on wealth than in the US. According to Schneider *et al.* (2017), the National Health System (NHS) in England was ranked the safest and most affordable healthcare system of a list of developing countries in 2017, for the second consecutive time, while the US was last[4].

This essay considers continuous and categorical changes in cognition, which further contributes to Pak and Babiarz (2018) who only considered continuous changes. Categorical changes in cognition capture change from high to low cognitive levels and reflect cognitive shocks. Cognitive-ageing is likely to take the form of cognitive shocks with periods of relatively constant cognition levels and stressful events (health) leading to sudden cognitive decline. This essay also analyses the intensive margin of financial investments, considering differences in investment levels. The main estimations are also free of the potential bias caused by individuals with lower cognition levels providing inaccurate information regarding their investments. Finally, heterogeneous effects of cognition by the level of risk aversion are investigated.

2.4 Do reactions to fluctuations of stock market prices differ according to cognitive levels?

The third research question, which investigates if reactions to fluctuations of stock market prices differ by levels of cognition, builds on the work of Browning and Finke (2015). They show that individuals with lower cognition reallocated their risky financial investments after the GFC more than individuals with higher cognition levels.

This essay differs from Browning and Finke (2015) by studying the effect of stock market fluctuations on financial investments and their interaction with cognition level, while they considered a one-time event. The GFC was a one-off event that had dramatic social and economic effects and therefore does not effectively capture the day-to-day processing of financial information. However, using continuous financial fluctuations makes it possible to identify how individuals reacted to sustained moderate changes in stock market prices.

3 Data

3.1 ELSA

The ELSA is an ongoing longitudinal, study that includes a large representative sample of older English adults. Individuals aged 50 and above and their spouses, regardless of age, were interviewed. The sample was generated from households that participated in the Health Survey for England (HSE) and agreed to

[4] Three research questions are explored that examine whether the relation between ageing, cognition, and willingness to take risk hold for real-life behaviours such as those concerned with risky and non-risky financial investments. Table A.1 summarises objectives, methodologies, and outcomes of the literature concerning these questions.

be followed up. The first wave of ELSA was collected between March 2002 and March 2003, after which the survey ran biennially until 2016. After that, individuals were followed over time, with refresher cohorts included in 2006/2007, 2008/2009, and 2012/2013. In each wave, ELSA included an extensive section on financial investments and a battery of psychometric tests. This longitudinal data makes it possible to measure changes in financial outcomes and cognition of individuals over time.

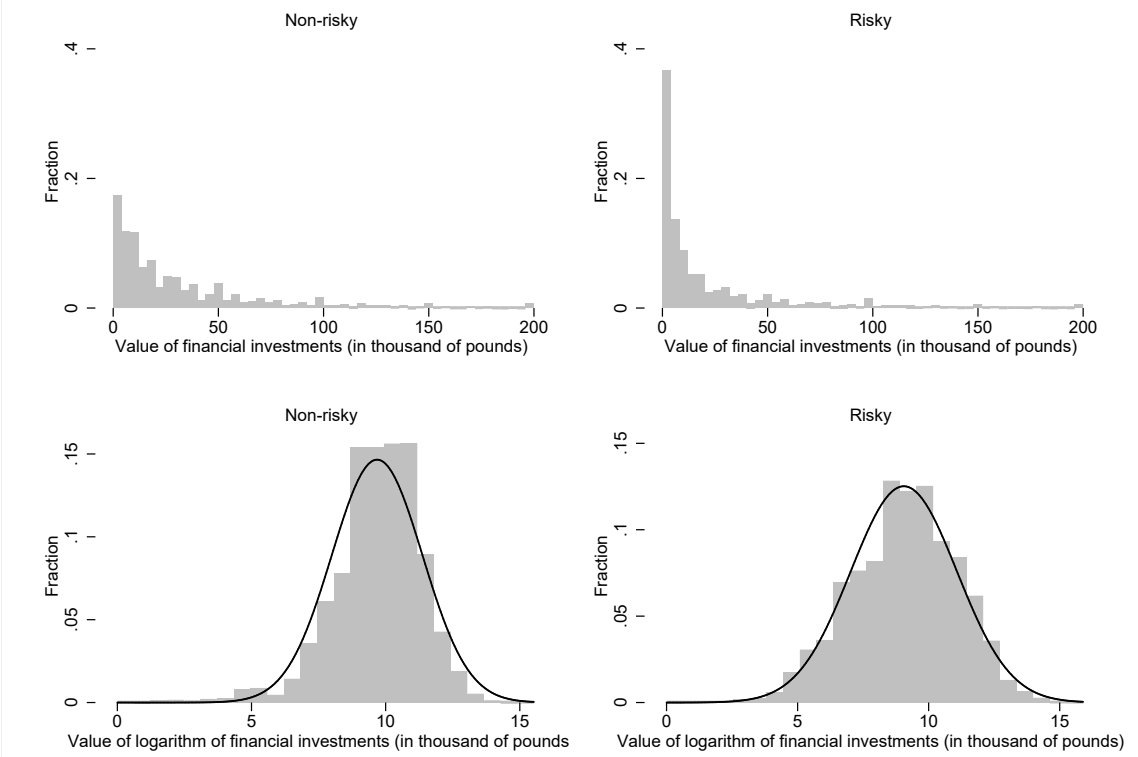
3.2 Outcome variable – Risky and non-risky financial investments

The outcome variables are non-risky and risky financial investments: (1) non-risky financial investments comprise national savings, trusts, bonds, life insurance savings and life insurance ISAs[5]; and (2) risky financial investments are shares and shares ISAs. In each wave, households are asked if they hold some of these savings and investments, and if so, what is their current value.

For each risky and non-risky financial investment the extensive and intensive margins are considered. The extensive margin is captured by a categorical variable measuring whether households hold positive amounts in financially risky or non-risky investments. The intensive margin is captured by a continuous variable that measures the logarithmic value of financially risky and non-risky investments. Figure 1 shows the distribution of these outcome variables[6]. For households with a positive amount in financial investments, the data is positively skewed. A logarithmic transformation is done to normalise the data[7].

[5] Individual savings account (ISA) is a class of retail investment arrangement with favourable tax status.
[6] 31% of households hold non-risky financial investments and 37% of households hold risky investments.
[7] The main results have been replicated excluding outliers with values in risky or non-risky financial investments above 500,000 pounds. The results are highly consistent.

Figure 1: Histogram of financial risky and non-risky investments for total pooled sample



Note: Sample size non-risky financial investments 15,308 observations and risky financial investments 18,724 observations. The proportion of individuals holding non-risky financial investments is 0.31. The proportion of individuals holding non-risky financial investments is 0.37. Outliers with investments value over 300,000 pounds excluded for graphical purposes. In the value of financial investments, 1,831 observations were excluded for non-risky investments and 2,139 observations excluded for risky investments. Normal distribution fitted. *Non-risky:* National savings, trusts, bonds, life insurance savings, life insurance ISAs. *Risky:* Shares and shares ISAs. *Source*: ELSA

Financial investments are measured at a household level. Therefore the analysis is run at the same level. In the ELSA, some households consist of several observations; several members can form a household. The existing literature has applied several methods to decide on which observation to consider, Banks and Oldfield (2007) run their analysis at an individual level, weighting observations at a household level. For robustness, they repeated their analysis using the information of household respondents with the highest cognition levels. There are no significant differences in the results between both methods. Christelis *et al.* (2010) used a mixed method. They considered: the maximum value of the household for cognition, education, probability of leaving an inheritance and social activities; the minimal value of the household for health status; and the average value of the household for age. Kim *et al.* (2012) considered the values of the financial respondent of the survey.

Similarly to Christelis *et al.* (2010), the observations of the individuals in the household with the maximal cognition level are chosen for this essay. This choice is subject to the assumption that the maximal cognition of two respondents is the crucial factor in their decision making. Alternative definitions could

consider the mean cognition in the household, cognition of the financial decision-maker and other definitions. Mean cognition would penalise households with one individual with high cognition who has a partner with low cognition. This choice seems to be an unreasonable assumption, as financial decision making could entirely rely on one of the household members. Regarding the financial decision-maker category, the male respondent could be systematically assigned for cultural reasons, while the couple could decide.

As a robustness check, the results of the primary analysis are repeated for alternative household definitions. These alternative definitions consider the values of the respondents in the household with minimum cognition and mean cognition. It also considers the values of respondents who are the primary financial decision-maker. And it takes into consideration single-person households and male/female households. Table C.2 in the Appendix, does not present significant differences among these definitions for the cross-sectional analysis, while Table C.3 does present significant differences for the longitudinal analysis. These differences suggest that, when considering changes in cognition, the household definition has significant importance. Still, following the arguments of the data section maximum cognition in the household is considered the best measure.

A potential problem posed by these outcome variables is that they are self-reported, and individuals with low cognition levels could systematically report inaccurate information on their financial investments, leading to a cognition bias. The ELSA has information on how respondents answered the financial module, and whether individuals consulted formal documents to answer it. There could be significant differences in the main results driven by individuals consulting formal documents.

Table C.4 in the Appendix presents the main cross-sectional results of this paper for the total sample, for households consulting formal documents, and for households not consulting formal documents. Within households consulting formal documents, it is examined whether there are differences between households consulting some documents and households frequently consulting documents. Regardless of the specifications of these models and its interpretation, the results are very similar for the extensive margin of risky financial investments, but there are significant differences for the intensive margin of risky financial investments. The coefficient of the intensive margin for households not consulting formal documents is much smaller and less significant than for that of the other groups in the intensive margin. These differences suggest that cognition bias affects only the intensive margin. A likely reason is that the cognitive load of remembering any holdings in financial investments should be lower than remembering the value of financial investments.

For this reason, the outcome for the extensive margin considers the entire sample, while for the intensive margin, it is restricted to households using formal documents. Importantly, these outcomes are drawn from different samples, which must be considered when interpreting the results. This cognition bias has not been

considered in the literature. Table C.4 suggests that cognition bias must be considered when working with complex, self-reported financial information[8].

3.3 Sample restriction

Samples size, number of households, and mean number of waves households are present are shown in Table 1. The initial sample consists of 17,236 households, with a mean of 3.3 survey waves and 56,316 observations. The sample is restricted to ages 40-89 and observations with non-missing information in memory and financial investments. There are different sample sizes for the binary outcome of holding or not holding financial investment (extensive margin) and the continuous outcome measuring the pounds amount of investments being restricted to households holding investments (intensive margin). There are 16,998 households and 54,152 observations for the extensive margin, with an average of 3.2 survey waves. Considering the intensive margin, there are 3,925 households and 7,374 observations, with an average of 1.9 survey waves that hold non-risky financial investments, and 4,379 households and 8,676 observations, with an average of 2.0 survey waves that hold risky financial investments[9].

Table 1: Number of observations, number of households and mean number of waves per households for intensive and extensive margin of risky and non-risky financial investments

	NON-RISKY			RISKY		
	Observations	Number households households	Mean waves waves	Observations	Number households households	Mean waves waves
Holds financial investments (0, 1)	54,152	16,998	3.2	54,152	16,998	3.2
Logarithm of financial investments (continuous)	7,374	3,925	1.9	8,676	4,379	2

Note: Sample restricted to individuals aged 40-89, answering the financial and cognitive module. For the intensive margin, the sample is restricted to individuals consulting formal documents during the financial module. *Source*: ELSA

These different samples are driven by the outcomes measuring the intensive margin of financial investments being restricted to households consulting formal documents to answer the financial module. This low number of sample waves per household is largely driven by refresher cohorts having a low number of follow up waves.

3.4 Main explanatory variable – Cognition

The primary variable of interest is cognition. The ELSA includes an extensive battery of psychometric tests that make it possible to measure different cognitive domains of respondents over time. These include

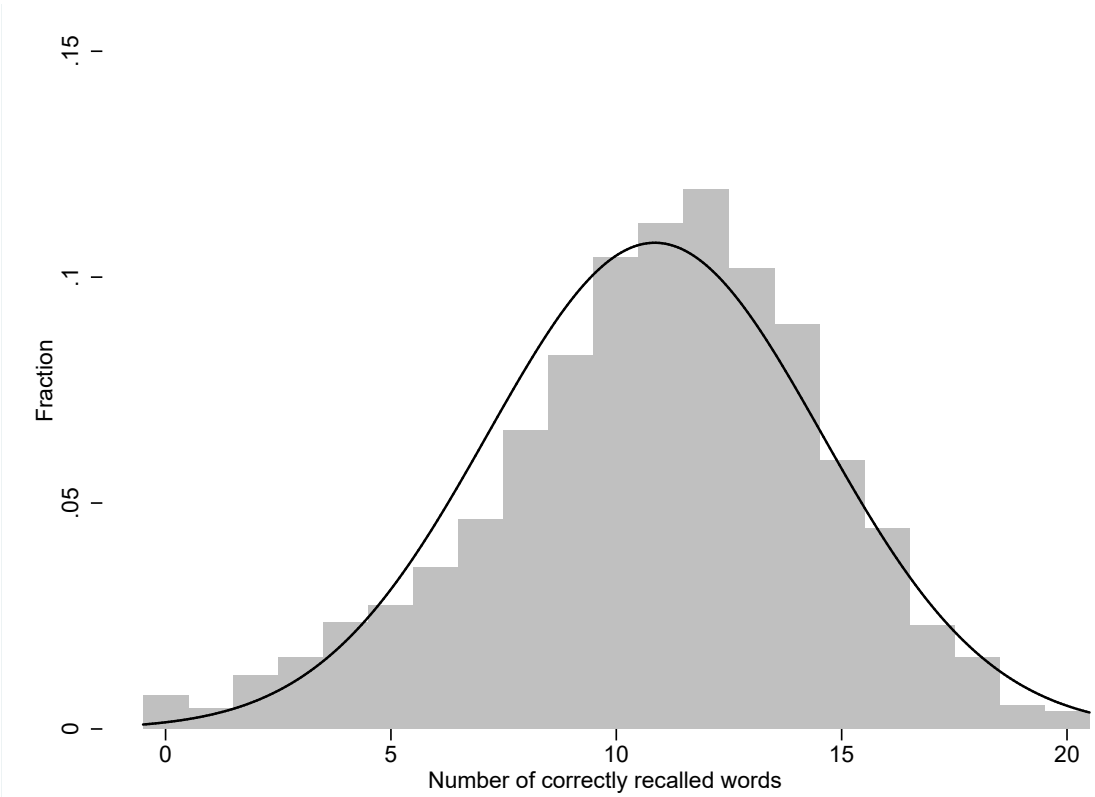
[8] Figure C.1 in the Appendix suggests that there is high stability in the reported outcomes. About 85–90% of the sample reports only one entry or exit into the financial market.

[9] Table C.1 presents descriptive results for the main sample and for the sample with no restrictions. The sample with no restrictions is three years younger and has an average memory score four points higher. If the sample with no restrictions could be considered, the main associations of this essay would probably be more moderate. Nevertheless, this would be driven by the younger age. It would be equivalent to notice that the results are weaker for a sample of younger individuals, which is already shown in Table 5.

memory, time orientation, verbal fluency, and numeracy. Most of these tests are only available for a few waves and are set out in Table B.1 at the Appendix. Only memory and time orientation are available for all waves. Appendix B shows the results of a factor analysis of all available psychometric information from the ELSA. The results show that memory captures the most variability of these cognitive variables. Therefore, only information on memory is used as the primary explanatory variable to use the maximum number of observations and ease the interpretation. Thus, memory can be interpreted as a proxy of general cognition. This is consistent with the literature that has shown memory to be a good predictor of overall cognition (lle Lépine *et al.*, 2005). Time orientation is excluded even if it is available for all waves because it is a very simple measure in the ELSA; it does not add much variability, and it makes the interpretation of the results more challenging.

Memory is measured by an immediate and delayed recall of a list of 10 words, which participants read and memorise. Both scores are added to yield a 20-point score, adding up the number of correctly recalled words, immediate and delayed. This variable is normally distributed with a mean of 10 correctly recalled words and a standard deviation of 3.7. A categorical variable that measures having correctly recalled 0 to 5, 6 to 14, and 15 to 20 words is also constructed; this divides memory into low, medium, and high cognition. The distribution of this variable can be examined in Figure 2.

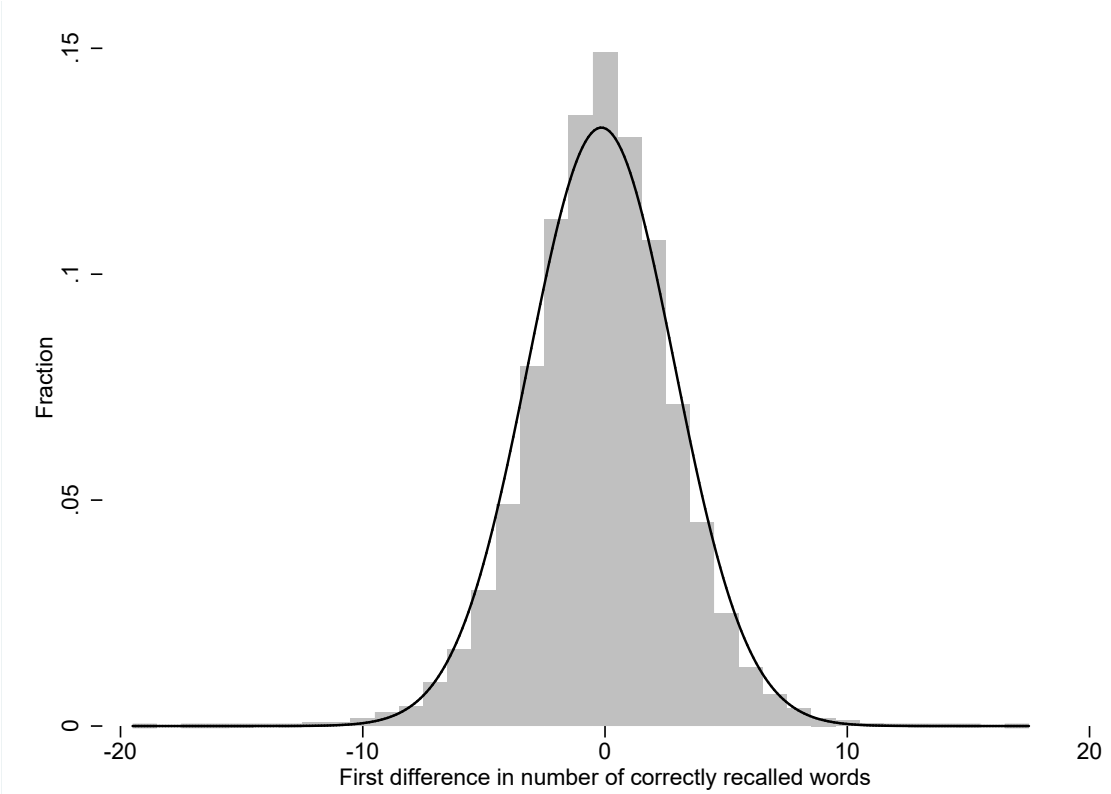
Figure 2: Histogram of memory test score for total pooled sample



Note: Sample size 54,152 observations. *Memory:* Number of correct words recalled, from 10 words immediate and delayed memory test. Score from zero (worse memory) to 20 (best memory). Normal distribution fitted. *Source*: ELSA

The distribution of the first differences can be seen in Figure 3, which shows a normal distribution with a mean of -0.15 and a standard deviation of 3.01.

Figure 3: Histogram of first difference in number of correctly recalled words for total pooled sample



Note: Sample size 37,156 observations. *Memory:* Number of correct words recalled, from 10 words immediate and delayed memory test. Score from zero (worse memory) to 20 (best memory). Normal distribution fitted. *Source*: ELSA

3.5 Control variables

ELSA also includes a comprehensive list of demographic, education, labour, wealth and health controls. These can be seen in Table 2. The basic demographics of the individual with the maximum cognition level are used, because the analysis is run at the household level. The other controls are included at the household level, and the age of the oldest respondent is considered. In other words, the models control for the gender, race and similar demographics of the individual with maximal cognition in the household. They control if any of both members in the household has a specific education level, labour status, or health conditions, while the household's net wealth is considered. Finally, the age of the oldest individual in the household is included.

Table 2: Descriptive statistics control variables

	Observations	Mean	Standard deviation	Min	Max
Age	54,152	67.04	9.88	40	89
Male	54,152	0.38	0.49	0	1
White	54,150	0.97	0.18	0	1
Non-immigrant	54,132	0.92	0.28	0	1
Mar. stat. - Married	54,152	0.55	0.50	0	1
Mar. stat. - Partnered	54,152	0.04	0.19	0	1
Mar. stat. - Separated	54,152	0.01	0.12	0	1
Mar. stat. - Divorced	54,152	0.12	0.32	0	1
Mar. stat. - Widowed	54,152	0.20	0.40	0	1
Mar. stat. - Never married	54,152	0.07	0.25	0	1
Formal documents	51,071	0.45	0.50	0	1
Educ. - Lower high school	54,152	0.41	0.49	0	1
Educ. - High school graduate	54,152	0.21	0.41	0	1
Educ. - Some college	54,152	0.22	0.42	0	1
Educ. - College and above	54,152	0.17	0.38	0	1
Lab. - Employed	54,152	0.26	0.44	0	1
Lab. - Self-employed	54,152	0.06	0.23	0	1
Lab. - Unemployed	54,152	0.01	0.10	0	1
Lab. - Partly retired	54,152	0.01	0.07	0	1
Lab. - Retired	54,152	0.54	0.50	0	1
Lab. - Disabled	54,152	0.05	0.21	0	1
Lab. - Look after family	54,152	0.07	0.26	0	1
Number of children	54,152	2.08	1.53	0	13
Net physical wealth	52,900	53,092.57	442,524.61	0	38,300,000
Net housing wealth	54,107	190,169.84	205,582.27	-1,608,000	4,500,000
High blood pressure	54,148	0.41	0.49	0	1
Diabetes	54,148	0.10	0.29	0	1
Cancer	54,147	0.09	0.28	0	1
Lung disease	54,147	0.06	0.24	0	1
Heart problem	54,148	0.18	0.39	0	1
Arthritis	54,146	0.37	0.48	0	1
Probability of living to age n	53,306	58.07	27.67	0	100

Note: Source: ELSA

These controls can be categorised into five groups: Firstly, demographics (age, gender, race, immigrant, marital status, and the number of children) account for differences in investment patterns and cognition due to gender, age, etc. Age fixed effects are included to control for the close relationship of age and cognition, with which differences in cognition within same-aged households are considered. Secondly, time variables (year and month of the survey) control for the business cycle and similar time-dependent events that could affect financial investments. Thirdly, education level and labour force status account for differences in investment patterns due to educational and occupational variables. Households with higher education or working status levels could be more likely to hold financial investments than lower-educated households and have higher cognition. Fourthly, total net physical and housing wealth consider differences in investments due to wealth levels, which should proxy overall wealth. More wealthy households are more likely to hold financial investments than less wealthy households. Physical and housing wealth are considered the most stable investments. Therefore, they are less likely to be affected by financial investments than other wealth categories. Finally, health controls (high blood pressure, diabetes, cancer, lung problems, heart problems, arthritis, life expectancy of 75, 80, 85, 90 and 95 years) account for differences in investments due to the health status of individuals. Households with worse health outcomes are more likely to disinvest to pay for

health care and prevention. Health conditions causing cognitive deterioration, such as Parkinson's disease and strokes, are excluded.

3.6 Stock market fluctuation - FTSE

The Financial Times Stock Exchange 100 Index (FTSE) is used to measure fluctuations in the stock market. The FTSE is a share index of the 100 companies with the highest market capitalization listed on the London Stock Exchange. The value of the FTSE is measured as the average of the logarithm of the FTSE in the last three months before each survey interview. The logarithm of the FTSE is taken in order to normalise the variable. The average of the last three months is taken owing to the fact households may require at least one financial quarter to react to stock market fluctuations.

4 Methodology

4.1 Identification

Multivariate regression models are estimated through OLS to examine association between financial investment decisions and cognition levels

$$Y_h = \beta_0 + \beta_1 Mem_h + \beta_2 X_h' + \epsilon_h \tag{1}$$

The dependent variable Y_h is either the intensive or extensive margin of risky and non-risky financial investments. For the intensive margin, the logarithm of the dollar amount invested is used. The primary variable of interest is Mem_h, which is the number of correctly recalled words (0-20). X_h' is a vector of control variables for: the individual in the household with the highest level of cognition; and household characteristics. These controls can be categorised into five groups: (1) demographics; (2) survey time variables; (3) education level and labour market status; (4) total net physical and housing wealth; and (5) health.

This model captures differences in the intensive and extensive margin of non-risky and risky financial investments associated with cognition for households with similar characteristics. The coefficient of interest is β_1, which can be interpreted for the extensive margin outcome, as the percentage point difference in the likelihood of having the investment between people with one word difference on the memory test. For the intensive margin, one additional recalled word changes the probability of holding financial investments by β_1*100%.

Further, in an extended specification household fixed effects are included to estimate the effects of cognitive deterioration.

$$Y_{ht} = \alpha_h + \beta_1' Mem_{ht} + \beta_2' Z_{ht}' + \epsilon_{ht} \tag{2}$$

Assuming that there are no further time-varying unobservables correlated with changes in cognition and financial investments, this model captures the effect of changes in cognition level within households on financial investments. The main explanatory variable Mem_h, either measures the number of correctly recalled answers (0–20) as above, or binary indicators of poor cognition (0–5 correct words) and moderate cognition (6–14 correct words). Z_h' is a vector of time changing control variables. It includes age, labour force status, health and health perception variables from vector *X'*.

These models capture the effect of changes in cognition from one wave to another within households on financial investments, controlling for time fixed household characteristics such as baseline cognition and financial investments. Their interpretation for the extensive margin is that remembering one word less from one wave to another changes the probability of holding financial investments in β_1 percentage points. Drops from a high cognition group to a low or medium cognition group from one wave to another change the value of financial investments in β_1 and β_2. For the intensive margin, remembering one word less from one wave to another changes the probability of holding financial investments in β_1*100%. Dropping from a high cognition group to a low or medium cognition group from one wave to another changes the value of financial investments by $\beta_1 * 100\%$ and $\beta_2 * 100\%$.

Further, the following multivariate regression model is estimated, to measure the reaction to stock market fluctuations, in terms of changes in financial investments, due to cognition,

$$Y_h = \beta_0'' + \beta_1'' Mem_h + \beta_2'' X_h' + \beta_3'' ln(FTSE)_h + \beta_4'' Mem_h * ln(FTSE)_h + \beta_5'' W_h' * ln(FTSE)_h + \epsilon_h \tag{3}$$

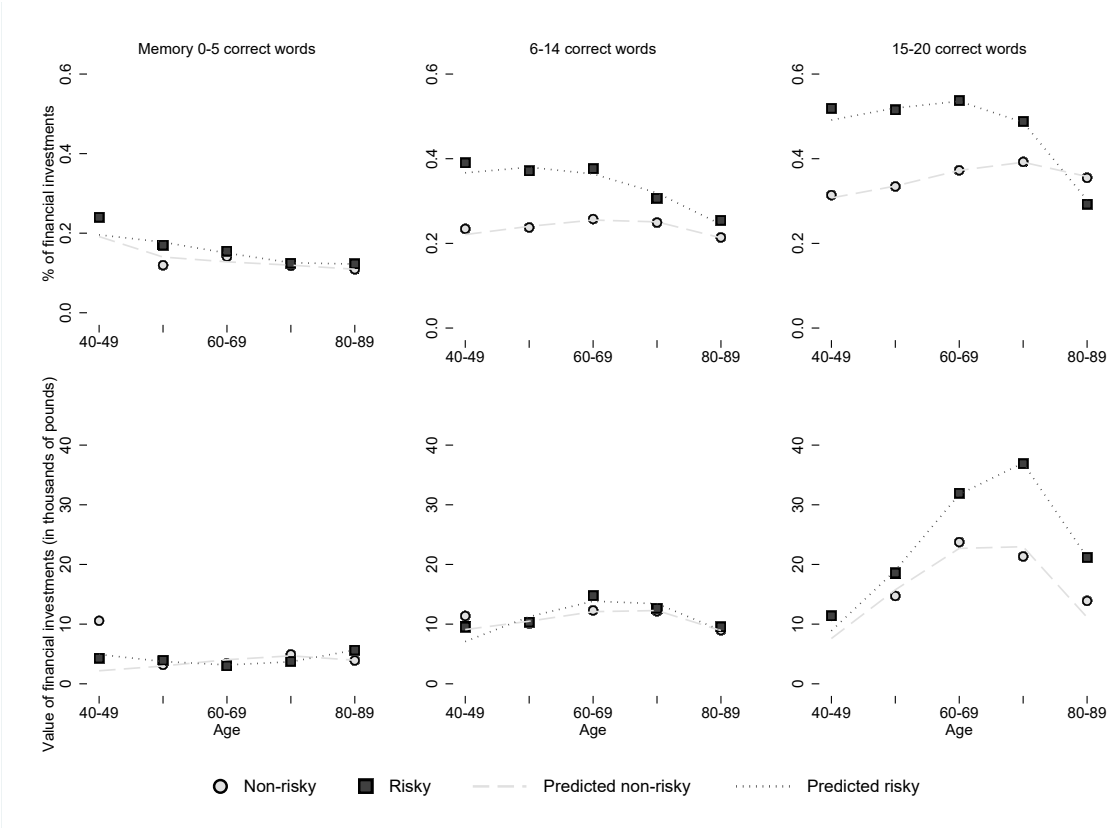
This model expands equation (1) by adding the variable ln(FTSE), in the sense that it adds the variable *ln(FTSE)* and its interaction with cognition and some basic control variables. The value of *ln(FTSE)* is measured as the average of the logarithm of the FTSE in the last three months before the survey interview. W' is a vector of basic control variables, and this is interacted with *ln(FTSE)*. It is composed of age, gender, employment status, and education level. These are variables that could be correlated with the reaction to the stock market returns and cognition.

For the extensive margin, the interpretation of *ln(FTSE)* is that a 1% increase in the FTSE increases the probability of holding risky financial investments by β_3 percentage points. For households recalling zero correct words, this is the main effect of the FTSE value at the months before the interview. For each additional word correctly recalled, this effect increases by β_4 percentage points. For the intensive margin, the interpretation of *ln(FTSE)* is that a 1% increase in the FTSE increases the probability of holding risky financial investments in β_3*100%. For households recalling zero correct words, this is the main effect of fluctuations in the FTSE. For one correctly recalled word, the effect of the FTSE is $\beta_3 * 100$+$\beta_4 * 100$ %. For two correctly recalled words, the effect of the FTSE is $\beta_3 * 100$+2*$\beta_4 * 100$ % and so on.

5 Results

Figure 4 presents descriptive graphs of the average levels of holdings and values of risky and non-risky financial investments by age and cognition level. Overall, there is a tendency in investments to increase up to age 70 and to decrease from thereon. This pattern is most substantial for households with middle and high levels of cognition, suggesting that cognition plays an important role when making decisions regarding financial investments. Higher cognition levels are associated with more financial investments, and this pattern is more substantial for risky investments than non-risky ones. Risky investments also have more variability compared to non-risky ones. These results do not control for any confounders and will be expanded in regression analysis in the following section.

Figure 4: Intensive and extensive margin of risky and non-risky financial investments by age and cognition



Note: Outliers with investments value over 500,000 pounds excluded for graphical purposes. 104 observations were excluded for non-risky investments, and 246 observations were excluded for risky investments. *Non-risky:* National savings, trusts, bonds, life insurance savings, life insurance ISAs. *Risky:* Shares and shares ISAs. *Memory:* Number of correct words recalled, from 10 words immediate and delayed memory test. Score from zero (worse memory) to 20 (best memory). *Source*: ELSA

5.1 Does risky and non-risky investment behaviour differ by cognition levels?

This section explores the association between risky and non-risky financial investments and cognition by estimating a series of regression models with increasingly larger covariate sets. Larger covariate sets will ideally control for a more significant proportion of the non-random variation in levels of cognition. Table 3 shows the estimated regression coefficients on cognition from equation (1).

Table 3: OLS with intensive and extensive margin of financial investments as outcome variables and cognition as main explanatory variable

OUTCOMES	(1)	(2)	(3)	(4)	(5)
Panel A - Holds non-risky financial investments (0, 1)					
Memory	0.019***	0.019***	0.012***	0.010***	0.009***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Panel B - Logarithm of non-risky financial investments (continuous)					
Memory	0.057***	0.049***	0.025***	0.020**	0.020**
	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)
Panel C - Holds risky financial investments (0, 1)					
Memory	0.030***	0.024***	0.014***	0.010***	0.010***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Panel D - Logarithm of risky financial investments (continuous)					
Memory	0.088***	0.091***	0.060***	0.049***	0.047***
	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)
Demographics	✗	✓	✓	✓	✓
Time FE	✗	✓	✓	✓	✓
Education - Labour	✗	✗	✓	✓	✓
Wealth	✗	✗	✗	✓	✓
Health	✗	✗	✗	✗	✓

Note: Standard errors clustered at household level in parentheses, *** p<0.01; ** p<0.05; * p<0.10. *Observations:* Panel A and C 47,790 observations, Panel B 7,374 observations and Panel D 8,676 observations. The average outcome variable for Panel A is 0.32, for Panel B is 9.76, for Panel C is 0.37 and for Panel D is 9.05. *Non-risky:* National savings, trusts, bonds, life insurance savings, life insurance ISAs. *Risky:* Shares and shares ISAs. *Memory:* Number of correct words recalled, from 10 words immediate and delayed memory test. Score from zero (worse memory) to 20 (best memory). *Demographics*: Age fixed effects, gender, race, immigrant. *Education-Labour*: Educational qualification, labour force status. *HHd characteristics*: Number of children, marital status. *Wealth*: Total net physical wealth, total net primary housing wealth. *Health*: High blood pressure, diabetes, cancer, lung problems, heart problems, arthritis, expectations of living until 75, 80, 85, 90 and 95. *Source*: ELSA

Column 1 presents the estimated association from a univariate regression which shows a highly significant positive relationship of cognition with financial investments. Individuals with high cognitive levels hold more risky and non-risky financial investments than individuals with low cognitive levels. One additional word recalled increases the probability of holding non-risky and risky financial investments by 1.9 and three percentage points. Among individuals holding financial investments, one additional word recalled increases non-risky and risky financial investments by 5.7 and 8.8 percent.

In Column 2, the regressions include survey year fixed-effects and control variables representing exogenous demographic characteristics (age, gender, race, immigrant status, marital status, and the number of children). The estimated associations are only slightly changed, with the biggest relative decrease occuring in Panel C: the likelihood of risky investments drops from three percentage points to 2.4 percentage points. However, controlling for educational attainment and labour status greatly affects all the coefficients, reducing them almost by half (Column 3). The substantial reduction in magnitude indicates that educational attainment and labour status are strongly associated with cognition and all financial investments outcomes.

The inclusion of wealth controls in Column (4) further reduces the size of the coefficients. Only physical and housing wealth are included because they are relatively stable and are less likely to be affected by financial investments. Surprisingly, controlling for health controls in Column (5) has only a mild effect on the size of the coefficients.

In all models, the coefficient size diminishes with the inclusion of controls but remains positive and highly significant. Column 5 is the preferred specification controlling for basic demographics, time dummies, education, labour force status, wealth and health[10]. One additional word recalled increases the probability of holding non-risky and risky financial investments by 0.9 (Panel A) and one percentage point (Panel C). Among individuals holding financial investments, one additional word recalled increases non-risky and risky financial investments by two (Panel B) and 4.7 percent (Panel D). Considering that the standard deviation of memory is 3.7, the results can be interpreted as one additional standard deviation in memory, increases the probability of holding risky financial assets by 3.7 percentage points. Relative to the mean, one additional standard deviation in memory increases the probability of holding risky financial assets by 10%. The other financial investments present a similarly strong, positive and significant relationship with cognition. These results are consistent with the findings of the existing literature (McArdle *et al.*, 2009; Kézdi and Willis, 2003; Kim *et al.*, 2012; Cole and Shastry, 2009; Grinblatt *et al.*, 2011; Delavande *et al.*, 2008; Almenberg and Widmark, 2011; Banks and Oldfield, 2007; Christelis *et al.*, 2010).

The results in Table 3 are in line with Figure 4 and confirm the existence of large differences in financial investments due to level of cognition. Figure 4 also shows big differences in investment trends over age for risky and non-risky financial investments. These trends are explored more in the section below, which studies the association between age-related cognitive deterioration and risky and non-risky financial investments[11].

5.2 Does old-age cognitive deterioration affect risky and non-risky investment behaviour?

Previous results demonstrate that the association between cognition and all forms of financial investments is strong and statistically significant. This analysis is extended by estimating household-level fixed-effects regressions, which control for all time-invariant, unobserved determinants of dementia. In these models, changes in cognition within households over time are considered. Results from this exercise are shown in Table 4[12]. The outcome variables are the intensive and extensive margin of risky- and non-risky financial investments. Panel A considers continuous cognition, while Panel B considers categorical cognition. This

[10] Results for the model in Column 5, including coefficients for all control variables, can be found in Table C.5 in the Appendix.

[11] These results have been replicated for the combined risky and non-risky financial investments. The results are highly consistent with the coefficient of the categorical variable being 0.01 and significant at a 99% confidence level, and the coefficient of the continuous variable being 0.02 and significant at a 90% confidence level.

[12] Results including coefficients for all control variables, can be found in Table C.6 in the Appendix.

last variable is divided into low, middle and high cognition groups, with high cognition being the baseline category.

Table 4: Household fixed effects model with intensive and extensive margin of financial investments as outcome variable and cognition as main explanatory variable

VARIABLES	(1) Holds non-risky financial investments (0,1)	(2) Logarithm of non-risky financial investments (continuous)	(3) Holds risky financial investments (0,1)	(4) Logarithm of risky financial investments (continuous)
Panel A				
Memory	0.001	0.003	0.001*	0.015
	(0.001)	(0.011)	(0.001)	(0.010)
Panel B				
Mem. 0-5 correct words	-0.007	0.157	-0.029***	-0.378**
	(0.010)	(0.179)	(0.009)	(0.174)
Mem. 6-14 correct words	-0.009	0.028	-0.013**	-0.057
	(0.007)	(0.061)	(0.006)	(0.058)
Mem. 15-20 correct words				
Baseline category				
Observations	54,152	7,374	54,152	8,676
Number of HHds	16,997	3,922	16,997	4,377
Mean waves HHd	3.186	1.880	3.186	1.982
HHds FE	✓	✓	✓	✓
Baseline covariates	✓	✓	✓	✓
Mean outcome	0.306	9.764	0.369	9.054

Note: Standard errors clustered at household level in parentheses, *** p<0.01; ** p<0.05; * p<0.10. *Non-risky:* National savings, trusts, bonds, life insurance savings, life insurance ISAs. *Risky:* Shares and shares ISAs. *Memory:* Number of correct words recalled, from 10 words immediate and delayed memory test. Score from zero (worse memory) to 20 (best memory). In panel B, the highest cognition is the baseline category (15-20 correct recalled words). *Controls*: Labour force status, total net physical wealth, total net primary housing wealth, high blood pressure, diabetes, cancer, lung problems, heart problems, arthritis, expectations of living until 75, 80, 85, 90 and 95. *Source*: ELSA

Table 4 shows that changes in non-risky financial investments are not significantly associated with changes in cognition within households. This holds for both the extensive and intensive margin of non-risky financial investments. Instead, changes in risky financial investments are strongly and significantly associated with changes in cognition. Panel A shows that recalling one word less from one wave to another reduces the probability of holding risky investments by 0.1 percentage points. This reduction is a small effect and only significant at a 90% confidence level. For the intensive margin, continuous changes in cognition are not significantly related to the value of risky investments. In Panel B, categorical drops in cognition within households are considered. There is a negative and significant association between large drops in cognition with the intensive and extensive margin of risky investments. Suffering a cognition drop from the highest cognition group (15–20 correct words) to the lowest cognition group (0–5 correct words) from one wave to another reduces the probability of holding risky financial investments by 2.9 percentage points at a 99% confidence level and the value of risky financial assets by 38% at a 95% confidence level

The results for risky financial investments are decomposed by age in Table 5. It considers age groups 40 to 64 in Panel A and 65 to 89 in Panel B. The effect of changes in cognition on financial investments is strongest among the age group of 65 to 89.

Table 5: Household fixed effects model with intensive and extensive margin of financial investments as outcome variable and cognition as main explanatory variable

VARIABLES	(1) Holds risky financial investments (0,1)	(2) Logarithm of risky financial investments (continuous)
Panel A - Age 40-64		
Memory	-0.000	-0.003
	(0.001)	(0.016)
Panel B - Age 40-64		
Mem. 0-5 correct words	0.024	-0.306
	(0.017)	(0.497)
Mem. 6-14 correct words	-0.011	-0.049
	(0.008)	(0.079)
Mem. 15-20 correct words - Baseline category		
Panel C - Age 65-89		
Memory	0.002**	0.027*
	(0.001)	(0.014)
Panel D - Age 65-89		
Mem. 0-5 correct words	0.004	-0.403**
	(0.015)	(0.204)
Mem. 6-14 correct words	-0.002	-0.049
	(0.012)	(0.090)
Mem. 15-20 correct words - Baseline category		
HHds FE	✓	✓
Baseline covariates	✓	✓

Note: Standard errors clustered at household level in parentheses, *** p<0.01; ** p<0.05; * p<0.10. Holds risky financial investments (0, 1) Panel A and B 25,791 number of observations and Panel C and D 28,361 number of observations. The logarithm of risky financial investments (continuous) Panel A and B 4,055 observations and Panel C and D 4,621 observations. *Risky investment:* Shares and shares ISAs. *Memory:* Number of correct words recalled, from 10 words immediate and delayed memory test. Score from zero (worse memory) to 20 (best memory). The highest cognition in Panels B and C is the baseline category (15–20 correct recalled words). *Controls*: Labour force status, total net physical wealth, total net primary housing wealth, high blood pressure, diabetes, cancer, lung problems, heart problems, arthritis, expectations of living until 75, 80, 85, 90 and 95. *Source*: ELSA

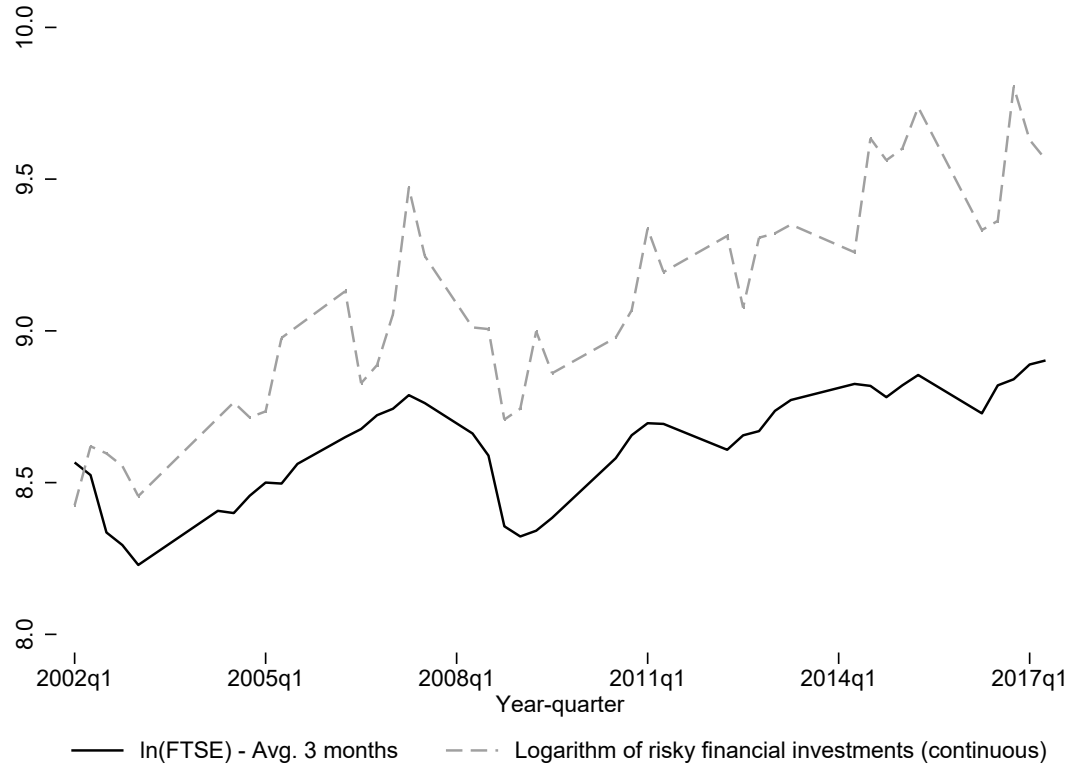
Tables 4, C.3 and 5 are consistent with the descriptive results of Figure 4, showing a stronger age trend for risky financial investments compared to non-risky financial investments over age. This is confirmed in Table 4, which shows that changes in cognition have a significant effect on risky financial investments, but not on non-risky financial investments. Figure 4 also suggests that older households have higher variability in investments compared to younger ones. This is corroborated in Table 5, which finds changes in cognition have significant effects on risky financial investments only for older households.

5.3 Do reactions to fluctuations in the stock market differ by cognition level?

Only risky financial investments are considered for the third research question, as they should be most affected by stock market fluctuations. These stock market fluctuations are measured as the logarithm of the average of the FTSE in the last three months before the survey interview. Figure 5 presents the value of the FTSE and the average of risky financial investments over the period of the ELSA. Both present a parallel

trend and high volatility, suggesting that households were aware of fluctuations in the FTSE when reporting the financial investments.

Figure 5: FTSE return and average ln(Shares) over time



Note: Source: ELSA

Table 6 presents a range of econometric models with the extensive and intensive margin of risky financial investments as outcome variables and cognition level and stock market fluctuations as main variables of interest. These models explore if reactions to stock market fluctuations differ due to the level of cognition of individuals.

Table 6: OLS with extensive and intensive margin of risky financial investments as outcome variables and cognition and five month average of logarithm of FTSE value as main explanatory variable

VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)
	Holds risky financial investments (0,1)			Logarithm of risky financial investments (continuous)		
Memory	0.009***	0.009***	0.009***	0.046***	0.046***	0.044***
	(0.001)	(0.001)	(0.001)	(0.009)	(0.009)	(0.009)
ln(FTSE) - Avg. 3 months (Demeaned)	0.051	0.051	-0.180*	1.011**	1.005**	-2.940**
	(0.043)	(0.043)	(0.105)	(0.471)	(0.475)	(1.290)
ln(FTSE) - Avg. 3 months (Demeaned) * Mem. - (Demeaned)		-0.009***	-0.005		0.006	0.068
		(0.003)	(0.003)		(0.044)	(0.047)
ln(FTSE) - Avg. 3 months (Demeaned) * Age			0.003**			0.054***
			(0.001)			(0.017)
ln(FTSE) - Avg. 3 months (Demeaned) * Gender			0.051**			0.493*
			(0.025)			(0.272)
ln(FTSE) - Avg. 3 months (Demeaned) * Employed			0.000			0.001
			(0.000)			(0.003)
ln(FTSE) - Avg. 3 months (Demeaned) * High education			-0.000*			-0.000
			(0.000)			(0.001)
Observations	54,152	54,152	54,152	8,676	8,676	8,676
Baseline covariates	✓	✓	✓	✓	✓	✓
Mean outcome	0.37	0.37	0.37	9.14	9.14	9.14

Note: Standard errors clustered at household level in parentheses, *** p<0.01; ** p<0.05; * p<0.10. *Risky investment:* Shares and shares ISAs. *Memory:* Number of correct words recalled, from 10 words immediate and delayed memory test. Score from zero (worse memory) to 20 (best memory). *ln(FTSE) - five mth. avg.*: Average of the daily logarithm of FTSE values of the last five months. *Controls*: Age fixed effects, gender, race, immigrant, educational qualification, labour force status, number of children, marital status, total net physical wealth, total net primary housing wealth, high blood pressure, diabetes, cancer, lung problems, heart problems, arthritis, expectations of living until 75, 80, 85, 90 and 95. *Source*: ELSA

Columns 1 and 4 only include the value of the FTSE, cognition and the vector of control variables *X'*. The value of the FTSE before the interview is positively correlated with the extensive and intensive margin of risky financial investments. However, it is only statistically significant for the intensive margin, which seems reasonable since individuals holding risky financial investments should be most aware of changes in stock market prices. Higher values of the FTSE during the three months before the interview increase the reported value of risky financial investments.

Columns 2 and 5 include the interaction of cognition and the value of the FTSE. This interaction is negative and statistically significant for the extensive margin, while for the intensive margin, the interaction value is close to zero and non-significant. The results of the extensive margin suggest that households with higher levels of cognition react less to stock market fluctuations than households with lower levels of cognition. These results are consistent with those of Browning and Finke (2015), who found that individuals with the highest levels of cognition dis-invested less after the GFC. Still, this specification could be capturing the effect of unobserved confounders related to the fluctuations of the FTSE and financial investments.

For this aim, in Columns 3 and 6, age, gender, employment, and education level controls interact with the FTSE's fluctuation. When including these controls, the interaction of cognition level and the FTSE fluctuations becomes insignificant. These results suggest that there are no significant differences in the reaction to the FTSE due to cognition. These results differ from those of Browning and Finke (2015). This divergence in results may have arisen due to consideration of continuous fluctuations in the stock market

and by the examination of the UK scenario. Browning and Finke (2015) also did not include interactions of control variables correlated with cognition, like the ones of Columns 3 and 6.

6 Discussion

This essay explored the association between cognition and cognitive deterioration with risky and non-risky financial investments. Three main findings were made. First, investing in financial assets is strongly associated with higher levels of cognition. Second, cognitive deterioration reduces both the probability of holding risky financial investments and their value. Third, cognition does not moderate households' financial reactions to stock market fluctuations.

These findings are consistent with the existing literature, which shows that a person's cognition level is strongly is strongly and significantly associated with risky and non-risky financial investments (McArdle *et al.*, 2009; Kézdi and Willis, 2003; Kim *et al.*, 2012; Cole and Shastry, 2009; Grinblatt *et al.*, 2011; Delavande *et al.*, 2008; Almenberg and Widmark, 2011; Banks and Oldfield, 2007; Christelis *et al.*, 2010). These results build on the work of Pak and Babiarz (2018), showing that age-related cognitive deterioration reduces the probability of holding risky financial investments while not affecting non-risky financial investments. These results are consistent with those derived from their fixed-effect model (Pak and Babiarz, 2018). The endogeneity problems raised by Pak and Babiarz (2018) are addressed, including detailed labour force status information, health and wealth controls and examination of the UK scenario, where health services access is less dependent on wealth compared to the US. The reaction of households to stock market fluctuations in terms of changes in risky financial investments and their interaction with cognition is also examined. Unlike Browning and Finke (2015), no significant relationship between cognition and re-allocations of risky financial investments due to stock market fluctuations is found.

These results suggest that households reduce risky financial investments when starting to suffer age-dependent cognitive deterioration because cognitive deterioration makes individuals more risk-averse and increases participation costs in the financial investment market. This interpretation is based on two assumptions extracted from empirical conclusions of the existing literature (Banks and Oldfield, 2007; Christelis *et al.*, 2010; Kézdi and Willis, 2003; McArdle *et al.*, 2009; Pak and Babiarz, 2018). Firstly, households with lower cognition levels are more likely to be risk-averse than households with higher cognition levels. This leads to differences in utility functions of households due to cognition. Secondly, households composed of individuals with lower cognition levels face a higher participation cost in the financial investments market compared to households composed of individuals with higher cognition levels.

Even if it is rational to reduce risky financial investments when facing cognitive deterioration, this is not necessarily optimal. Arrow (1965) and others suggested that it is optimal to maintain some investments

in risky financial investments. Therefore, reductions in risky financial investments could lead to a sub-optimal wealth outcome reducing households' wealth. Suboptimal investments are particularly worrying when considering households composed of older individuals whose wealth depends on investments, which is compounded by their increased likelihood of suffering cognitive deterioration. It is not only worrying from an individual perspective but also from a societal one. There could be a high opportunity cost of unnecessarily dis-investing risky financial investments at old age caused by natural cognitive deterioration.

Kim *et al.* (2019) show that individuals with higher levels of cognition are more likely to take professional financial advice than individuals with lower cognition levels. Conversely, individuals with lower cognition levels are more likely to take financial advice from family members or "free" financial advice, which often entails conflicts of interest and can lead them to dis-invest sub-optimally. Thus, access to financial information could be one of the potential mechanisms in this essay. When individuals in this sample start suffering cognitive deterioration, their access to financial advice could deteriorate, leading them to make sub-optimal financial investments. A potential solution to this problem is to provide a specialised assessment to households composed of older individuals for managing their financial investments. Particularly, this could be encouraged by training bank tellers to recognise signs of age-related cognitive decline (Gunther, 2015). This way individuals who suffer age related cognitive decline could be targeted to receive financial assessment. This could be beneficial for such individuals (Kim *et al.*, 2016), reduce the cost of managing financial investments, and re-balance risky and non-risky financial investments.

References

AARTSEN, M. J., SMITS, C. H., VAN TILBURG, T., KNIPSCHEER, K. C. and DEEG, D. J. (2002). Activity in older adults: cause or consequence of cognitive functioning? A longitudinal study on everyday activities and cognitive performance in older adults. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, **57** (2), P153–P162.

AGE UK (2019). Later life in the United Kingdom 2018.

ALMENBERG, J. and WIDMARK, O. (2011). Numeracy, financial literacy and participation in asset markets. *Available at SSRN 1756674*.

ALZHEIMER'S SOCIETY (2019). Alzheimer's Society's view on demography. https://www.alzheimers.org.uk/about-us/policy-and-influencing/what-we-think/demography, accessed: 2020-03-09.

AMISHI, G. and HAN, Y. (2019). *Investing in (and for) Our Future*. Tech. rep., World Economic Forum.

ARROW, K. J. (1965). Aspects of the theory of risk-bearing (yrjo jahnsson lectures). *Yrjo Jahnssonin Saatio, Helsinki*.

BANKS, J. and OLDFIELD, Z. (2007). Understanding pensions: Cognitive function, numerical ability and retirement saving. *Fiscal Studies*, **28** (2), 143–170.

BEAUCHAMP, J. P., CESARINI, D. and JOHANNESSON, M. (2017). The psychometric and empirical properties of measures of risk preferences. *Journal of Risk and Uncertainty*, **54** (3), 203–237.

BOGAN, V. (2008). Stock market participation and the internet. *Journal of Financial and Quantitative Analysis*, **43** (1), 191–211.

BOGAN, V. L. and FERTIG, A. R. (2013). Portfolio choice and mental health. *Review of Finance*, **17** (3), 955–992.

BONSANG, E. and DOHMEN, T. (2015). Risk attitude and cognitive aging. *Journal of Economic Behavior & Organization*, **112**, 112–126.

BROWNING, C. and FINKE, M. (2015). Cognitive ability and the stock reallocations of retirees during the Great Recession. *Journal of Consumer Affairs*, **49** (2), 356–375.

CHRISTELIS, D., JAPPELLI, T. and PADULA, M. (2010). Cognitive abilities and portfolio choice. *European Economic Review*, **54** (1), 18–38.

COLE, S. A. and SHASTRY, G. K. (2009). *Smart money: The effect of education, cognitive ability, and financial literacy on financial market participation*. Harvard Business School Boston, MA.

CRIMMINS, E. M., KIM, J. K., LANGA, K. M. and WEIR, D. R. (2011). Assessment of cognition using surveys and neuropsychological assessment: the Health and Retirement Study and the Aging, Demographics, and Memory Study. *Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, **66** (suppl_1), i162–i171.

DELAVANDE, A., ROHWEDDER, S. and WILLIS, R. J. (2008). Preparation for retirement, financial literacy and cognitive resources. *Michigan Retirement Research Center Research Paper*, (2008-190).

DOHMEN, T., FALK, A., GOLSTEYN, B. H., HUFFMAN, D. and SUNDE, U. (2017). Risk attitudes across the life course.

—, —, HUFFMAN, D. and SUNDE, U. (2010). Are risk aversion and impatience related to cognitive ability? *American Economic Review*, **100** (3), 1238–60.

—, —, — and — (2018). On the relationship between cognitive ability and risk preference. *Journal of Economic Perspectives*, **32** (2), 115–34.

—, —, —, —, SCHUPP, J. and WAGNER, G. G. (2011). Individual risk attitudes: Measurement, determinants, and behavioral consequences. *Journal of the European Economic Association*, **9** (3), 522–550.

GRINBLATT, M., KELOHARJU, M. and LINNAINMAA, J. (2011). IQ and stock market participation. *The Journal of Finance*, **66** (6), 2121–2164.

GUNTHER, J. (2015). Banksafe safeguards against exploitation. https://www.aarp.org/ppi/info-2015/age-friendly-banking-an-initiative-promoting-the-financialresilience-of-people-age-50.html, accessed: 2022-01-01.

KENT, S. T., MCCLURE, L. A., CROSSON, W. L., ARNETT, D. K., WADLEY, V. G. and SATHIAKUMAR, N. (2009). Effect of sunlight exposure on cognitive function among depressed and non-depressed participants: a REGARDS cross-sectional study. *Environmental Health*, **8** (1), 34.

KÉZDI, G. and WILLIS, R. J. (2003). Who becomes a stockholder? expectations, subjective uncertainty, and asset allocation. *Expectations, Subjective Uncertainty, and Asset Allocation (April 2003). Michigan Retirement Research Center Research Paper No. WP*, **39**.

KIM, E. J., HANNA, S. D., CHATTERJEE, S. and LINDAMOOD, S. (2012). Who among the elderly owns stocks? The role of cognitive ability and bequest motive. *Journal of Family and Economic Issues*, **33** (3), 338–352.

KIM, H. H., MAURER, R. and MITCHELL, O. S. (2016). Time is money: Rational life cycle inertia and the delegation of investment management. *Journal of Financial Economics*, **121** (2), 427–447.

—, — and — (2019). *How Cognitive Ability and Financial Literacy Shape the Demand for Financial Advice at Older Ages*. Tech. rep., National Bureau of Economic Research.

KOSCIELNIAK, M., RYDZEWSKA, K. and SEDEK, G. (2016). Effects of age and initial risk perception on balloon analog risk task: The mediating role of processing speed and need for cognitive closure. *Frontiers in psychology*, **7**, 659.

LILLEHOLT, L. (2019). Cognitive ability and risk aversion: A systematic review and meta analysis. *Judgment and Decision Making*.

LLE LÉPINE, R., PARROUILLET, P. and CAMOS, V. (2005). What makes working memory spans so predictive of high-level cognition? *Psychonomic Bulletin & Review*, **12** (1), 165–170.

MATA, R., JOSEF, A. K. and HERTWIG, R. (2016). Propensity for risk taking across the life span and around the globe. *Psychological Science*, **27** (2), 231–243.

MAZZONNA, F., PERACCHI, F. *et al.* (2018). *Self-assessed cognitive ability and financial wealth: Are people aware of their cognitive decline?* Tech. rep., Einaudi Institute for Economics and Finance (EIEF).

MCARDLE, J. J., SMITH, J. P. and WILLIS, R. (2009). *Cognition and economic outcomes in the Health and Retirement Survey*. Tech. rep., National Bureau of Economic Research.

PAK, T.-Y. and BABIARZ, P. (2018). Does cognitive aging affect portfolio choice? *Journal of Economic Psychology*, **66**, 1–12.

SCHNEIDER, E. C., SARNAK, D. O., DAVID, S., ARNAV, S. and MICHELLE, M. D. (2017). Mirror, mirror 2017: International comparison reflects flaws and opportunities for better u.s. health care. https://www.commonwealthfund.org/publications/fund-reports/2017/jul/mirror-mirror-2017-international-comparison-reflects-flaws-and, accessed: 2020-08-09.

TYMULA, A., BELMAKER, L. A. R., RUDERMAN, L., GLIMCHER, P. W. and LEVY, I. (2013). Like cognitive function, decision making across the life span shows profound age-related changes. *Proceedings of the National Academy of Sciences*, **110** (42), 17143–17148.

Appendices

A Literature review

Table A.1: Literature three research questions

Paper	Objective	Data	Methodology	Conclusions
Research question 1 - Do risky and non-risky financial investments differ due to cognition?				
McArdle et al. (2009)	Examine the association of cognitive skills with financial wealth and the fraction of financial wealth held in stocks	Heatlh and Retirement Study (HRS), US, panel (2000 & 2006), N=18,382 & 14,270; Cognitive Economics Survey (CogEcon), US, cross sectional	OLS (for HRS 2000 wave is only used to gernerate first differences in the outcome variable)	Cognition is positivelly and significantly associated with financial wealth and the fraction of financial wealth held in stocks
Kézdi et al. (2003)	Examine determinants of stock holdings. Consider cognition amongst others cognition	Heatlh and Retirement Study (HRS), US, panel (1992-2000), N=12,670	Pooled OLS	Stock ownership and the probability of becoming a stockholder are strongly positively associated with subjective measures of preferences and expectations and cognition
Kim et al. (2012)	Examines factors related to stockholdings, such as cognition	Heatlh and Retirement Study (HRS), US, cross sectional (2004), N=5,763	Logit	Cognition is positivelly and significantly associated with stockholdings
Cole et al. (2009)	Examine the association between cognition and stockholding and amount hold in stocks	National Longitudinal Survey of Youth (NLSY79), US, panel (1979-1994, in 1980 respondents take the Armed Services Vocational Aptitude	Sibling groups fixed effects OLS	Cognition is positivielly and statistically significantly associated with holding stocks and amount held in stocks
Grinblat et al. (2011)	Examine the association between IQ and stockholding	Finnish Central Securities Depository (FCSD) Registry, Finland, cross sectional (2000); Finnish Armed Forces (FAF) Intelligence Assessment, Finland, cross sectional (1998-2001); Supplementary databases: Finnish Tax	Probit and random effects probit	Stock market participation is monotonically related to IQ
Almenberg et al. (2011)	Examine the association of numeracy with assets market participation	Own source telephone survey, Sweden, cross sectional (2010), N=1,300	Probit	Numeracy is positivelly and significantly associated with assets market participation
Christelis et al. (2010)	Examine the association between cognition and stockholding	The survey of Health, Ageing and Retirement in Europe (SHARE), EU, cross sectional	Probit	Cognition is positivelly and significantly associated with stockholdings
Banks et al. (2007)	Examine the association between numeracy and retirment savings and investments, consider sharesholdings	English Longitudinal Study of Ageing (ELSA), UK, cross sectional (2002), N=11,392	Weighted quantile regression and probit	Numeracy is positivelly and significantly associated with shareholdings
Research question 2 - Does old age cognitive deterioration affect risky and non-risky financial investments?				
Pak et al. (2018)	Examine cognitive decline and risky financial investments and its causal path	Heatlh and Retirement Study (HRS), US, panel (1998-2012), N=15,597	Individual fixed effects OLS and IV regression with seasonal variation as	Cognitive ageing is negatively and significantly associated with share of stocks
Research question 3 - Do reactions to stock market fluctuations in terms of changes in risky and non-risky financial investments differ				
Browning et al. (2015)	Examine if cognitive ability is related to stock reallocations among retirees during the Great Recession	Heatlh and Retirement Study (HRS), US, panel (2006-2008), N=1,204	OLS, logit	Cognition predicts reallcoation toward safe assets. Individuals with higher cognition reallocate less with respect to individuals with lower cognition

Note: Own source

B Cognition

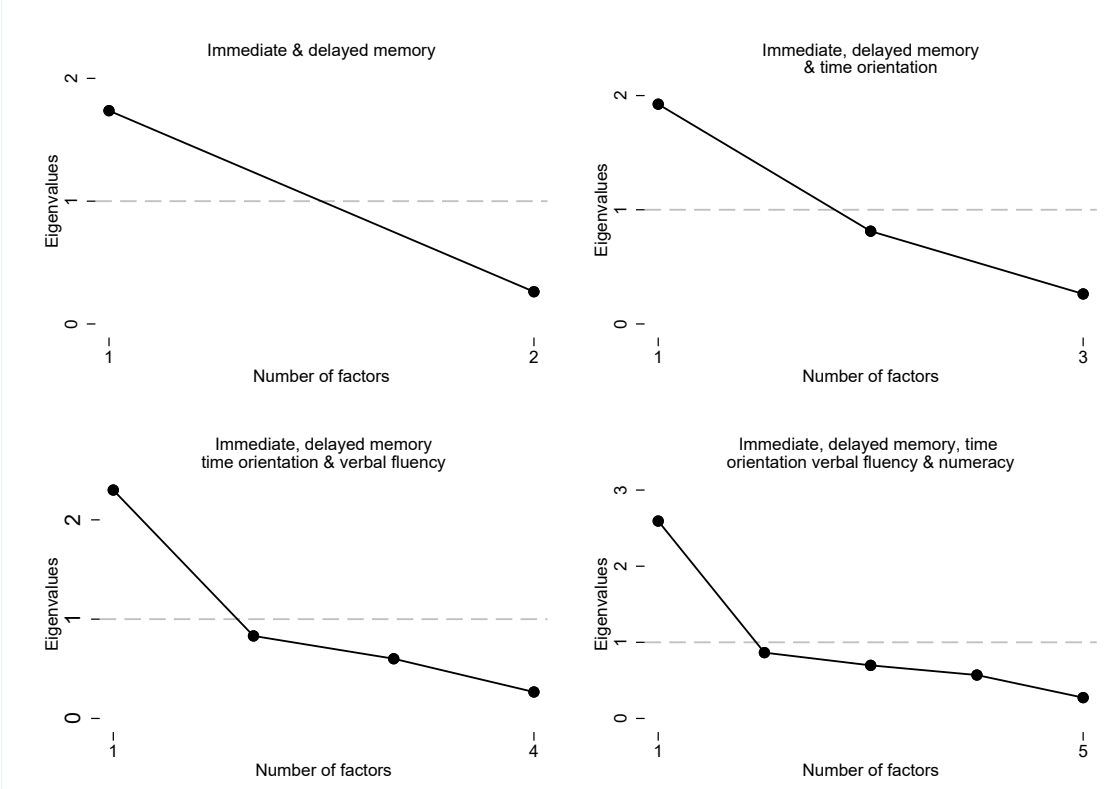
The cognition variables available at most waves of ELSA are memory, time orientation, verbal fluency and numeracy. In a similar manner to Bonsang and Dohmen 2015, a factor analysis is run over different combinations of these variables. For the different combinations, one factor is always kept, considering only factors with Eigenvalues above 1. Eigenvalues can are shown in Figure B.1. These different factors are highly correlated, as can be seen in Table B.2. In order to keep with the maximum number of observations and ease the interpretation of the results, memory is our main explanatory variable.

Table B.1: Cognition variables ELSA by wave

	W1	W2	W3	W4	W5	W6	W7	W8
Verbal Fluency	11713	9252	9480	10541	9662		9057	7972
Memory	11701	9234	9483	10544	9688	9971	9040	7921
Date	12099	9432	9771	11050	10274	10601	9666	8445
Health Literacy		8888			9281	1489	548	149
Numeracy	10064			9409		1320	499	139

Note: Source: ELSA

Figure B.1: Screeplot for different cognition variables



Note: Source: ELSA

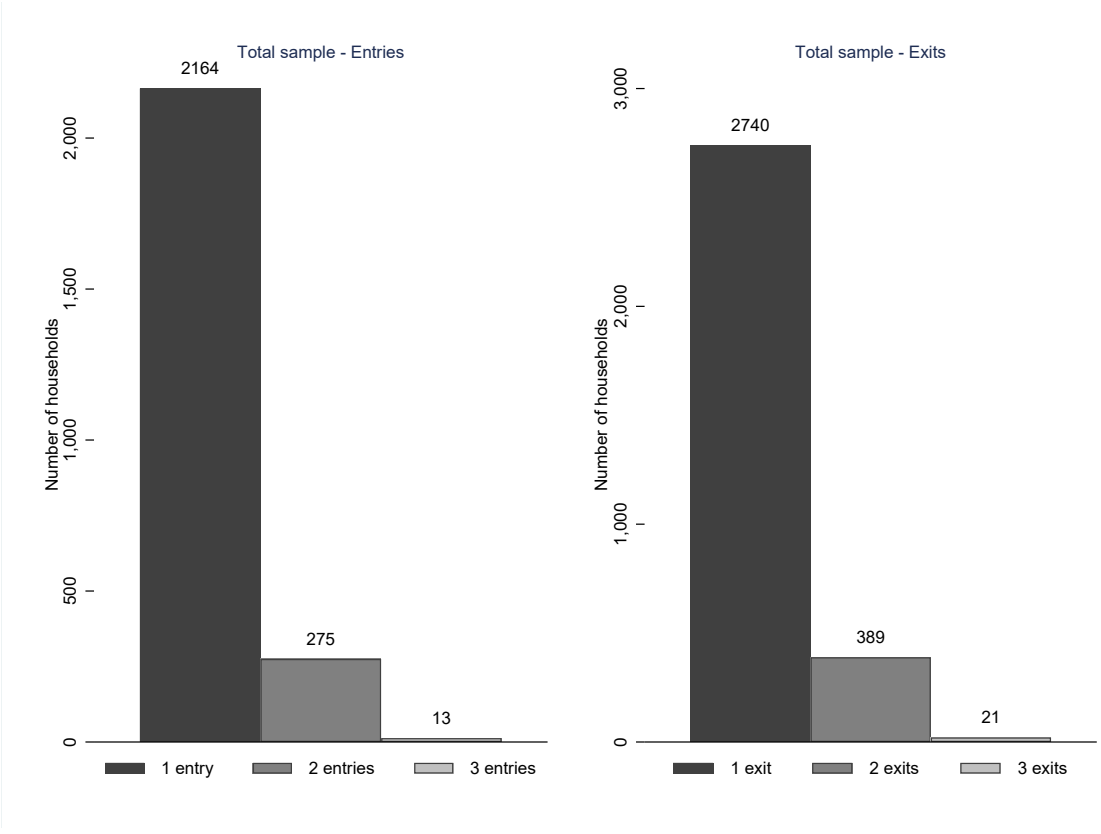
Table B.2: OLS with intensive and extensive margin of financial investments as outcome variables and cognition as main explanatory variable

	Memory	+Time orientation	+Verbal fluency	+Numeracy
Memory	1			
+Time orientation	0.976***	1		
+Verbal fluency	0.950***	0.963***	1	
+Numeracy	0.852***	0.857***	0.901***	1

Note: Memory: Immediate and delayed memory. *+Time orientation:* Immediate and delayed memory and time orientation (respondent reports current date). *+Verbal fluency:* Immediate and delayed memory, time orientation and verbal fluency (respondent names as many animal names as he can in one minute). *+Verbal fluency:* Immediate and delayed memory, time orientation, verbal fluency and numeracy (respondent answers mathematical questions). *Source*: ELSA

C Extended results

Figure C.1: Number of entries and exits into the financial investments market by household



Note: Source: ELSA

Table C.1: Descriptive statistics largest possible sample

	(1) Main sample		(2) Sample – largest possible sample	
	mean	sd	mean	sd
Age	71.25	9.72	67.37	9.93
Memory	6.24	4.19	10.77	3.77
Male	0.36	0.22	0.38	0.47
Non-white	0.97	0.05	0.97	0.17
Non-immigrant	0.92	0.10	0.92	0.27

Note: Memory: Number of correct words recalled, from 10 words immediate and delayed memory test. Score from zero (worse memory) to 20 (best memory). *Source*: ELSA

Table C.2: OLS with intensive and extensive margin of financial investments as outcome variables and cognition as main explanatory variable

OUTCOMES	(1) Max Mem. in HHd	(2) Min Mem. in HHd	(3) Mean Mem. in HHd	(4) Financial decision maker Mem.	(5) Male Mem.	(6) Female Mem.	(7) Single HHd. Mem. (no couples)
Panel A - Holds non-risky financial investments (0, 1)							
Memory	0.010***	0.009***	0.011***	0.008***	0.008***	0.010***	0.009***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Observations	54,152	54,152	54,152	53,936	32,915	33,624	25,155
Panel B - Logarithm of non-risky financial investments (continuous)							
Memory	0.025***	0.025***	0.029***	0.019***	0.035***	0.021**	0.019
	(0.007)	(0.007)	(0.008)	(0.006)	(0.009)	(0.010)	(0.012)
Observations	7,374	7,374	7,374	7,374	5,025	4,555	2,549
Panel A - Holds risky financial investments (0, 1)							
Memory	0.010***	0.009***	0.011***	0.008***	0.010***	0.009***	0.009***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Observations	54,152	54,152	54,152	53,936	32,915	33,624	25,155
Panel B - Logarithm of risky financial investments (continuous)							
Memory	0.047***	0.041***	0.051***	0.031***	0.052***	0.048***	0.047***
	(0.009)	(0.008)	(0.009)	(0.008)	(0.011)	(0.011)	(0.015)
Observations	8,676	8,676	8,676	8,676	6,096	5,216	2,816
Baseline covariates	✓	✓	✓	✓	✓	✓	✓

Note: Standard errors clustered at household level in parentheses, *** p<0.01; ** p<0.05; * p<0.10. *Observations:* Panel A and B 47,790 observations and panel C and D 8,580 observations. *Risky investment:* Shares and shares ISAs. *Memory:* Number of correct words recalled, from 10 words immediate and delayed memory test. Score from zero (worse memory) to 20 (best memory). *Max Mem.:* Memory considered of the household member with highest cognition level. *Min Mem.:* Memory considered of the household member with lowest cognition level. *Mean Mem.:* Mean cognition of the household considered. *Fin. dec. maker Mem.:* Memory considered of the household financial decision-maker. *Male:* Memory considered of the male household member. *Female:* Memory considered of the female household member. *Single HHd Mem.:* Only households with one member considered. *Controls*: Age fixed effects, gender, race, immigrant, formal documents, educational qualification, labour force status, number of children, marital status, total net physical wealth, total net primary housing wealth, high blood pressure, diabetes, cancer, lung problems, heart problems, arthritis, expectations of living until 75, 80, 85, 90 and 95. *Source*: ELSA

Table C.3: Household fixed effects OLS with intensive and extensive margin of financial investments as outcome variables and cognition as main explanatory variable

OUTCOMES	(1) Max Mem. in HHd	(2) Min Mem. in HHd	(3) Mean Mem. in HHd	(4) Financial decision maker Mem.	(5) Male Mem.	(6) Female Mem.	(7) Single HHd. Mem. (no couples)
Panel A - Holds risky financial investments (0, 1)							
Memory	0.001	-0.001	0.000	0.000	0.000	0.001	0.000
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Panel B - Holds risky financial investments (0, 1)							
Mem. 0-5 correct words	-0.025***	-0.003	-0.012	-0.017*	-0.017	-0.025**	-0.016
	(0.009)	(0.009)	(0.009)	(0.009)	(0.013)	(0.011)	(0.012)
Mem. 6-14 correct words	-0.012**	0.006	-0.001	-0.005	-0.012	-0.007	-0.002
	(0.006)	(0.007)	(0.007)	(0.006)	(0.009)	(0.007)	(0.009)
Mem. 15-20 correct words - Baseline category							
HHds FE	✓	✓	✓	✓	✓	✓	✓
Baseline covariates	✓	✓	✓	✓	✓	✓	✓
Observations	54,152	54,152	54,152	53,936	32,915	33,624	25,155
Number of HHds	16,998	16,998	16,998	16,951	10,529	11,173	7,165
Mean outcome	0.37	0.37	0.37	0.37	0.41	0.35	0.28

Note: Standard errors clustered at household level in parentheses, *** p<0.01; ** p<0.05; * p<0.10. *Non-risky:* National savings, trusts, bonds, life insurance savings, life insurance ISAs. *Risky:* Shares and shares ISAs. *Memory:* Number of correct words recalled, from 10 words immediate and delayed memory test. Score from zero (worse memory) to 20 (best memory). In panel B, the highest cognition is the baseline category (15–20 correct recalled words). *Controls*: Labour force status, total net physical wealth, total net primary housing wealth, high blood pressure, diabetes, cancer, lung problems, heart problems, arthritis, expectations of living until 75, 80, 85, 90 and 95. *Source*: ELSA

Table C.4: OLS intensive and extensive margin of risky financial investments as outcome variables and cognition as main explanatory variable for individuals using and not using formal documents to answer the financial module

	(1)	(2)	(3)
OUTCOMES	Total	Doc.	No doc.
Panel A - Holds non-risky financial investments (0, 1)			
Memory	0.009***	0.009***	0.009***
	(0.001)	(0.001)	(0.001)
Observations	**54,152**	22,786	28,285
Panel B - Logarithm of non-risky financial investments (continuous)			
Memory	0.011*	0.020**	-0.000
	(0.006)	(0.008)	(0.008)
Observations	13,298	**6,448**	6,555
Panel C - Holds risky financial investments (0, 1)			
Memory	0.010***	0.010***	0.010***
	(0.001)	(0.001)	(0.001)
Observations	**54,152**	22,786	28,285
Panel D - Logarithm of risky financial investments (continuous)			
Memory	0.029***	0.047***	0.012
	(0.006)	(0.008)	(0.008)
Observations	18,724	**8,676**	9,666
Baseline covariates	✓	✓	✓

Note: Standard errors clustered at household level in parentheses, *** p<0.01; ** p<0.05; * p<0.10. *Risky investment:* Shares and shares ISAs. *Total:* Total sample. *Doc.:* Sample that consulted documents to answer questions in the financial module. *No doc.:* Sample that did no consult documents to answer questions in the financial module. *Some doc.:* Sample that consulted documents to answer questions in the financial module. Not frequently. *Frequently doc.:* Sample that frequently consulted documents to answer questions in the financial module. *Memory:* Number of correct words recalled, from 10 words immediate and delayed memory test. Score from zero (worse memory) to 20 (best memory). *Controls*: Age fixed effects, gender, race, immigrant, educational qualification, labour force status, number of children, marital status, total net physical wealth, total net primary housing wealth, high blood pressure, diabetes, cancer, lung problems, heart problems, arthritis, expectations of living until 75, 80, 85, 90 and 95. *Source*: ELSA

Table C.5: OLS with intensive and extensive margin of financial investments as outcome variables and cognition as main explanatory variable

VARIABLES	(1) Holds non-risky financial investments (0 1)	(2) Logarithm of non-risky financial investments (continuous)	(3) Holds risky financial investments (0 1)	(4) Logarithm of risky financial investments (continuous)
Memory	0.009***	0.020**	0.010***	0.047***
	(0.001)	(0.008)	(0.001)	(0.009)
Male	0.012**	0.086*	0.031***	0.185***
	(0.005)	(0.052)	(0.006)	(0.056)
Non-white	0.025*	0.115	0.053***	0.146
	(0.015)	(0.255)	(0.018)	(0.245)
Non-immigrant	0.052***	0.106	0.040***	-0.178
	(0.010)	(0.111)	(0.012)	(0.128)
Mar. stat. - Married	0.127***	0.085	-0.064	-0.172
	(0.038)	(0.163)	(0.095)	(0.171)
Mar. stat. - Partnered	0.159***	-0.030	-0.041	-0.147
	(0.040)	(0.198)	(0.096)	(0.207)
Mar. stat. - Separated	0.135***	-0.157	-0.113	-0.041
	(0.041)	(0.336)	(0.097)	(0.300)
Mar. stat. - Divorced	0.117***	-0.224	-0.130	-0.506***
	(0.039)	(0.154)	(0.095)	(0.169)
Mar. stat. - Widowed	0.161***	-0.142	-0.072	-0.289**
	(0.039)	(0.135)	(0.095)	(0.146)
Mar. stat. - Never married	0.176***		-0.082	
	(0.040)		(0.096)	
Number of children	-0.014***	-0.061***	-0.015***	-0.002
	(0.002)	(0.022)	(0.002)	(0.022)
Non-single Household	0.077***	-0.062	0.058***	-0.169
	(0.010)	(0.114)	(0.012)	(0.124)
Educ. - Lower high school	-0.068***	-0.207***	-0.083***	-0.235***
	(0.007)	(0.071)	(0.008)	(0.079)
Educ. - High school graduate	-0.000	-0.015	0.020**	-0.006
	(0.008)	(0.069)	(0.009)	(0.077)
Educ. - Some college	0.029***	0.051	0.054***	0.071
	(0.008)	(0.062)	(0.009)	(0.075)
Educ. - College and above	0.095***	0.325***	0.102***	0.406***
	(0.009)	(0.068)	(0.011)	(0.079)
Lab. - Employed	-0.055*	-0.328	0.029	0.107
	(0.031)	(0.448)	(0.032)	(0.352)
Lab. - Self-employed	-0.073**	-0.397	0.019	0.013
	(0.032)	(0.463)	(0.034)	(0.368)
Lab. - Unemployed	-0.092***	-0.480	-0.092***	-0.379
	(0.033)	(0.569)	(0.035)	(0.474)
Lab. - Partly retired	0.031	-0.423	0.050	0.626
	(0.041)	(0.510)	(0.042)	(0.489)
Lab. - Retired	0.017	-0.005	0.046	0.526
	(0.031)	(0.446)	(0.032)	(0.351)
Lab. - Disabled	-0.075**	-0.659	-0.090***	0.178
	(0.032)	(0.503)	(0.032)	(0.395)
Lab. - Look after family	-0.013	-0.100	0.018	0.546
	(0.031)	(0.455)	(0.032)	(0.362)
Net physical wealth	-0.000	0.000*	0.000***	0.000***
	(0.000)	(0.000)	(0.000)	(0.000)
Net housing wealth	0.000***	0.000***	0.000***	0.000***
	(0.000)	(0.000)	(0.000)	(0.000)
High blood pressure	-0.012**	-0.033	-0.001	-0.106*
	(0.005)	(0.053)	(0.006)	(0.058)
Diabetes	-0.035***	-0.092	-0.032***	-0.176
	(0.007)	(0.116)	(0.009)	(0.110)
Cancer	0.019**	0.010	0.010	0.084
	(0.009)	(0.079)	(0.010)	(0.089)
Lunge disease	-0.045***	-0.039	-0.037***	-0.153
	(0.009)	(0.108)	(0.011)	(0.118)
Heart problem	-0.014**	-0.076	-0.001	0.126*
	(0.006)	(0.070)	(0.007)	(0.074)
stroke_max_cog	-0.023**	-0.339**	-0.036***	-0.221
	(0.011)	(0.152)	(0.012)	(0.166)
Arthritis	-0.018***	-0.112**	-0.028***	-0.220***
	(0.005)	(0.054)	(0.006)	(0.060)
parkine_max_cog	-0.036	-0.631*	-0.033	-0.066
	(0.029)	(0.337)	(0.034)	(0.282)
Prob. living to 75-120	0.000**	-0.001	0.000***	-0.000
	(0.000)	(0.001)	(0.000)	(0.001)
Observations	54,152	7,374	54,152	8,676
Age FE	✓	✓	✓	✓
Time FE	✓	✓	✓	✓
Baseline covariates	✓	✓	✓	✓
Mean outcome	0.31	9.76	0.37	9.05

Note: Standard errors clustered at household level in parentheses, *** p<0.01; ** p<0.05; * p<0.10. *Risky investment:* Shares and shares ISAs. *Memory:* Number of correct words recalled, from 10 words immediate and delayed memory test. Score from zero (worse memory) to 20 (best memory). In panel B, the highest cognition is the baseline category (15–20 correct recalled words). *Source*: ELSA

Table C.6: Household fixed effects model with intensive and extensive margin of financial investments as outcome variable and cognition as main explanatory variable

VARIABLES	(1) Holds non-risky financial investments (0 1)	(2) Logarithm of non-risky financial investments (continuous)	(3) Holds risky financial investments (0 1)	(4) Logarithm of risky financial investments (continuous)
Memory	0.001	0.003	0.001*	0.015
	(0.001)	(0.011)	(0.001)	(0.010)
Lab. - Employed	-0.005	0.522	0.032	0.214
	(0.026)	(0.465)	(0.026)	(0.354)
Lab. - Self-employed	0.002	0.844*	0.066**	0.509
	(0.028)	(0.496)	(0.028)	(0.370)
Lab. - Unemployed	-0.001	0.673	0.023	0.412
	(0.030)	(0.511)	(0.030)	(0.395)
Lab. - Partly retired	0.017	0.328	0.027	0.385
	(0.037)	(0.579)	(0.035)	(0.413)
Lab. - Retired	0.014	0.681	0.037	0.436
	(0.026)	(0.464)	(0.026)	(0.351)
Lab. - Disabled	-0.001	0.654	0.017	0.413
	(0.026)	(0.592)	(0.027)	(0.427)
Lab. - Look after family	0.014	0.416	0.042	0.415
	(0.026)	(0.477)	(0.027)	(0.366)
Net physical wealth	-0.000**	0.000**	0.000***	0.000
	(0.000)	(0.000)	(0.000)	(0.000)
Net housing wealth	0.000	-0.000	0.000*	0.000**
	(0.000)	(0.000)	(0.000)	(0.000)
High blood pressure	0.012	-0.061	-0.000	-0.051
	(0.008)	(0.084)	(0.007)	(0.083)
Diabetes	0.013	0.079	0.013	0.047
	(0.010)	(0.133)	(0.010)	(0.147)
Cancer	-0.002	-0.071	0.010	0.103
	(0.012)	(0.097)	(0.011)	(0.117)
Lunge disease	0.010	-0.040	0.011	0.042
	(0.013)	(0.155)	(0.012)	(0.192)
Heart problem	-0.007	0.065	-0.001	0.040
	(0.009)	(0.097)	(0.008)	(0.085)
Arthritis	-0.000	-0.129*	0.004	-0.002
	(0.008)	(0.078)	(0.007)	(0.074)
Prob. living to 75-120	0.000	0.002	0.000***	0.001
	(0.000)	(0.001)	(0.000)	(0.001)
Observations	54,152	7,374	54,152	8,676
Number of HHds	16,996	3,925	16,996	4,381
HHds FE	✓	✓	✓	✓
Age FE	✓	✓	✓	✓
Time FE	✓	✓	✓	✓
Mean outcome	0.306	9.764	0.369	9.054
Mean waves HHd	3.186	1.879	3.186	1.980

Note: Standard errors clustered at household level in parentheses, *** p<0.01; ** p<0.05; * p<0.10. *Risky investment:* Shares and shares ISAs. *Memory:* Number of correct words recalled, from 10 words immediate and delayed memory test. Score from zero (worse memory) to 20 (best memory). In panel B, the highest cognition is the baseline category (15–20 correct recalled words). *Source*: ELSA