Household Finance under the Shadow of Cancer

Dániel Kárpáti*

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Abstract

I study the causal effects of life expectancy on households' financial and economic decisions. My sample consists of individuals who undergo genetic testing for a hereditary cancer syndrome. Genetic testing randomizes tested persons into two groups. Those who test positive learn that they face a high risk of cancer and a shorter life expectancy. Those who test negative learn that their cancer risk is not elevated. The differences in outcomes between these two groups identify the effects of life expectancy. I find that life expectancy has a positive effect on wealth accumulation. Lower savings rates, safer portfolios, decreased labor supply, and different preferences for household composition explain lower wealth accumulation under reduced life expectancy.

JEL Classifications: G51, D14, E21, I10, J22

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*Department of Finance, Tilburg University, d.karpati@uvt.nl, Warandelaan 2, 5037 AB Tilburg, the Netherlands. I owe special thanks to Julio Crego, Jens Kværner and Luc Renneboog. I am grateful to the colleagues of the Stichting Opsporing Erfelijke Tumoren (StOET) for their help with accessing the Dutch Hereditary Cancer Registry. The paper has greatly benefited from the conference discussions of Tianyue Ruan, Kim Fe Cramer, Brendan Walsh, Luís Teles Morais, Vincent Tena, Roger Prudon, Steffen Meyer, Federica Teppa, and Qingyuan Yang. Results based on calculations by Dániel Kárpáti using non-public microdata from Statistics Netherlands. The research for this publication was partly funded by the Open Data Infrastructure for Social Science and Economic Innovations (ODISSEI) in the Netherlands (www.odissei-data.nl). Previously circulated as Household Finance and Life-Cycle Economic Decisions under the Shadow of Cancer.

1 Introduction

Life expectancy plays a fundamental role in the economic behavior of individuals and households in standard life-cycle models. Yet, empirical evidence on the causal effects of life expectancy on wealth accumulation, labor supply, portfolio choice and other important economic and financial choices is still limited. This is due to at least two empirical challenges.

First, life expectancy is correlated with income, health behavior, and other possibly unobserved variables that may directly affect economic decisions (De Nardi, Pashchenko, and Porapakkarm, 2017). Identifying the causal effects of life expectancy therefore requires exogenous variation in the mortality risks individuals face. Second, such exogenous variation should be independent of episodes of bad health. While bad health does reduce life expectancy, it may also directly impact labor productivity, medical expenditures, disability, access to credit, and other determinants of economic choices (García-Gómez et al., 2013; Dobkin et al., 2018).

To overcome these empirical challenges, I exploit a natural experiment that presents exogenous variation in life expectancy without imposing current bad health. I study individuals who undertake genetic testing for Lynch Syndrome (LS), a hereditary disorder that drastically increases the lifetime risk of colorectal, endometrial, and ovarian cancer.[1] LS may reduce the median lifespan by more than 13 years, which exceeds the life expectancy loss suffered by lifetime smokers (Doll et al., 2004).[2]

Facing these risks, individuals in my sample decide to undergo genetic testing to learn if they have inherited the gene mutation that causes LS in their families. Tested individuals are still healthy, they have not yet developed cancer. Genetic testing randomizes tested individuals into two groups. Those who test positive learn that they face elevated risks of cancer and a shorter life expectancy. Those who test negative learn that their cancer risks are similar to those of the general population. Both groups may react to genetic testing: The differences in their reactions identify the causal effects of the life expectancy reduction in LS. My genetic dataset contains a balanced sample of both positive- and negative-tested individuals. I merge this dataset with Dutch administrative panel data on a rich set of socio-economic variables, including household balance sheets, income, and demographic characteristics.

[1] Individuals who carry a Lynch Syndrome gene mutation face a 50 to 80% lifetime risk of colorectal cancer, a 25 to 50% risk of endometrial cancer, and a 5 to 10% risk of ovarian cancer. In comparison, the lifetime risk in the general population for colorectal, endometrial, and ovarian cancer are 5.5%, 2.7%, and 1.6%, respectively (Wolf, Buchanan, and Farkas, 2013).

[2] Many of the people in my sample experienced the very high mortality of LS at first hand because one parent of each tested individual was affected by the condition. On the other hand, due to improved preventive care and cancer treatment, LS-affected individuals in my sample may eventually experience a smaller reduction in their longevity. Based on the deaths that have occurred in my sample to date, I estimate that the median lifespan of LS-positive individuals in my sample is reduced by 3 to 4 years. This reduction is similar to the life expectancy loss suffered by smokers who quit smoking at the age of 50.

I start my analysis by estimating the effects of changing life expectancy on household wealth accumulation. In a canonical life-cycle model, households that face a lower life expectancy would have lower needs to save for retirement, and would consequently accumulate lower wealth (Bloom et al., 2003). In line with this prediction, I find that the financial assets of households of positive-tested individuals exhibit a gradual decline following genetic testing, compared to the financial assets of households of negative-tested individuals. Financial assets are the sum of bank deposits and financial securities (stocks, bonds, and investments in funds). By the fifth year after testing, households of Lynch-positive individuals hold on average EUR 50,000 lower financial assets than household of negative-tested individuals. During the next years, the difference slightly increases, and subsequently it stabilizes. On average, during the first 15 years after testing, households of positive-tested individuals hold EUR 60,000 lower financial assets. This is a substantial difference given that households of negative-tested individuals hold on average EUR 91,000 in financial assets. Quantile regressions show that not only households with the highest financial asset holdings are affected: During the first 15 years after testing, median financial assets are also EUR 9,000 lower among households of positive-tested individuals. This is a sizeable negative effect compared to the median financial asset holding of EUR 25,500 among households of negative-tested individuals. I also study a specification where the dependent variable is the natural logarithm of financial assets, and find that households of positive-tested individuals hold on average about 40% lower financial assets during the post-testing period. The negative effect on financial asset accumulation appears to be stronger among men (who face a greater reduction of life expectancy in LS), and individuals who had no children before testing (who possibly have weaker bequest motives), although these differences are not statistically significant. While financial asset accumulation is strongly affected, I find no significant treatment effects on the other wealth components of tested individuals, including real estate wealth, other assets (e.g., businesses), and other debt (e.g., student loans).

Next, I explore four channels that may explain the negative effects of Lynch Syndrome and the associated reduced life expectancy on financial asset accumulation. These are changing household composition, decreasing labor and household income, lower portfolio allocation to risky financial assets, and lower savings rates.

First, I estimate the effects of Lynch Syndrome on two important demographic outcomes, having a partner and having children. These are important and interesting outcomes to study on their own, in addition they may also impact wealth accumulation. Having a partner may mechanically affect the level of financial assets in my sample because balance sheets are aggregated at the household level. Household composition may also impact wealth accumulation

by shifting preferences. I find that among individuals who are tested 45 years old or younger, five years after testing positive-tested individuals have an 8 pp. lower probability of having a partner (married or unmarried). By year nine the difference grows to 10 pp., although it shrinks in later years. The negative effect is present both among men and women, and also both among individuals with a partner before testing and those without a partner. Individuals who are tested after turning 45 years old are not affected. Genetic testing also impacts reproductive choices among previously childless individuals who are tested when still in the reproductive age (here defined as 45 years old or younger). Positive-tested individuals in this group are on average 12 pp. less likely to ever have children. This is a sizeable effect given that about half of the negative-tested individuals in this group will eventually have children. Although these treatment effects on household composition and childbearing are far from negligible, they likely only explain a minor part of the negative effect of LS on financial asset accumulation. This is because in the whole sample the probability of having a partner is only slightly negatively affected (-2.8 pp.), while the negative effects on having any children are restricted to about $1/4^{th}$ of the sample who have no children before testing and who test in a childbearing age. Indeed, when I augment the regressions models of financial asset accumulation with indicators of having a partner and having any children, the estimated treatment effects are hardly diminished (e.g., from EUR 60,000 to EUR 58,000).

Life expectancy may also affect wealth accumulation by impacting individuals' labor and household income. In the model of Bloom et al. (2003), a decrease in longevity leads to increased demand for leisure and reduced labor supply. Indeed, I document that LS has a negative effect on labor income, but only among males. Female labor income is not affected, although I document some negative effects on the labor participation of women in the medium term. During the first five years after testing, positive-tested males earn on average EUR 7,500 lower annual labor income. This is a major (19%) difference compared to the mean male labor income in the sample. During the next five years, the difference further increases to EUR 9,800 annually, although subsequently it slightly drops to EUR 8,000 annually. The negative effect on male labor income can be explained both by lower labor participation (-3.2 pp., not statistically significant), and by lower labor supply among those who work. In the first 15 years after testing, positive-tested working males work on average 19 fewer full-time equivalent days per year than negative-tested working males. This is a 5.7% reduction compared to the mean annual FTE days among all working men in the sample. I estimate that it is in the pre-retirement age (60-64) when working men's labor earnings and labor supply are the most negatively affected. Contrary to the negative effect on labor supply, I find that wages are unaffected. Finally, I

turn to estimating the effects of LS on disposable household income, the income measure that presumably has the strongest influence on household wealth accumulation. I estimate slightly larger negative effects on household income than on labor income. This is predominantly because the labor income of the partners of tested individuals is also negatively affected, although in most specifications not statistically significantly. Notwithstanding the sizeable treatment effects on tested individuals' labor and household income, I argue that the income channel likely plays only a minor role in explaining the negative effects on financial asset accumulation. This is because Dutch households typically save a low share of their disposable income.

Households' portfolio choices may also change following a shift in their life expectancy. Individuals who face a lower life expectancy, and consequently a shorter planning horizon, may find it optimal to invest a lower share of their financial wealth into risky assets because of mean reversion in returns (Barberis, 2000). Decreasing lifetime labor income, which is often considered a safe asset, may also prompt people to shift away from risky financial investments. In line with these predictions, I find that LS has a strong negative effect on households' risky investments. By the end of the first year after genetic testing, households of positive-tested individuals hold a 6.3 pp. lower share of their financial assets in financial securities compared to households of negative-tested individuals. Dutch households' financial securities mostly comprise stocks and investments in equity mutual funds. Therefore, I interpret this estimate as a 6.3 pp. negative effect on the risky share of financial assets. The negative effect slightly increases in the following years, but remains largely unchanged during the follow-up period. On average, during the first 15 years of the post-testing period, positive-tested households hold a 9 pp. lower share of their financial assets in financial securities. This is an economically significant effect given the unconditional risky share of 12 pp. in the sample. I find that most of this effect is due to changes at the extensive margin, i.e., households of positive-tested individuals are less likely to hold any financial securities. While these results show that LS has a strong effect on the risky share, a back-of-the-envelope calculation suggests that lower financial returns are also not a major factor in the documented lower accumulation of financial assets.[3]

Finally, I study the effects of life expectancy on the strongest determinant of wealth accumulation, savings behavior. In the model of Bloom et al. (2003), reduced life expectancy leads to lower savings rates at every age due to the lower need to save for retirement. The previously documented large negative effect on financial asset accumulation strongly suggests that reduced life expectancy indeed leads to lower savings rates in my sample. This is particularly true given

[3]Multiplying the average financial assets of households in my sample (EUR 77,000) by the negative treatment effect on the risky share (9 pp.) and by an equity risk premium of 6% yields an estimate of EUR 415 annual returns foregone due to the lower risky share. This is a small amount compared to the total negative effects on financial assets, which is about EUR 50,000 five years after genetic testing.

that the other channels through which Lynch Syndrome may affect asset accumulation (changing household composition, lower incomes, and more conservative portfolio allocations) are likely of limited importance. To further investigate the effects of life expectancy on households' savings behavior, I construct a savings rate measure that is equal to the share of disposable household income that the household saves. An important caveat of this exercise is that the savings rate is measured with considerable noise because I impute savings from changes in household wealth corrected for capital gains. Notwithstanding the noisy measure, I estimate a strong negative treatment effect on savings rates: Households of positive-tested individuals indeed save a lower share of their disposable income than households of negative-tested individuals.

The core of this paper studies the differences in outcomes of positive- and negative-tested individuals following genetic testing. On the other hand, it is interesting to understand how these two groups react to genetic testing separately. Are the observed differences in outcomes due to the changing behavior of positive- or negative-tested individuals? Answering this question can help to determine the costs and benefits of genetic testing, and it can also shed light on how people react to upward and downward shifts in life expectancy. The ideal natural experiment to answer this question would randomize people into untested, positive-tested, and negative-tested groups. Because I lack such experiment, I apply a matching strategy and use individuals from the general Dutch population as a benchmark group of untested individuals. I find that compared to this benchmark group, both positive- and negative-tested individuals change their behavior following genetic testing. These changes are in the expected opposite directions. For example, those who test positive and experience a drop in life expectancy start to accumulate fewer financial assets. On the contrary, those who test negative and experience an increase in life expectancy start to accumulate more financial assets. I observe similar reactions for most of the other outcomes as well, including having a partner and having children. These findings suggest the people react to both good and bad news about their life expectancy. These results also highlight the potential benefits of genetic testing on negative-tested individuals: by alleviating health and mortality risks, genetic testing can help improve the socio-economic outcomes of these people.[4]

My work aims to contribute to our understanding on how individuals incorporate life expectancy into their decision-making. This research question is closely related to several active research areas in the fields of household finance, household economics, macroeconomics, and health economics.

At least since Hamermesh (1985), a broad literature studies the role of expectations and

[4]Assuming that having children, earning a higher labor income, and holding more financial assets is beneficial.

particularly life expectancy on household financial and economic behavior. In macroeconomics, most work relies on cross-country samples, and mostly documents a positive correlation between average life expectancy at birth and savings rates (e.g., Bloom et al., 2007). At the micro-level, the majority of previous work focuses on the later years of life, and reports that individuals who expect a shorter lifespan exhibit lower consumption growth (Salm, 2010), adjust their consumption expenditures upwards (Bíró, 2013), and have a greater propensity not to save (Heimer, Myrseth, and Schoenle, 2019). My findings are consistent with this literature. In addition, my work aims to complement the literature in several ways. First, I exploit the natural experiment of genetic testing to identify the causal effects of life expectancy. Genetic testing offers a research design close to a randomized experiment, which helps to overcome the challenges of weak instruments and exclusion restriction violations that previous studies potentially faced.[5] Second, I use comprehensive administrative data to estimate the effects of life expectancy on household wealth accumulation. Previous work often focused on limited measures of consumption (e.g., food consumption). Third, my sample includes both young and old people, while previous studies mostly focused on the behavior of retirees or people close to retirement. This is important because younger people may react to changes in life expectancy differently, e.g., they can adjust their labor supply. Fourth, I study the short-term and long-term effects of life expectancy on a comprehensive set of outcomes including wealth accumulation, labor supply, and portfolio allocation decisions[6], in a uniform framework. Fifth, I document that people react both to positive and negative shocks to life expectancy.

This paper is also connected to the literature on the economic effects of health shocks. Adverse health shocks are a major source of economic risk for individuals, with a possible impact on labor earnings (Dobkin et al., 2018; García-Gómez et al., 2013), consumption/saving decisions (Kolsrud, Landais, and Spinnewijn, 2020; Meyer and Mok, 2019), and portfolio composition (Døskeland and Kvaerner, 2021), among many other outcomes. While the literature

[5]Not all previous work relies on IV strategies. Oster, Shoulson, and Dorsey (2013) and Oster et al. (2010) also exploit the natural experiment of genetic testing (but for Huntington's disease, a progressive neurological disorder) to study the effects of life expectancy on investments in human capital and long-term care insurance choices, respectively. Baranov and Kohler (2018) estimate that increases in life expectancy due to better access to AIDS treatment lead to increased savings, expenditures on education, and children's schooling in rural Malawi. In comparison, I study the effects of life expectancy in a developed country where saving for old age is more important, and where the extensive welfare system limits other health-related (precautionary) savings motives. In contemporaneous work, Horn (2022) estimates that the death of a close friend induces a reduction in saving rates. The death of a close friend may contribute to increased survival pessimism, even if it is uninformative about the mortality risks that the individual faces. In comparison, genetic testing provides individuals in my sample relevant information on their mortality risks.

[6]Some literature studies the effects of life expectancy on portfolio allocation. Spaenjers and Spira (2015) estimate a positive effect of subjective life expectancy, instrumented by the current age or age at death of the survey respondent's parents, on equity portfolio shares. Balasubramaniam (2021) reports that survival pessimism, instrumented by experiences of mass shootings and natural disasters, reduces the time horizon for financial planning and investment in risky assets. My findings are consistent with this literature.

mostly focuses on how individuals and households react to contemporaneous health shocks, I exploit the setting of genetic testing to show that human behavior and socio-economic outcomes also react to expected future health shocks. My results also illustrate that the extensively documented negative correlation between wealth and health (e.g., De Nardi, Pashchenko, and Porapakkarm, 2017) may arise partially because individuals with worse health expectations and higher mortality risks accumulate lower wealth.

The primary goal and contribution of my work is to understand the effects of life expectancy on economic behavior. However, I argue that my work also contributes to the medical literature. Understanding how people react to the results of genetic testing may be an important consideration for clinical geneticists and other medical professionals. This is especially true since predictive genetic testing might soon be offered for the general population[7], and many private providers (e.g., 23andMe) already offer testing for some of the more frequent single-gene genetic disorders (e.g., hereditary breast cancer).

The paper proceeds as follows. Section 2 describes the sample, presents the cancer risks and mortality associated with Lynch Syndrome, and discusses the theoretical predictions of changing life expectancy. Section 3 presents the data sources and the main variables, and discusses the empirical strategy. Section 4 presents the main results on the effects of life expectancy on wealth accumulation. Section 5 studies five channels through which life expectancy and Lynch Syndrome may affect wealth accumulation, household composition, labor and household income, portfolio allocation, savings behavior, and mental health. Section 6 studies the effects of genetic testing on those who test positive and those who test negative. Section 7 concludes.

2 Background and Incentives

Lynch Syndrome (LS) is a hereditary condition that gives rise to a substantially increased lifetime risk of cancer. LS is caused by a mutation in one of five genes (MLH1, MSH2, MSH6, PMS2, EPCAM), which leads to an impaired ability to suppress tumor growth. LS mostly increases the risk of colorectal and endometrial cancer, but the risks of many other types of cancer are also elevated. Lynch Syndrome historically had a large negative effect on life expectancy (about 13 years of reduction in the median lifespan), although in recent years screening and preventive surgeries have improved survival. The condition is inherited in an autosomal dominant manner: individuals with one parent who carries a Lynch mutation have a 50% probability of

[7] In late 2022, a nationally collaborative project was launched in Australia that will screen at least 10,000 people aged 18-40 for genes that increase risk of certain types of cancers (including Lynch Syndrome) and heart disease. Source: https://www.monash.edu/news/articles/world-first-preventative-dna-screening-for-cancer-and-heart-disease-risk2

inheriting the faulty gene. Individuals who test negative for the mutation responsible for Lynch Syndrome in their families face a lifetime cancer risk and life expectancy similar to that of the general population.

My sample consists of 890 individuals who started their life at a 50% risk of having inherited the gene mutation that causes Lynch Syndrome in their families. Before the mid-1990s, the exact genetic cause of LS was unknown. Families suspected of the condition were identified based on their family history of cancer. The vast majority of individuals in my sample belong to such families. Given their risk exposure, and following the recommendations of their doctors, these people registered with the Dutch Hereditary Cancer Registry (DHCR). The goal of the registry is to promote the identification of families with various forms of hereditary cancer and to encourage high-risk individuals to participate in medical surveillance programs.

Once genetic testing became available, individuals in my sample decided to undergo testing to learn if they have actually inherited the bad gene. By this time, the exact gene mutation responsible for LS in their families had already been determined following the testing of family members who had a Lynch-specific cancer. These initially tested family members are not part of my sample as I only consider individuals who had no cancer before genetic testing. Studying people who have not yet developed cancer offers two advantages. First, these people can learn from their genetic test results, instead of simply receiving a confirmation of a highly-likely case of LS based on their cancer history. Second, the behavior of these people is not affected by current bad health. This enables me to separate the effects of changing life expectancy from the effects of current health conditions, such as cancer.

Testing is a choice. Although I estimate that over 70% of the family members in the families in my sample decided to get tested, it is well possible that individuals who decided to test differ in some observable or unobservable characteristics (e.g., risk aversion) from those who faced similar risks but decided not (yet) to test.[8] Importantly, my identification strategy does not build on comparing tested and untested individuals. Instead it compares people who test positive or negative for the suspected mutation. Individuals in both of these groups can learn from the results of genetic testing. The former group receives confirmation about the bad risk they were potentially exposed to, while the latter group is relieved from this risk. The differences between the outcomes of these two groups identify the causal effects of facing the risks associated with Lynch Syndrome. The majority of this paper is concerned with studying the differences in the outcomes of these two groups after they learn about their differential mortality risks.

[8]For example, as Panel A of Table A2 in Appendix A presents, tested people in my sample are slightly different in observable characteristics from a birth year-gender matched sample of the general Dutch population: They are more likely to have children and a partner, earn a somewhat higher household income, have about 18% higher financial assets, and are 9 percentage points more likely to own a home.

Section 6 sets out to separately study the reaction of positive- and negative-tested individuals to genetic testing, using the benchmark of the untested Dutch population.

2.1 Life Expectancy in Lynch Syndrome

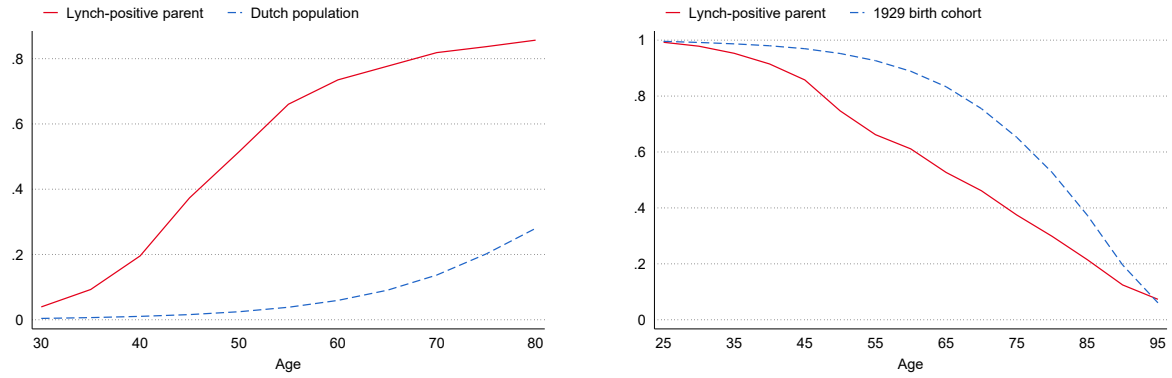
To contextualize the economic reaction to a Lynch Syndrome diagnosis, it is important to understand the risks associated with the condition. The medical literature reports that people with a Lynch mutation, depending on their sex and the exact type of the mutation, are exposed to a 50 to 80% lifetime risk of colorectal cancer. Female carriers face an additional 25 to 50% risk of endometrial cancer and a 5 to 10% risk of ovarian cancer. In comparison, the lifetime risks in the general population for colorectal, endometrial, and ovarian cancer are 5.5%, 2.7%, and 1.6%, respectively (Wolf, Buchanan, and Farkas, 2013). The risks of some other forms of cancer such as stomach, small bowel, upper urological tract, biliary tract, and brain cancer are also elevated for Lynch mutation carriers.

As a letter from a clinical geneticist presented in Appendix A Figure A3 reveals, these baseline risks are clearly communicated to those who undergo genetic testing. People in my sample had also experienced these risks personally: they are part of families with a long history of Lynch Syndrome-related cancers. Panel (a) of Figure 1 (red line) presents the lifetime cancer incidence that the Lynch-affected parents[9] of individuals in my sample experienced. By the age of 50, over half of these parents developed some form of cancer (mostly colorectal). By the age of 70 this portion exceeded 80%. In comparison, the estimated lifetime cancer incidence faced by the general Dutch population (dashed blue line) is considerably lower, reaching about 16% by the age of 70. These excessive cancer risks were also reflected in a substantially reduced life expectancy. Panel (b) compares the age-dependent survival of the Lynch-affected parents with the survival of a comparable cohort from the Dutch population.[10] The difference in lifespan is dramatic: while the median length of life in the population was 81 years (84 for females, 77 for males), I estimate a median lifespan of only 68 years (75 for females, 63 for males) for the Lynch-affected parents. Pylvänäinen et al. (2012) report a similar lifespan reduction in a Finnish sample. To put it into context, the 13 years reduction in median lifespan even exceeds the negative effects of lifetime smoking (10 years) (Doll et al., 2004).

Although the experiences of their parents are likely important factors in how tested individuals think about the risks of Lynch Syndrome, my data also enables me to give an estimate

[9] As discussed in Section 3, one of the parents of the individuals in my sample was almost certainly carrying a Lynch-specific gene mutation, although they might have died before genetic testing became available. I determine if this parent is the mother (maternal inheritance) or the father (paternal inheritance) using family trees preserved in the Dutch Hereditary Cancer Registry. I refer to this parent as the Lynch-affected parent. It seldom happens that both parents carry a Lynch mutation, and in these cases their children very rarely survive to adulthood.

[10] 1929 is the median birth year of the Lynch-affected parents.



(a) Cumulative cancer incidence

(b) Survival

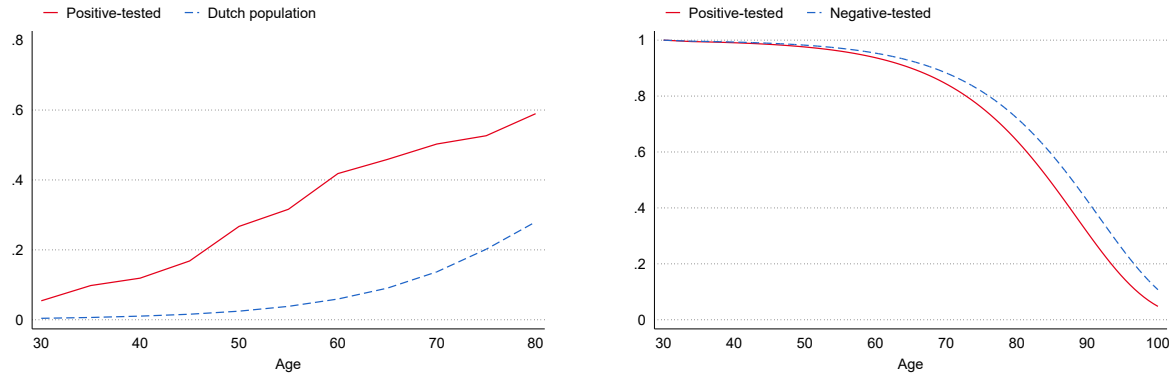
In both figures, the line *Lynch-positive parent* presents non-parametric Kaplan-Meier estimates on cancer incidence and survival, respectively, for the Lynch-affected parents of the tested individuals in my sample. I determine the Lynch-affected parent (mother or father) based on family information stored in the Dutch Hereditary Cancer Registry. Data on cancer incidence is also from the DHCR. Data on birth and death years are partially from the DHCR and partially from Statistics Netherlands. In figure (a), the line *Dutch population* refers to the probability of being diagnosed with at least one cancer among the general Dutch population, calculated following Sasieni et al. (2011), based on current cancer incidence statistics. In figure (b), the line *1929 birth cohort* refers to the survival probabilities of those Dutch individuals who were born in 1929 and who were still alive at the age of 21 in 1950. I estimate these survival probabilities using public data of Statistics Netherlands.

Figure 1: Effects of Lynch Syndrome on the cancer incidence and survival of the *Lynch-affected parents* of people in the sample

of tested individuals' realized cancer and mortality risks. As Panel (a) of Figure 2 presents, LS-positive individuals in my sample have experienced a lower increase in cancer incidence compared to the general population than their parents (a gap of about 30 percentage points by the age of 50 vs. a gap of 50 pp. for their parents at the same age). They have also suffered, thus far, from lower mortality: In Panel (b) of Figure 2 I fit a parametric survival model on the observed mortality patterns of positive- and negative-tested individuals in my sample. The estimates suggest a 3.2 years reduction in median lifespan for the LS-positive group.[11] This is still a non-negligible reduction in lifespan, comparable to the negative effects of smoking if someone quits at the age of 50 (Doll et al., 2004). The main reason behind this improvement is the availability of risk mitigating procedures, periodical cancer screenings and in some cases preventive surgeries.[12]

[11] The parametric Gompertz model controls for sex, the age at testing in groups of 10 years, the mutation type, and the partnership status in the year before testing. I also perform a non-parametric Kaplan-Meier survival analysis, which suggests a 4 year median lifespan difference between positive- and negative-tested individuals. These estimates should be treated with caution as they are not statistically significant, presumably due to the very low number of deaths (77 out of the 890 people) that I observe.

[12] Preventive surgery against colon cancer (colectomy, the surgical removal of most of the colon) is rarely applied due to the associated reduction of quality of life, but regular colonoscopy screenings can substantially reduce colorectal cancer incidence and mortality among LS patients (De Jong et al., 2006). The evidence on the benefits of gynecological screenings is more limited; on the other hand, prophylactic hysterectomy (removal of the uterus/womb) and salpingo-oophorectomy (removal of the ovaries and the Fallopian tubes) can eliminate



(a) Cumulative cancer incidence

(b) Survival

In figure (a) the line *Positive-tested* presents non-parametric Kaplan-Meier estimates on the cancer incidence (any cancers) of positive-tested people in my sample. The start of the observation time is the year of genetic testing, failure is defined as developing cancer for the first time, and observations are censored at the year of death or 2018 at the latest. Observations are not censored at the year of preventive surgeries. The line *Dutch population* refers to the probability of being diagnosed with at least one cancer among the general Dutch population, calculated following Sasieni et al. (2011), based on current cancer incidence statistics. In figure (b), the two lines refer to positive- and negative-tested individuals in my sample. Survival curve estimates from a parametric (Gompertz) survival model are presented. The model controls for sex, the age at testing in groups of 10 years, the mutation type, and the partnership status in the year before testing.

Figure 2: Effects of Lynch syndrome on the cancer incidence and survival of people in the sample

Individuals tested for Lynch Syndrome take their genetic test results seriously: They correctly recall their test outcome, exhibit high compliance with recommended cancer screening protocols, and positive-tested individuals in general report their cancer risks as high (Aktan-Collan et al., 2001; Järvinen et al., 2009). I also estimate a very high compliance with the recommended cancer screening protocols in my sample (Figure A5 of Appendix A). Before testing, both eventually positive-tested and negative-tested individuals participate in cancer screening on average once every five years (1/0.2). These screenings include colonoscopies and gynaecological check-ups. Following testing, negative-tested individuals completely stop with the screenings, while those who test positive start to screen on average every two years. This is in line with the medical recommendations.

The changes in the cancer screening behavior of the two groups highlight that tested individuals understand and internalize their test results. It is less obvious how they update their beliefs on cancer risks and mortality, as this depends on how they perceived the risks in LS before testing. A large literature argues that individuals rely on the longevity of relatives when

the risks of endometrial and ovarian cancer. These procedures are recommended to LS-positive women after the age of 40, and/or once they do not wish to have (more) children. The increased risks of extra-colonic and extra-endometrial cancers (e.g. upper urological tract, pancreatic cancer, and brain cancer) in LS are hard to mitigate. Pylvänäinen et al. (2012) estimate that in recent years these types of cancer are responsible for about half of all cancer-related mortality among LS-positive individuals.

they form subjective survival expectations (Hamermesh, 1985; Hurd and McGarry, 2002, 1995). In this case tested individuals may fear a LS that reduces their life expectancy by 13 years. These beliefs would not necessarily be irrational given that tested individuals might not have perfectly foreseen that improved screening and prevention would reduce the mortality of Lynch Syndrome. On the other hand, positive-tested individuals in my sample regularly participate in cancer screenings, which suggests that they expect to benefit from these procedures. My estimates on objective mortality bound the subjective beliefs on the negative mortality effects of LS between 3.2 years and 13 years.[13]

Cancers associated with Lynch Syndrome may not only increase mortality but might also threaten with high medical costs and disability. The impact of these risks appears to be limited in the Netherlands. The Dutch universal health insurance largely alleviates the threat of excessive medical costs, although due to deductibles a part of cancer screening and eventual cancer treatment costs must be paid out of pocket (up to approximately EUR 300 per year during the sample period).[14] The Netherlands also offers a generous and comprehensive public long–term care system, where the role of out-of-pocket expenses, about 9% of total costs, are small relative to other countries (Bakx, O'Donnell, and Van Doorslaer, 2016). The threat of earnings loss due to disability also appears to be bounded. I estimate that in the first 15 years after testing, positive-tested individuals in my sample are on average only slightly more likely (1.9 pp., not statistically significant) to receive disability benefits than negative-tested individuals (column 3 of Table A1 in Appendix A). It is possible that tested individuals form their beliefs on disability risks in LS based on their parents' experiences. There are no good data available on the frequency of disability among these Lynch-affected parents. Based on cases of both hereditary and non-hereditary cancers, Crego et al. (2022) estimate that disability risks in the most frequent types of cancers in LS (colon cancer and cancers of the female reproductive organs) are not excessive. Among the 5-year survivors of colon cancer (cancers of the female reproductive organs), 14% (6%) are disabled and those working lose about 3% (0%) of their labor income. The risk of income loss in LS is also limited by the very generous Dutch disability insurance scheme. This statutory insurance covers non-work-related disability as well, and on average replaces 70 percent of lost gross earnings. In the past, the statutory scheme was often supplemented by non-statutory benefits for specific collective labor agreements, raising the replacement rate of

[13]Data on subjective mortality beliefs are not available in administrative datasets. In an ongoing study, in collaboration with the Netherlands Foundation for the Detection of Hereditary Tumors, I develop a survey that offers the possibility to measure the subjective beliefs and preferences of individuals who have tested positive for a LS gene. This study may shed some light on this question.

[14]People diagnosed with Lynch Syndrome may be recommended to follow a healthy diet. They might also decide to exercise more. There are no good data on the costs of these lifestyle behaviors, but their effects on household budgets are likely limited.

workers from 70 to 80 or even 90% (Koning and Lindeboom, 2015). The progressive Dutch tax system and a broad range of means-tested benefits further increase the (net) replacement rate. Finally, Dutch laws guarantee access to health and non-health insurance products under generous conditions for those affected by hereditary conditions (proven by genetic testing or only suspected).

2.2 Theoretical Predictions

This paper studies the role of life expectancy on individuals' wealth accumulation, labor supply and portfolio allocation decisions. In life-cycle models of savings, decreasing longevity implies that individuals are less likely to live into the retirement age when they may need to supplement their pension income from their savings. This decreases the motivation to save at all ages, and leads to slower wealth accumulation (or faster de-accumulation).[15] On the other hand, decreasing longevity may also be associated with increasing morbidity and decreasing labor productivity, which may shorten the active working life and *increase* the need for saving (Bloom et al., 2003). As discussed in the previous section, this latter channel is likely of limited importance for Lynch-affected Dutch individuals whose labor income is well-insured by the generous disability insurance schemes. Precautionary saving is another savings goal. The high cancer risks in LS might motivate people to precautionarily save against medical costs. However, the Dutch universal health insurance scheme greatly limits this motive.[16] Finally, people may also save to leave bequests. People with strong bequest motives may reduce their savings rate less when faced with decreasing longevity. On the other hand, they might also decide to transfer their wealth to the next generation when they receive information on their mortality, in order to optimize inheritance tax payments (Kvaerner, 2022). Such transfers might lead to decreasing wealth levels.

Changing life expectancy may also impact optimal labor supply and retirement decisions. Bloom et al. (2003) shows that under the assumption that consumption and leisure are normal goods (i.e., the demand for both rises when wealth increases), decreasing life expectancy reduces labor supply and the optimal life spent working.

Reduced life expectancy can also impact individuals' portfolio allocation. Decreasing labor supply reduces the present value of future labor income, which is often considered as a safe asset in portfolio allocation problems. To keep overall portfolio risks unchanged, the individual

[15] The effects of a probabilistic increase in mortality risks would be more moderate than the effects of a non-probabilistic reduction of longevity. This is because individuals precautionarily save against longevity risk, the risk of outliving their resources (De Nardi, French, and Jones, 2009).

[16] People may also save to cover the costs of their funerals. However, about $3/4^{rd}$ of the Dutch population is covered by a funeral insurance.

would then need to decrease their allocation to risky assets within their financial portfolio. A shorten life expectancy also reduces the planning horizon. With mean-reverting returns, Barberis (2000) shows that a reduced planning horizon leads to a decreased allocation to risky assets. Finally, if individuals' preferences exhibit decreasing relative risk aversion (DRRA), lower wealth accumulation under reduced life expectancy may lead to a decreasing portfolio allocation to risky assets (Doeskeland and Kvaerner, 2022).

3 Estimation

3.1 Empirical Strategy

Genetic testing offers the prospect of an ideal natural experiment to estimate the causal effects of the changing risks associated with a positive or negative test result. This is because conditional on the probability of testing positive, the potential outcomes of tested individuals are independent of their realized test results.

There are two principal empirical challenges that we must overcome to reach this ideal experiment. The first is to establish the probability of testing positive for tested individuals in the sample, or to control for the covariates that determine this probability. The second is to observe both positive and negative test results. The institutional setting of genetic testing among families in my sample ensures that tested individuals almost certainly faced a 50% probability of inheriting a LS mutation. This is because within the Lynch-affected families registered in the DHCR, testing in general followed a systematic procedure referred to as cascade screening. The idea of cascade screening is to only test individuals who have a proven mutation carrier first-degree relative (parent or sibling). Having a mutation carrier first-degree relative puts the risk that the tested individual had inherited the mutation at 50%.[17] Figure A4 in Appendix A presents a simplified pedigree (family tree) of a family registered with the DHCR, and discusses how cascade screening was implemented in the family.

[17]The cascade screening procedure can not always be implemented, for example in the case of the initially tested family member (the so-called proband). However, probands are usually individuals with a history of Lynch-specific cancer. As I exclude all individuals who had cancer before testing, I also exclude such probands. Another case when cascade screening is not possible is when an individual has no surviving first-degree relatives (FDRs), and none of the deceased FDRs has previously tested positive. This mostly occurs when an individual's LS-suspected parent has already passed away. Nevertheless, if this parent has previously developed any cancer that is highly specific of LS (e.g., colon or endometrial cancer at a young age) that would very strongly suggest that they were Lynch mutation carriers. To verify that almost all individuals in my sample faced a 50% at-birth risk, I collect data on the cancer history and DNA test results of tested individuals' FDRs from family trees and other registers available at the DHCR. Based on criteria discussed in Appendix C.1 (e.g., previously positive tested FDR, parent that had LS-specific cancer, parent that died at a very young age), I find that at least 93% of my sample faced almost certainly a 50% risk of inheriting LS. This is a lower bound estimate because cancer and DNA testing history of family members is incomplete for many families/individuals. In robustness tests (in Panel D of Table A3 in Appendix A), I repeat my main analyses on this sub-sample of almost certainly 50% at-birth risk individuals. I find results close to identical to those in my baseline sample.

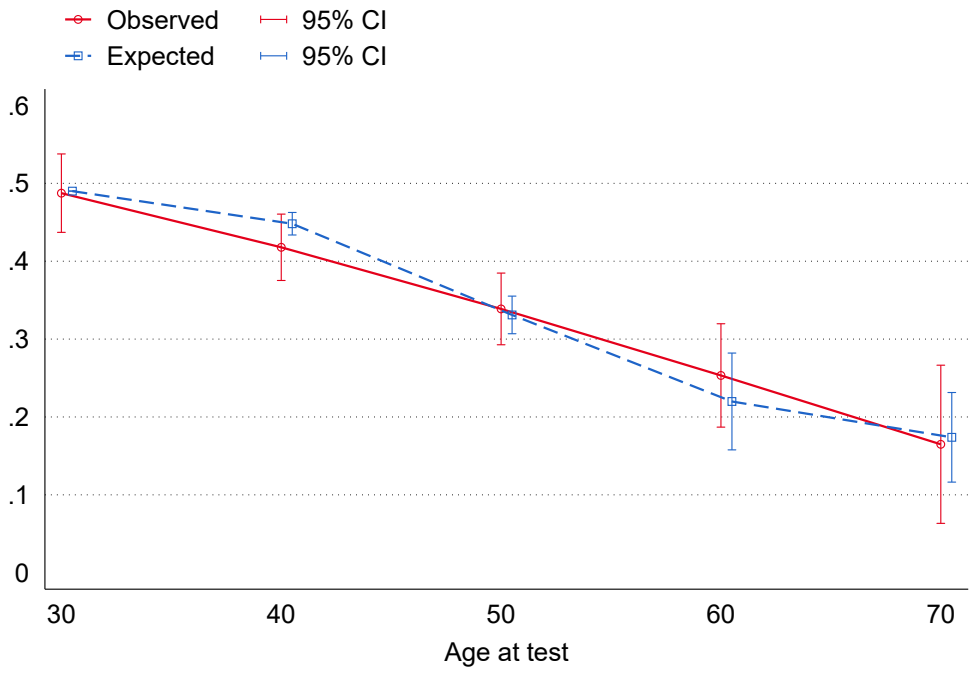
While individuals in my sample start their life at a 50% risk of LS, they may also learn about their mutation status by developing or not-developing a LS-specific cancer. For example, staying cancer-free by the age of 70 greatly reduces the risk of carrying a LS mutation. On the contrary, being diagnosed with colon cancer at the age of 40 is highly indicative of LS. The DHCR contains comprehensive information on the cancer diagnoses of registered individuals. Because individuals who already had cancer may learn little from genetic testing, and because they might suffer from bad health, I exclude them from my sample. To ensure that positive- and negative-tested individuals are comparable conditional on entering my sample (i.e., not having developed cancer yet), I control for three observable characteristics that are the most important determinants of cancer-manifestation in Lynch Syndrome. These are the age at testing, sex, and the gene that causes LS in the tested individual's family (MLH1, MSH2, MSH6, PMS2, or EPCAM).[18]

The second empirical challenge is to collect the genetic test results of all individuals who undergo testing, irrespective of the test outcome. This is crucial because as Oster, Shoulson, and Dorsey (2013) also highlight, the kind of persons who want to share their test results despite testing negative for a condition may form a highly selective group. For example, they may have greater family involvement of Lynch Syndrome, they may be more altruistic and aim to help scientific research, or they might simply doubt their test results. Using data from the Dutch Hereditary Cancer Registry makes it possible to overcome this challenge. The key characteristic of my sample is that it is based on individuals who had registered with the DHCR *before they underwent genetic testing*. This is because they belong to families where the presence of Lynch Syndrome had been strongly suspected based on family history of cancer. Crucially, the DHCR strives to obtain the genetic test results of all registered and tested individuals, irrespective of the test outcome. The registry can achieve this because it has strong connections with the registered individuals and their doctors.

That the DHCR does an excellent job in tracing genetic test results is also illustrated by Figure 3. The figure presents the share of positive-tested individuals (y-axis) at different ages of testing (x-axis). The solid red line shows the share that I observe in my sample (*Observed*). The dashed blue line presents the share that we would expect among cancer-free individuals with a 50% at-birth risk of LS (*Expected*). I calculate this latter measure using Bayes' rule based on

[18]Because testing is voluntary, individuals might select into testing based on any observable or unobservable characteristics. However, selection into testing could only bias the comparison of positive- and negative-tested individuals if it is based on a characteristic that is correlated with the LS mutation carrier status. As Figure A5 of Appendix A reveals, positive- and negative-tested individuals were equally likely to participate in cancer screenings before learning about their genetic test results. Also, as Table 1 below presents, these two groups exhibited very similar pre-testing characteristics. These facts provide strong evidence that selection into testing was not different between mutation-carrier and non-carrier individuals.

cancer incidence estimates for LS mutation carriers and for non-carriers. The two lines almost perfectly coincide. As previously discussed, individuals who start their life at a 50% risk of having inherited LS are equally likely to test positive and negative at the age of 25. This is because LS-specific cancers do not manifest before this age. In older ages, as I exclude from the sample individuals who already had cancer, the expected share of positive-tested individuals decreases.



The figure presents the probability of testing positive conditional on no prior cancer. *Observed* refers to the probability observed in my sample, fitted values from a regression of an indicator of testing positive on a 3^{rd} degree age polynomial. *Expected* is calculated using Bayes' rule assuming a 50% probability of carrying the mutation at birth, and cancer incidence estimates for LS mutation carriers and for non-carriers. Cancer incidence estimates are based on the estimates for the Lynch-affected parents and the general Dutch population presented in Panel (a) of Figure 1.

Figure 3: Probability of testing positive conditional on no prior cancer

While Figure 3 presents evidence that the sample includes a balanced number of positive and negative tested individuals, Table 1 reveals that these individuals are also very similar before they undergo genetic testing. The table presents results from linear regressions where the outcome variable is regressed on an indicator of having a positive test result. The sole significant differences between positive- and negative-tested individuals are in the age at testing and an indicator for facing the risk of a mutation in the MLH1 or MSH2 genes. This is as expected because both higher age and having an MLH1/MSH2 mutation are increasing the cumulative risk of cancer among LS-mutation carriers, i.e., mutation carriers with these characteristics are less likely to be included in the sample of *cancer-free* individuals. As previously discussed, I

Table 1: Comparing pre-testing characteristics of positive- and negative-tested individuals

	Variable	Positive	S.e.	Mean	N
(1)	Age at test	-5.07***	0.78	43.03	890
(2)	MLH1/MSH2 mutation	-0.05*	0.03	0.84	890
(3)	Female	0.00	0.03	0.55	890
(4)	Year of DNA test	0.06	0.31	2002.54	890
(5)	Number of siblings	-0.05	0.17	4.33	713
(6)	Maternal inheritance	0.02	0.04	0.45	789
(7)	Parent had cancer before test	0.00	0.03	0.82	789
(8)	Has child	-0.02	0.03	0.72	877
(9)	Has partner	0.04	0.03	0.79	856
(10)	Working	-0.03	0.04	0.78	591
(11)	Annual salary if working (EUR)	2,365	2,454	34,668	463
(12)	Disposable household income (EUR)	172	2,080	46,447	395
(13)	Financial assets (EUR)	3,166	20,116	65,219	212
(14)	Homeowner	0.02	0.04	0.74	554

The table reports coefficient estimates of regression models where individual and household characteristics (measured in the year before genetic testing) are regressed on an indicator of testing positive. Robust standard errors are presented in the column 'S.e.'. The unconditional mean in the sample is presented in the column 'Mean'. N refers to the number of observations, which varies between variables due to the different sample periods (e.g., wealth variables for most individuals are only available from 2006) and the differences in the sample selection criteria. The control variables are as follows. (1) Age at test: mutation type fixed effects and gender; (2) MLH1/MSH2 mutation: age at test and gender; (3) Female: mutation type fixed effects and age at test; (4) Year of DNA test: mutation type fixed effects, age at test, and gender; (5)-(14): mutation type fixed effects, age at test, gender, and year fixed effects. Regressions (1) to (7) are estimated on the whole sample, irrespective of the age at testing. For the remaining variables I apply the same sample selection criteria as elsewhere in the paper and include individuals who are (8)-(9) at least 20 years old, (10)-(12) 25 to 64 years old and diagnosed before turning 61 years old, (13)-(14) at least 25 years old and classified as the household head or their partner. * p < 0.1, ** p < 0.05, *** p < 0.01

control for these two characteristics (and for sex) when I compare the economic outcomes of positive- and negative-tested individuals.

Regression Models – Although, as Table 1 presents, positive- and negative-tested individuals in my sample are very similar before testing, some small imbalances are still present between the two groups. For example, positive-tested individuals are slightly more likely to have a partner but less likely to have children before testing. These small initial imbalances could bias our estimates of the causal effects of testing positive if we simply compared the outcomes of positive- and negative-tested individuals in the period after testing. Therefore, to further sharpen my identification strategy, I also exploit pre-testing data and control for individual fixed effects in my regression models. This eliminates the effects of potential differences in time-invariant characteristics. My baseline regression model is,

$$y_{i,t} = \alpha_i + \delta_t + \sum_{k=-6, k\neq -1}^{14} \beta_k \{K_{i,t} = k\} \cdot T_i + \boldsymbol{\gamma}'_{K_{i,t}} \mathbf{X_i} + \boldsymbol{\lambda}' \mathbf{Z_{i,t}} + \varepsilon_{i,t} \tag{1a}$$

where $y_{i,t}$ stands for outcome y (such as having a partner or labor earnings) of individual

i in calendar year t. α_i are individual fixed effects. δ_t are calendar year fixed effects. $K_{i,t}$ represents the relative years since the genetic test, where $K_{i,t} = 0$ is the year of the test. T_i is the treatment indicator that takes the value 1 for positive-tested and 0 for negative-tested individuals. β_k are the coefficients of interest, they represent the differential time trend (in relative years) of positive-tested individuals compared to negative-tested individuals (whose time-trend is picked up by $\boldsymbol{\gamma}'_{K_{i,t}}\mathbf{X_i}$). β_{-1} (the difference between positive- and negative-tested in the year before testing) is normalized to 0 due to the individual fixed effects. $\boldsymbol{\gamma}'_{K_{i,t}}\mathbf{X_i}$ are the relative year-specific effects of the three characteristics that I assume to drive cancer risk in LS, sex, age at testing, and mutation type. I control for these characteristics because they affect selection into the sample (due to the condition of having had no cancer before genetic testing). Age at testing enters $\mathbf{X_i}$ linearly, while mutation type refers to a set of indicators for each of the five Lynch genes (with the gene that the individual was tested for coded as 1, the remaining genes as 0). $\mathbf{Z_{i,t}}$ are a set of indicators for the individual's sex (female, male) interacted with the individual's age. The error term $\varepsilon_{i,t}$ is clustered at the individual level. The sample period is from 6 years before the genetic test to 14 years after.

For outcome variables that are derived from household balance sheet data, such as wealth components, the active savings rate, and the stock market participation indicator, I primarily rely on a version of Model 1a that controls for group (positive-tested) fixed effects instead of individual fixed effects. This is because wealth data are only available from end-2005 for the majority of individuals in my sample. Because many people tested earlier than 2006, for $3/4^{th}$ of the sample no wealth data are available before testing. Using group fixed effects instead of individual fixed effects enables me to exploit the information provided by these individuals too, while still controlling for time-invariant differences between the positive and negative-tested groups. The resulting model is

$$y_{i,t} = \alpha_{T_i} + \delta_t + \sum_{k=-6, k\neq -1}^{14} \beta_k \{K_{i,t} = k\} \cdot T_i + \boldsymbol{\gamma}'_{K_{i,t}}\mathbf{X_i} + \boldsymbol{\lambda}'\mathbf{Z_{i,t}} + \varepsilon_{i,t} \tag{1b}$$

where α_{T_i} was substituted for α_i. For all outcomes where my primary specification is Model 1b, I also execute robustness tests using Model 1a. These robustness tests provide similar results to the primary specification, although often yield larger standard errors.

While models 1a and 1b make it possible to evaluate pre-trends by observing the estimated β_k for all $k < 0$, as Borusyak and Jaravel (2017) note, they do not estimate the treatment effects efficiently: Given no pre-trends all β_k, $k < 0$, should be set to zero. I estimate two difference-in-differences specifications corresponding to Model 1a and Model 1b, respectively, where I compare changes between the pre-testing and post-testing periods,

$$y_{i,t} = \alpha_i + \delta_t + \beta\{K_{i,t} \geq 0\} \cdot T_i + Controls + \varepsilon_{i,t} \tag{2a}$$

$$y_{i,t} = \alpha_{T_i} + \delta_t + \beta\{K_{i,t} \geq 0\} \cdot T_i + Controls + \varepsilon_{i,t} \tag{2b}$$

where β represents the average treatment effect for the first 15 years (0 to 14) of the treatment. *Controls* stands for $\boldsymbol{\gamma}'_{K_{i,t}}\mathbf{X_i} + \boldsymbol{\lambda}'\mathbf{Z_{i,t}}$. To summarize treatment dynamics, I also split the post-testing period into three 5-year periods (year 0 to 4, 5 to 9, and 10 to 14),

$$y_{i,t} = \alpha_i + \delta_t + \beta_s\{K_{i,t} \in [0,4]\} \cdot T_i + \beta_m\{K_{i,t} \in [5,9]\} \cdot T_i + \beta_l\{K_{i,t} \in [10,14]\} \cdot T_i + Controls + \varepsilon_{i,t} \tag{3a}$$

$$y_{i,t} = \alpha_{T_i} + \delta_t + \beta_s\{K_{i,t} \in [0,4]\} \cdot T_i + \beta_m\{K_{i,t} \in [5,9]\} \cdot T_i + \beta_l\{K_{i,t} \in [10,14]\} \cdot T_i + Controls + \varepsilon_{i,t} \tag{3b}$$

where β_s, β_m, and β_l identify the short-, medium-, and long-term treatment effects, respectively. *Controls* stands for $\boldsymbol{\gamma}'_{K_{i,t}}\mathbf{X_i} + \boldsymbol{\lambda}'\mathbf{Z_{i,t}}$.

I study treatment heterogeneity in Models 2a-2b and 3a-3b by partitioning the sample into sub-samples. Finally, when I study differential treatment effects across age groups, I interact the Post*Treated indicator $\{K_{i,t} \geq 0\} \cdot T_i$ in Models 2a-2b with age group indicators $A_{i,t}$. I also include the interaction of $A_{i,t}$ with the relative year in $\gamma_{K_{i,t},i}$.

Attrition – Although by its nature administrative data do not suffer from attrition problems, attrition does arise due to death, emigration, and most importantly in my design, due to cancer diagnoses and preventive surgeries. Because I aim to separate the effects of current bad health from expectations of future health shocks, I only keep individuals in my sample as long as their health is unimpaired by Lynch Syndrome. I exclude all observations after an individual has developed cancer or has undergone preventive surgery. As Figure A6 in Appendix A reveals, by 14 years after genetic testing about 30% of the positive-tested individuals are removed from the sample due to having cancer or preventive surgeries. Excluding these individuals might introduce attrition bias. On the other hand, as Table A3 in Appendix A presents, my main estimates change only slightly when I do not to exclude people who had cancer (panel B) or those who had cancer or preventive surgeries (panel C).

Staggered Difference-in-Differences – A recent literature highlights that two-way fixed effects estimators may yield biased estimates of the average treatment effect if treatment effects

are heterogeneous over time or across units (De Chaisemartin and D'Haultfoeuille, 2022). At the heart of the problem are "forbidden comparisons" between outcomes of earlier and later treated units (Goodman-Bacon, 2021). Several authors propose alternative estimators that are based on the comparison of treated units with never-treated, not-yet-treated or last-treated units (Callaway and Sant'Anna, 2021; Sun and Abraham, 2021). This problem is not applicable to my setup because my regression models do not compare earlier and later treated individuals. In each regression, I control for the years passed since the genetic test, i.e., I compare positive and negative tested individuals who got tested exactly K years ago. Controlling for the years passed since treatment is not possible in standard staggered difference-in-differences models because the time period of treatment is undefined for the never-treated (control) units. In my set-up there are no classical control (never-treated) units, instead individuals receive one of two types of treatment in the year of testing, i.e., they are either tested positive or negative.

3.2 Data

This paper uses administrative microdata from the Netherlands, which I enrich with data on genetic testing for Lynch Syndrome from the Dutch Hereditary Cancer Registry. This subsection presents an overview of the main data sources and variables used in the paper. For a detailed description see Appendix B. Table B1 in Appendix B provides summary statistics for the main dependent variables.

The Dutch Hereditary Cancer Registry – My data on people at risk of Lynch Syndrome, the genetic tests they undertake, their cancer diagnoses, and the preventive surgeries they undergo come from the Dutch Hereditary Cancer Registry administered by The Netherlands Foundation for the Detection of Hereditary Tumors (www.stoet.nl). The registry was established in 1985 by a collaborative group of physicians with an interest in hereditary colorectal cancer. Its main goals are to promote the identification of families with various forms of hereditary cancer, including Lynch Syndrome, and to encourage high-risk individuals to participate in medical surveillance programs.

Being established before the discovery of the genes responsible for Lynch Syndrome, during its initial phase of operation the DHCR registered individuals who were at risk of Lynch Syndrome based on clinical criteria (personal and family history of cancer). Starting from the family members already in scope, genetic fields workers drew up family trees (pedigrees), identified other at-risk family members, and provided information on cancer surveillance options. Family members were prompted to register with the DHCR by signing a written consent form. The discovery of the major gene defects responsible for most of the hereditary cancer syndromes

during the 1990s profoundly changed the identification of Lynch Syndrome families and family members at risk. Diagnosis shifted from using clinical criteria to testing for genetic mutations in the Lynch genes. On the one hand, this enabled a more precise diagnosis of LS for individuals with cancer. On the other hand, it also contributed to the better identification of LS families and made predictive testing at the individual level possible.

Once genetic testing became available, many registered participants decided to undergo testing. Crucially, because these individuals had already registered with the DHCR, administrators strived to obtain their genetic test results regardless of the test outcome. This ensures a balanced sample of positive and negative tested individuals among people who registered with the DHCR before genetic testing.

Genetic testing - Information on the date/year of the genetic test, the tested gene (MLH1, MSH2, MSH6, EPCAM, or PMS2.), and the test outcome (mutation carrier or non-carrier). Figure A1 in Appendix A presents the distributions of the year of testing and the age at testing in the sample of 890 individuals.

Cancer diagnoses and preventive surgeries - Cancer diagnoses (date, classification code) and preventive surgeries (date, type of surgery) are reported either by the registered individual, the individual's physicians (general general practitioner, medical specialists), or the individual's family members. As the DHCR establishes strong relations with the registered families, their general practitioners, and their medical specialists, the registry receives regular updates on the cancer screenings, cancer diagnoses, and preventive surgeries of registered individuals.

Administrative Data – Statistics Netherlands (SN) offers a broad set of microdata files that can be matched at the individual level using pseudo-anonymized identifiers. External datasets can be matched to the existing collection of microdata files securely. Matching requires identifying information in the form of either a social security number (BSN number) or the combination of date of birth, sex, and address details. Due to the high-quality identifying information, about 98% of all individuals in the DHCR who underwent DNA testing following registration could be matched to the SN microdata files.

Demographics - Information on age, sex, household composition, partners (married and non-married), and children, among others. Address information (pseudo-anonymized).

Labor outcomes - Pre-tax labor earnings are available for the whole Dutch working population from 1999. From 2001 on the number of full-time equivalent days worked is also available. For a random sample of about 1/3rd of all Dutch households, pre-tax labor earnings are also available between 1995 and 1998.

Household income - Disposable household income for the whole Dutch population from 2003,

and for a 1/3rd random sample between 1995 and 2002. Disposable household income is the sum of the gross personal income (pre-tax labor income, entrepreneurial income, transfers such as unemployment, sickness, disability insurance benefits, pension benefits, social security benefits, housing allowance, alimony) of all household members plus household-level income (income from wealth, and some subsidies received at the household level such as child-related subsidies) reduced with alimony and other transfers paid at the household level and taxes on income and wealth.

Household balance sheets - From 2006 on Statistics Netherlands collects annual microdata on all Dutch households' wealth, including information on assets (financial assets, financial securities, primary residence, other real estate, entrepreneurial capital, substantial interests, and other assets) and debts (mortgage and other). These data are collected either from income tax declarations (wealth is taxed above a certain exemption amount) or from registers of financial institutions that are directly linked to the tax authorities and/or Statistics Netherlands (e.g. stock ownership registry). The level of observation is the household, the wealth of partners is aggregated. My main dependent variable among balance sheet items is financial assets, the sum of bank deposits/savings and financial securities. In my baseline specification, I winsorize financial assets at the 1^{st} and 99^{th} percentiles to reduce the influence of extreme asset values. I also use the log of financial assets as a dependent variable. In addition, I construct a measure of financial assets scaled to the mean household disposable income of the individual during the sample period.

Homeowner - Indicator based on the household income files and ownership status files. Available for the whole Dutch population from 1999.

Stock market participation and share of risky financial assets - A household is assumed to participate at the stock market if they have a non-zero holding of financial securities[19]; the share of risky financial assets is defined as the ratio of financial securities to total financial assets (including bank and savings accounts). In my baseline specifications, I only consider household-year observations if the household has at least EUR 2,500 in bank deposits/savings (i.e. non-risky financial assets).

Savings rate - One minus the ratio of household-level consumption and household disposable income. Household-level consumption is derived from the accounting identity that total household spending is equal to income plus capital gains minus the change in wealth over the

[19] Financial securities might also include direct bond holdings and investment in mutual funds that partially invest in safe assets; however, the share of safe assets in total financial securities appears to be limited. Using detailed survey data, Gaudecker (2015) finds that only 5% of Dutch households with financial securities do not own any shares or mutual funds but instead own only bonds or options, and that the majority of mutual funds held are equity funds. Using data from Statistics Netherlands, I estimate that only 17% of households with financial securities in 2011 received any interest payments from bonds.

period (Eika, Mogstad, and Vestad, 2020). I correct for capital gains on financial securities using national account data on the mutation in stocks and bonds due to financial transactions and due to changing prices, following Ji, Teulings, and Wouterse (2019). For the principal residence, if a homeowner household does not change address and continues to own its home, I assume that all value changes are from capital gains. In case a homeowner household moves but stays a homeowner, I assume that capital gains for the whole year are proportional to the growth rate of home values in the municipality of origin. If a homeowner household becomes a renter or a renter household becomes a homeowner, I assume that it earns capital gains for the fraction of the year it was a homeowner based on the growth rate of home values in the municipality of origin.

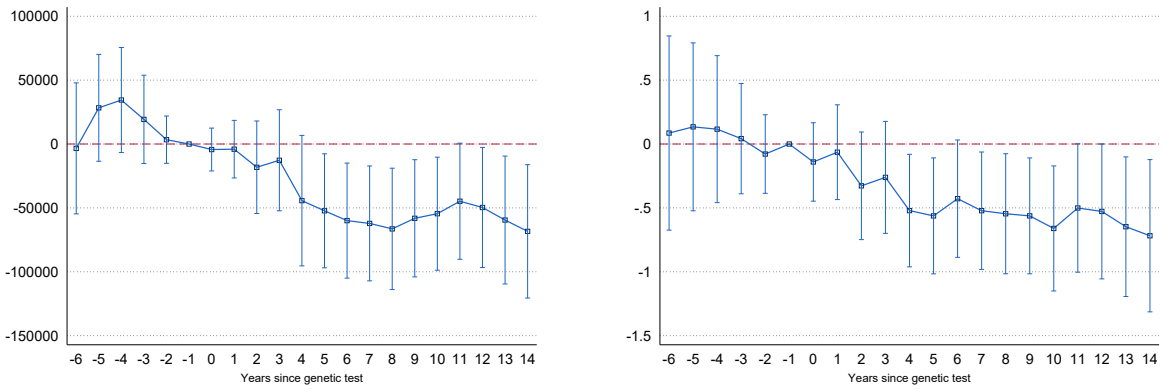
For other real estate, I assume zero capital gains if the households moves from not owning any other real estate to owning any, or vice-versa. If the household continues to own other real estate, I assume all year-on-year value changes up to 15% of the base year value to be capital gains, following Ji, Teulings, and Wouterse (2019). I assume that capital gains on savings accounts, entrepreneurial wealth, substantial interests, and other assets can be neglected. For additional sample selection criteria and trimming/winsorization see Appendix B Table B3.

4 Results on Wealth Accumulation

I start my analysis by documenting the effects of Lynch Syndrome on household wealth accumulation. It is important to keep in mind that the treatment effects presented below are differences between the outcomes of positive- and negative-tested individuals. Both groups may learn from their genetic test results and both may change their behaviors afterwards. The differences between the outcomes of the two groups identify the causal effects of the risks associated with LS.

Figure 4 presents dynamic treatment effect estimates from Model 1b. The figure reveals a strong negative effect on households' financial assets. Following genetic testing, households of positive-tested individuals accumulate lower financial assets than those of negative-tested individuals: five years after testing they have about EUR 50,000 (panel a) or 39% ($exp(-0.5)-1$) (panel b) lower financial assets. Financial assets include both bank deposits and financial securities (stocks, bonds, and investments in funds). After the initial 5 years, the difference between the two groups appears to stabilize, although as the wide confidence bounds suggest, treatment dynamics should be interpreted cautiously.

While financial assets constitute a large part of the household balance sheet, genetic testing may affect other balance sheet items as well. Table 2 presents treatment effect estimates on



(a) Financial assets (EUR)

(b) Log financial assets

The figure shows the dynamic effects of testing positive on financial assets (panel a) and log financial assets (panel b). Coefficient estimates from Model 1b are presented. The x-axis shows the year relative to the year of the genetic test. The figure presents 95% confidence intervals based on standard errors clustered at the individual level.

Figure 4: Dynamic treatment effects on financial assets and log financial assets

the different items of the household balance sheet. I decompose household net wealth into four components, (1) financial assets, plus (2) net real estate (the sum of all real estate minus the mortgage on the primary residence), plus (3) other assets (business wealth, additional assets incl. cash), minus (4) other debt (student loans, consumer loans etc.).[20]

Column (1) of Table 2 summarizes the treatment effects on financial assets in a single difference-in-differences coefficient (DiD) and three time-period specific coefficients based on Models 2b and 3b, respectively. The DiD coefficient shows that the year-end financial assets of positive-tested individuals are on average EUR 60,000 lower in the period after testing (years 0 to 14), compared to the financial assets of negative-tested individuals and the period before testing. This is an economically significant effect equal to about 80% of the sample mean of financial assets (EUR 77,000). The three time-period specific coefficients show the average treatment effect for years 0 to 4, 5 to 9, and 10 to 14, respectively. These coefficients reflect the dynamics presented in panel (a) of Figure 4, a large negative effect during the first years with subsequent stabilization. Columns (2) and (3) study the two components of financial assets separately: Both bank deposits and financial securities (stocks, bonds, and investments in funds) are negatively affected. Column (4) reports no effects on a binary indicator of homeonwership. Columns (5) to (7) study other net wealth components, net real estate, other assets, and other debt. None of these other wealth components are statistically significantly affected by genetic testing,

[20]Net real estate is the largest wealth element of the households in my sample (mean: EUR 135,000, median: EUR 65,000), followed by financial assets (mean: EUR 77,000, median: EUR 24,000), other assets (mean: EUR 61,000, median: EUR 0), and other debt (mean: EUR -23,000, median: EUR 0).

Table 2: Treatment effects on wealth components

	(1) Financial assets (EUR)	(2) Deposits (EUR)	(3) Securities (EUR)	(4) Home owner (binary)	(5) Net real estate (EUR)	(6) Other assets (EUR)	(7) Other debt (EUR)
DiD	-60,126***	-25,142***	-28,479**	-.0066	-12,462	-2,694	3,815
	(19,829)	(8,481)	(12,072)	(.025)	(26,137)	(33,428)	(10,218)
t=0-4	-31,091**	-11,998	-16,010	-.0012	-7,142	13,121	2,266
	(15,828)	(7,445)	(9,773)	(.024)	(21,275)	(23,084)	(7,240)
t=5-9	-71,817***	-25,751***	-36,402***	-.0074	-10,038	-18,700	-1,466
	(22,590)	(9,096)	(13,720)	(.029)	(28,326)	(37,034)	(12,572)
t=10-14	-66,435***	-33,151***	-27,997**	-.017	-18,639	4,472	10,647
	(22,690)	(9,981)	(13,703)	(.033)	(31,552)	(40,087)	(12,323)
Cons	94,538***	55,430***	33,024***	.78***	138,655***	61,983***	21,953***
	(9,946)	(4,048)	(5,873)	(.007)	(10,431)	(14,517)	(4,612)
Ind	826	826	826	857	826	826	826
N	8,752	8,752	8,752	12,501	8,752	8,752	8,752

The table presents the differential changes in wealth components following genetic testing for positive-tested individuals compared to negative-tested individuals. Financial assets (column 1) are the sum of deposits (2) and financial securities (3). Financial securities include direct stock holdings, investments in funds, but may also include bond holdings. Homeowner (4) is a binary indicator of owning a primary residence. Net real estate (5) is the value of all real estate holdings minus the mortgage on the primary residence. Other assets (6) include entrepreneurial wealth, private businesses, declared cash holdings etc. Other debt (7) includes educational loans, bank account overdrafts, consumer durable loans, tax debt etc. All dependent variables, besides Homeowner, are winsorized at the 1^{st} and 99^{th} percentiles. The row *DiD* reports the difference-in-differences coefficient β from Model 2b, which estimates the treatment effects using all periods (from -6 to 14). The rows *t=0-4*, *t=5-9*, and *t=10-14* report the coefficients β_s, β_m, β_l from Model 3b, receptively. These coefficients represent the treatment effects in different years after genetic testing. *Cons* reports the constant, *N* stands for the number of individual-year observations, while *Ind* represents the number of unique individuals in the sample. The sample includes individual-year observations when the individual is at least 25 years old and when they are classified by Statistics Netherlands as the household head or the partner thereof. Standard errors clustered at the level of the individual are reported in parentheses, * p < 0.1, ** p < 0.05, *** p < 0.01

although we can observe some negative impact on net real estate wealth. In summary, Table 2 presents evidence that the reduced life expectancy associated with LS has a negative effect on financial asset accumulation, while it does not significantly affect other wealth components.

The baseline effect of a EUR 60,000 lower financial asset accumulation is an average treatment effect across all tested individuals. As the distribution of financial assets is skewed, even after winsorization, it is well possible that the large negative effect is due to large treatment effects on the right tail of the financial assets distribution. Panel A of Table 3 estimates quantile treatment effects on financial assets based on the non-linear difference-in-differences method applied among others by Havnes and Mogstad (2015). The quantile treatment effects show how the specific quantiles of the outcome variable changed due to the treatment, i.e., due to

testing positive compared to testing negative.[21] As the panel reveals, it is indeed the highest quantiles of the financial assets distribution that are the most negatively affected in EUR terms. However, lower quantiles of the distribution are also negatively impacted: The median financial asset holdings among the households of positive-tested individuals are EUR 9,000 lower compared to the median of negative-tested households. These results suggest that genetic testing does not only affect the financial asset accumulation of the richest households.

Panel B of Table 3 estimates the treatment effects on two alternative transformations of financial assets, and also presents results from three robustness tests. Column (1) shows the estimated treatment effects on the natural logarithm of financial assets. The mean effect of -0.52 suggests that households of positive-tested individuals have about 40% ($exp(-0.52) - 1$) lower financial assets in the post-testing period, compared to households of negative-tested individuals and the period before testing. Column (2) show estimates where the dependent variable is financial assets scaled by the household's mean disposable income in the sample period. The treatment effects are also large under this scaling: In the period after testing, positive-tested households on average hold financial assets equal to a 72 percentage points (pp.) lower share of their household income than negative-tested households. Columns (3) to (5) present results from three robustness tests. In column (3), I do not winsorize financial assets at the 1^{st} and 99^{th} percentiles as in column (1) of Table 2. Instead, I exclude the top 1% of individuals with the highest mean financial assets in the sample period (9 individuals), and use the unwinsorized values of financial assets. I observe strong negative effects and similar treatment dynamics under this specification as well. Column (4) is identical to the baseline specification but controls for individuals fixed effects (Models 2a and 3a) instead of group fixed effects. The results hardly change under this specification, although the standard errors are larger. Column (5) alters the baseline specification by controlling for indicators of having a partner and having any children. As the results show, the treatment effects are only slightly reduced in this specification. This suggests that changing household composition, as documented below in Section 5, can only account for a small part of the negative effects on financial asset accumulation.

[21] Havnes and Mogstad (2015) use non-linear DiD methods to estimate how a child care reform affected the outcome distribution of exposed children as adults. Their main method, the one I adopt, is based on the unconditional quantile regressions of Firpo, Fortin, and Lemieux (2009). I use the Stata command *rifhdreg* to compute the quantile treatment effect estimates based on this method.

Table 3: Treatment effects on financial asset quantiles, alternative measures, and robustness

Panel A: Unconditional Quantile Regressions

	(1) 10th (EUR)	(2) 25th (EUR)	(3) Median (EUR)	(4) 75th (EUR)	(5) 90th (EUR)
DiD	-1,284	-3,070**	-8,948***	-29,698***	-137,662***
	(918)	(1,365)	(3,231)	(8,201)	(35,568)
Cons	2,493***	8,405***	26,725***	71,271***	208,322***
	(295)	(438)	(1,088)	(3,367)	(12,226)
N	8,752	8,752	8,752	8,752	8,752
Ind	826	826	826	826	826

Panel B: Alternative measures of financial assets and robustness

	(1) Log financial assets	(2) Scaled to income	(3) Excluding richest 1% (EUR)	(4) Individual f.e. (EUR)	(5) Partner control (EUR)
DiD	-.52**	-.72***	-40,343***	-58,018**	-57,759***
	(.21)	(.25)	(14,005)	(25,945)	(19,804)
t=0-4	-.31*	-.39*	-18,482	-47,302**	-30,065*
	(.19)	(.22)	(11,775)	(22,942)	(15,742)
t=5-9	-.55**	-.78***	-46,083***	-72,502**	-69,125***
	(.22)	(.26)	(16,222)	(30,887)	(22,446)
t=10-14	-.63**	-.87***	-48,460***	-68,498**	-63,442***
	(.25)	(.28)	(16,477)	(29,893)	(22,799)
Cons	10***	1.4***	76,890***	96,008***	93,879***
	(.084)	(.11)	(7,182)	(8,403)	(9,880)
N	8,608	8,676	8,661	8,752	8,731
Ind	823	818	817	813	825

The table presents the differential changes in financial assets following genetic testing for positive-tested individuals compared to negative-tested individuals. Panel A shows treatment effects on the quantiles of the financial assets distribution. These estimates are based on the non-linear difference-in-differences method used among others by Havnes and Mogstad (2015), which applies the unconditional quantile regressions of Firpo, Fortin, and Lemieux (2009). Panel B shows two specifications based on alternative transformations of financial assets, and presents results from three robustness tests. Log financial assets (1) are the natural logarithm of financial assets. Scaled to income (2) refers to financial assets divided by mean household disposable income in the sample period (winsorized at the 1^{st} and 99^{th} percentiles). The dependent variable in columns 1 to 3 is financial assets. Excluding richest 1% (3) excludes the individuals within the top 1% of the mean financial assets distribution (over the whole sample period). This column uses non-winsorized values of financial assets. Individual f.e. (4) controls for individual fixed effects instead of group (positive-tested) fixed effects. Partner control (5) controls for indicators of having a partner and any children. The row *DiD* reports the difference-in-differences coefficient β from Model 2b, which estimates the treatment effects using all periods (from -6 to 14). The rows *t=0-4*, *t=5-9*, and *t=10-14* report the coefficients β_s, β_m, β_l from Model 3b, receptively. These coefficients represent the treatment effects in different years after genetic testing. *Cons* reports the constant, *N* stands for the number of individual-year observations, while *Ind* represents the number of unique individuals in the sample. The sample includes individual-year observations when the individual is at least 25 years old and when they are classified by Statistics Netherlands as the household head or the partner thereof. Standard errors clustered at the level of the individual are reported in parentheses, * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Finally, Table 4 studies treatment heterogeneity along three dimensions, gender, age at testing, and having any children before testing. Panel A shows treatment effect estimates on financial assets in EUR, panel B studies financial assets in logs, while panel C is based on a measure which scales financial assets by mean household income. Columns (1) and (2) split the sample into the sub-sample of males and females, respectively. Although none of the differences are statistically significant[22], treatment effects appear to be larger for males along all three panels. A possible explanation is that Lynch Syndrome affects male life expectancy more negatively: the Lynch-affected fathers of individuals in my sample lost on average 14 years of their lives due to LS, while the Lynch-affected mothers lived 9 years shorter than the general population. Columns (3) and (4) split the sample into those who test at an older and those who test at a younger age than the median age at testing (41), respectively. While panel A shows a slightly larger negative effect on the financial asset accumulation of the latter group, this difference is hardly present in the other panels. Columns (5) and (6) split the sample into those how have children before testing and those who do not have any children, respectively. One could argue that individuals with children should be less sensitive to news about their life expectancy, as they save not only for their retirement but may also have bequest motives. Indeed, the results in all three panels point to weaker treatment effects for people who had children before testing. Still, the previous caveat applies: the sample is not large enough to statistically identify these differences.

5 Channels

This section considers possible channels that may explain the documented negative effects of Lynch Syndrome on financial wealth accumulation. I consider four main sets of explanations. First, I investigate whether Lynch Syndrome affects household composition and childbearing. A different propensity to have a partner may mechanically affect the level of financial assets in my data because balance sheets are aggregated at the household level. Household composition may also impact wealth accumulation by shifting preferences. Next, I explore the treatment effects on labor income and household income. With a sufficiently high savings rate, lower income may quickly translate into lower wealth accumulation. Then I proceed to study the treatment effects on the composition of financial portfolios and on the share of income that households save. Lower financial wealth may also be the result of a more conservative portfolio allocation, especially in the long run. On the other hand, lower savings rates (higher consumption rates)

[22] In general, my sample is not large enough to detect statistically significant treatment heterogeneity, and the results in Table 4 should be treated as indicative. The sole case in the paper where treatment heterogeneity is statistically significant is for female and male labor income in Table 7.

Table 4: Treatment heterogeneity on the change in financial assets

	(1) Male	(2) Female	(3) >41 before test	(4) ≤ 41 before test	(5) Had child before test	(6) No child before test
Panel A: Financial assets (EUR)						
DiD	-63,553**	-48,977	-47,393**	-66,403***	-48,716**	-108,529***
	(28,132)	(31,462)	(22,563)	(20,909)	(21,043)	(41,847)
Ind	378	446	410	414	598	225
N	3,959	4,793	4,454	4,298	6,404	2,347
Panel B: Log financial assets						
DiD	-.63*	-.47	-.42	-.45	-.49**	-.87*
	(.33)	(.3)	(.28)	(.29)	(.23)	(.46)
Ind	377	445	410	412	598	223
N	3,885	4,723	4,386	4,222	6,311	2,296
Panel C: Financial assets scaled to mean household income						
DiD	-.91**	-.56*	-.5	-.54***	-.53*	-1.5**
	(.45)	(.32)	(.37)	(.18)	(.28)	(.64)
Ind	376	441	408	409	591	225
N	3,936	4,740	4,436	4,240	6,328	2,347

The table presents the differential changes in financial assets following genetic testing for positive-tested individuals compared to negative-tested individuals. The table studies treatment heterogeneity in three pairs of sub-samples formed by gender (columns 1 and 2), the age at testing (columns 3 and 4), and the status of having children before testing (columns 5 and 6). Panel A shows estimates on financial assets in EUR (winsorized at the 1^{st} and 99^{th} percentiles). Panel B shows estimates on the natural logarithm of financial assets. Panel C shows estimates on financial assets scaled by mean household disposable income (winsorized at the 1^{st} and 99^{th} percentiles). The row *DiD* reports the difference-in-differences coefficient β from Model 2b, which estimates the treatment effects using all periods (from -6 to 14). *N* stands for the number of individual-year observations, while *Ind* represents the number of unique individuals in the sample. The sample includes individual-year observations when the individual is at least 25 years old and when they are classified by Statistics Netherlands as the household head or the partner thereof. Standard errors clustered at the level of the individual are reported in parentheses, * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

can affect wealth accumulation immediately. Lastly, I consider changes in the mental health of tested individuals as an alternative explanation.

5.1 Household Composition

Changes in household composition might impact the outcomes that I study, especially in the long run. Most economic decisions may be correlated with family composition, including labor supply and entrepreneurship, retirement (Heyma, 2004), consumption and savings decisions (Browning and Ejrnæs, 2009; De Nardi et al., 2021), homeownership (Bacher, 2021), and financial portfolio allocation (Calvet and Sodini, 2014; Hubener, Maurer, and Mitchell, 2016). In addition, family

composition also mechanically affects wealth levels in my data because in Statistics Netherlands' datasets wealth data are aggregated at the household level.

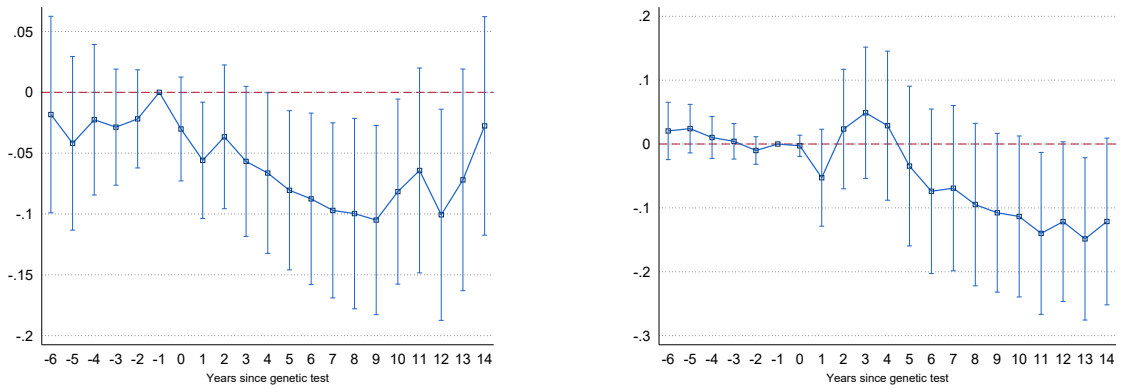
Carrying a Lynch Syndrome gene mutation may affect household composition in multiple ways. First, patient testimonies suggest that positive-tested individuals might find it more difficult to find a partner.[23] Second, childbearing might also be affected. Positive-tested individuals might be afraid of passing down the faulty gene to their children. Prenatal testing can help to eliminate this risk by identifying whether the mutation is present in the embryo; however, on average only every second embryo will be free of the mutation. Also, some individuals prefer not to undergo prenatal testing. Female mutation carriers are also recommended to undergo preventive surgeries at an early age, which might shorten their reproductive period. These different considerations of childbearing might in turn affect the probability of having a partner.

Partnerships – Although some survey-based research (e.g., Dewanwala et al., 2011) and anecdotal evidence suggest that Lynch Syndrome patients might face difficulties with family and relationship formation, to the best of my knowledge, no prior study has quantified the effects of genetic testing among pre-symptomatic individuals. Panel (a) of Figure 5 presents dynamic treatment effect estimates on having a partner. I restrict my sample to those who test under the age of 46. I choose this cut-off age because it is mostly in the earlier life when individuals form partnerships. As Figure 5 illustrates, positive-tested individuals face an immediate reduction in the probability of having a partner following genetic testing, compared to negative-tested individuals and the year before testing. The negative effect grows over time, reaching about 10 pp by year 10; however, we can observe some recovery in the long run.[24]

Table 5 summarizes the treatment effects on partnership formation using Models 2a and 3a, and presents treatment heterogeneity in sub-samples. Contrary to panel (a) of Figure 5, column (1) considers all individuals in my sample, also those who were tested in an older age. The results show a much milder negative effect on having a partner, which can be explained by the lack of effect among individuals who tested at an older age (column 3) compared to those who tested when younger (column 2). In columns 4 to 7, I again restrict the sample to the group of individuals who tested when 45 years old or younger. Columns (4) and (5) report no substantially different treatment effects among women and men. Columns (6) and (7) split the sample into individuals who had a partner before testing and those who did not have one, respectively. As the results show, both groups appear to be negatively affected, although most coefficient estimates are not statistically significant.

[23] See for example: https://www.thecut.com/2018/05/how-to-date-when-youre-waiting-for-cancer.html

[24] I find similar initial effects but a much milder recovery in case I do not censor my sample at the time of the first preventive surgery. This suggests that positive-tested individuals without a partner might be more likely to undertake preventive surgeries and select out of the sample.



(a) Having a partner

(b) Having any children

The figure shows the dynamic effects of testing positive on having a partner (panel a) and on having any children (panel b). Coefficient estimates from Model 1a are presented. The x-axis shows the year relative to the year of the genetic test. Both panels (a) and (b) only include individuals who underwent genetic testing younger than 46 years old. In addition, panel (b) only considers individuals who had no children before testing. The figure presents 95% confidence intervals based on standard errors clustered at the individual level.

Figure 5: Dynamic treatment effects on having a partner and having any children

Childbearing – Being diagnosed with a Lynch mutation may also influence the propensity to have children at least for three reasons. First, as presented above, positive-tested individuals in the reproductive age (here defined as 45 years old or younger) face a lower probability of having a partner following testing. Second, Lynch mutation carriers have a 50% probability of passing the faulty gene to their children, which may discourage some from childbearing. Third, female mutation carriers are recommended to undergo hysterectomy (surgical removal of the womb) and risk-reducing salpingo-oophorectomy (removal of fallopian tubes and ovaries) after the age of 40, and/or once they do not wish to have (more) children. This might prompt some women to have children earlier, or to have fewer children (Dewanwala et al., 2011). To date, no study have examined whether mutation status consciously influences reproductive choices among pre-symptomatic (cancer-free) Lynch mutation carriers (Corrado et al., 2021).

As panel (b) of Figure 5 shows, being diagnosed with a Lynch mutation indeed influences reproductive choices among previously childless reproductive-age individuals in my sample. Positive-tested individuals appear to shift their childbearing earlier in the years following genetic testing. However, in the long run they have a substantially lower probability of ever having children. While about 50% of the negative-tested childless individuals who get tested before the age of 46 will eventually have children, this ratio is 12 pp. lower among positive-tested individuals.

Column (1) of Table 6 summarize the results presented in panel (b) of Figure 5. Columns

Table 5: Treatment effects on having a partner

				≤ 45 before test			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	All	≤ 45 before test	> 45 before test	Male	Female	Had partner before test	No partner before test
DiD	-.028	-.048*	.027	-.039	-.053	-.039	-.014
	(.021)	(.027)	(.032)	(.041)	(.035)	(.025)	(.076)
t=0-4	-.0072	-.028	.037	-.023	-.031	-.03	.014
	(.019)	(.024)	(.027)	(.037)	(.031)	(.023)	(.074)
t=5-9	-.048*	-.072**	.019	-.061	-.072*	-.054*	-.065
	(.026)	(.033)	(.043)	(.052)	(.042)	(.031)	(.088)
t=10-14	-.038	-.05	.016	-.036	-.065	-.033	.014
	(.031)	(.038)	(.049)	(.055)	(.054)	(.037)	(.1)
Cons	.79***	.8***	.79***	.76***	.82***	.93***	.34***
	(.005)	(.0078)	(.005)	(.013)	(.0097)	(.0071)	(.024)
Ind	889	555	333	238	316	409	130
N	15,510	9,816	5,694	4,365	5,451	7,379	2,305

The table presents the effects of testing positive for the suspected Lynch syndrome gene mutation on having a partner at the end of the year (binary indicator).The row *DiD* reports the coefficient β from Model 2a, which is the average treatment effect after genetic testing. The rows *t=0-4*, *t=5-9*, and *t=10-14* report the coefficients β_s, β_m, β_l from Model 3a, receptively. These coefficients represent the treatment effects in different years after genetic testing. *Cons* reports the constant, *N* stands for the number of individual-year observations, while *Ind* represents the number of unique individuals in the sample. The sample includes individual-year observations when the individual is at least 20 years old. Column (1) includes all observations that meet this criterium. Columns (2) and (3) restrict the sample to individuals who underwent genetic testing not older than 45 years or older than 45 years, respectively. Columns (4) to (7) only consider individuals who tested not older than 45 years. Columns (4) and (5) split the sample of these individuals into males and females. Columns (6) and (7) include individuals who had a partner in the year before testing, and those who had no partner, respectively. Standard errors clustered at the level of the individual are reported in parentheses, * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

(2) and (3) present the treatment effects separately for males and females, respectively. The estimates suggests that the childbearing of males is more negatively affected, although these results should be interpreted very cautiously due to the small number of individuals in the sub-samples (109 and 99, respectively). Finally, column (4) considers tested individuals who already had at least one child before testing, and estimates the treatment effects on the number of children they have. As the results reveal, this intensive margin of childbearing is little affected.

Effects on wealth accumulation – Can the changing household composition documented in this section account for the lower wealth accumulation of Lynch-positive households? The results previously presented in Table 3 suggest otherwise: controlling for an indicator of having a partner and having children only slightly reduces the treatment effect estimate on financial asset accumulation (from EUR 60,000 to EUR 58,000). This is as expected given that in the

Table 6: Treatment effects on having children

	(1)	(2)	(3)	(4)
	All	Male	Female	# children (had child before)
DiD	-.064	-.084	-.048	-.027
	(.044)	(.061)	(.067)	(.059)
t=0-4	.0018	.0014	-.0064	-.027
	(.038)	(.044)	(.066)	(.049)
t=5-9	-.083	-.1	-.072	-.034
	(.059)	(.08)	(.088)	(.065)
t=10-14	-.14**	-.17*	-.089	-.018
	(.062)	(.09)	(.087)	(.075)
Cons	.25***	.24***	.27***	2.1***
	(.015)	(.021)	(.022)	(.015)
Ind	209	109	99	330
N	3,831	2,057	1,774	6,340

The table presents the effects of testing positive for the suspected Lynch syndrome gene mutation on having children. The row *DiD* reports the coefficient β from Model 2a, which is the average treatment effect after genetic testing. The rows *t=0-4*, *t=5-9*, and *t=10-14* report the coefficients β_s, β_m, β_l from Model 3a, receptively. These coefficients represent the treatment effects in different years after genetic testing. *Cons* reports the constant, *N* stands for the number of individual-year observations, while *Ind* represents the number of unique individuals in the sample. The sample includes individual-year observations when the individual is at least 20 years old. Columns (1) to (3) are based on the sample of individuals who had no children before genetic testing and who underwent testing no older than 45 years. The dependent variable in these columns is a binary indicator of having any children. Column (4) is based on the sample of individuals who *had children before genetic testing* and who underwent testing no older than 45 years. The dependent variable in this column is the number of children an individual has. Standard errors clustered at the level of the individual are reported in parentheses, * p < 0.1, ** p < 0.05, *** p < 0.01

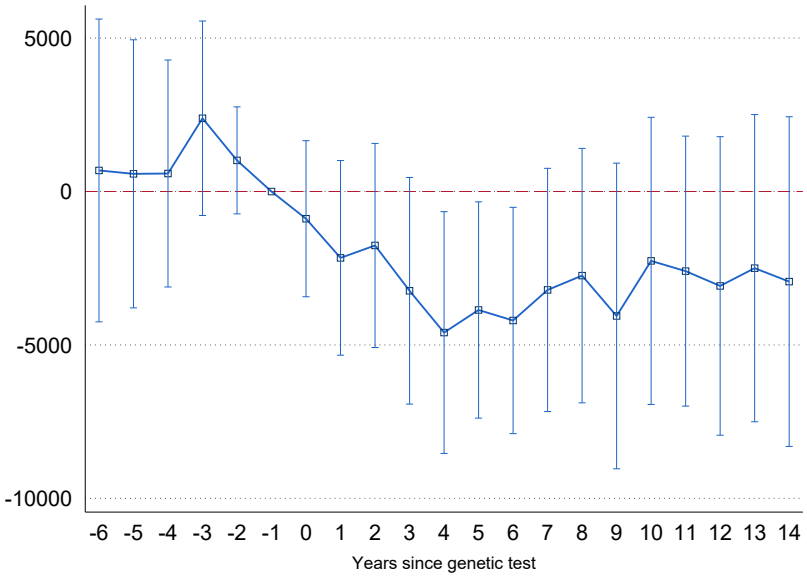
whole sample the probability of having a partner is only slightly negatively affected (-2.8 pp.), while childbearing is only negatively impacted for about $1/4^{th}$ of the sample who had no children before testing and who tested in a childbearing age.

5.2 Income

Next, I turn to estimating the impact of testing positive for Lynch Syndrome on individuals' labor and household income. A lower income may translate into the lower financial wealth accumulation documented in Section 4 as long as household save a sufficiently high share of their income.

Labor income – Figure 6 presents the dynamic effects of testing positive on labor income. Labor income is equal to the gross salary for individuals who work, while it is set to zero for non-workers. As the figure illustrates, the treatment effects on labor income are immediate and rather persistent. Four years after testing, positive-tested individuals earn on average close

to EUR 5,000 lower labor income compared to negative-tested individuals and the year before testing. This is an economically meaningful (18%) reduction compared to the mean labor income of EUR 28,000 in the sample. Although we can observe some recovery in the long run, the treatment effect is still negative (albeit not statistically significant) 14 years after testing.



The figure shows the dynamic effects of testing positive on labor income (EUR). Labor income is set to zero for non-working individuals. Coefficient estimates from Model 1a are presented. The x-axis shows the year relative to the year of the genetic test. The figure presents 95% confidence intervals based on standard errors clustered at the individual level.

Figure 6: Dynamic treatment effects on labor income

Table 7 studies heterogeneity in the treatment effects on labor income, and also separates these effects into extensive and intensive margin components. Column (1) summarizes the effects presented in Figure 6, based on Models 2a and 3a. The results show that positive-tested individuals have on average EUR 3,720 lower labor income in the period after testing compared to negative-tested individuals and the period before testing. This is an economically significant (13%) reduction compared to the previously cited EUR 28,000 mean labor income in the sample. Columns (2) and (3) estimate the treatment effects for males and females, respectively. The results reveal a striking difference: while male labor income is reduced by EUR 8,400 (or 21% compared to the mean labor income in the sample), female labor income is hardly affected. This difference is also statistically significant in a regression where I interact the DiD indicator with an indicator for gender.[25]

What could explain this striking difference? One possibility is the historically stronger labor market attachment of men in the Netherlands.[26] On the other hand, the difference in the treat-

[25] Results are available upon request.

[26] This differential labor force attachment is also present in my sample. In the year before testing, 82% of the

ment effect stays nearly unchanged if in the above mentioned regression with the DiD*Gender interaction term I also control for interaction terms of the DiD indicator and indicators of (1) working full-time, (2) having a partner, and (3) having children.[27] It appears that the gender difference in the treatment effect on labor income is not driven by the different labor force attachment of males and females during the sample period. Another possible explanation is that men react stronger to genetic testing because male life expectancy is more severely affected by Lynch Syndrome. As previously discussed, the Lynch-affected fathers of individuals in my sample lived on average 14 years shorter than the general population, while the Lynch-affected mothers lost on average 9 years of their lives due to LS.

The last four columns of Table 7 study treatment effects on the extensive and intensive margin components of male labor income.[28] The results in column (4) reveal a negative, albeit not statistically significant, effect on the probability of working. As the estimates in column (5) show, positive-tested males also earn a relatively lower labor income if they work (labelled here as 'salary'). Thus both the intensive and the extensive margins of labor income appear to be negatively affected. The last two columns further split salary into the full-time equivalent days worked (column 6) and the wage (column 7). While wages are hardly affected, positive-tested individuals work on average 19 fewer FTE days in the period after testing compared to negative-tested individuals and the period before testing. This is a non-negligible 5.7% relative effect compared to the mean 335 annual FTE days among all working men in the sample.[29] As Table A5 in Appendix A shows, it is in the pre-retirement period when the negative effect on working men's labor supply is the largest. Between the ages of 60 and 64, working positive-tested men work on average 42 fewer FTE days annually than working negative-tested men (a 15.5% relative effect). In summary, the negative treatment effect on male labor income arises from a negative effect on labor supply, i.e., a lower probability to work and/or less time worked.

Household income – While labor income is an important outcome to study on its own, it is *household* income that ultimately matters for *household* wealth accumulation. Table 8 summarizes the treatment effects on disposable household income and its two main components, the tested person's and their partner's labor income. All coefficients in Table 8 are difference-in-

tested men and 65% of the tested women worked. Conditional on working, women were also more likely to work part-time: while women worked on average 235 FTE days in the year before testing, men worked 332 FTEs. These differences were also reflected in the labor income of men (EUR 36,600) and women (EUR 14,000).

[27] Results are available upon request.

[28] Table A4 in Appendix A presents the decomposition of the treatment effect on female labor income into the same components. The results show that although in euro terms female labor income is little affected, positive-tested females were on average 5.7 pp. less likely to work in the post-testing period, compared to negative-tested females and the pre-testing period.

[29] In the datasets of Statistics Netherlands, an employee who is employed on a full-time contract throughout the year works 365 or 366 FTE days.

Table 7: Treatment effects on labor income

	Labor income			Decomposition of male labor income			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	All	Male	Female	Working	Salary	FTE day	Daily wage
	(EUR)	(EUR)	(EUR)	(binary)	(EUR)	(days)	(EUR)
DiD	-3,720**	-8,412**	-654	-.032	-6,122*	-19**	1.7
	(1,703)	(3,513)	(1,166)	(.04)	(3,172)	(8.7)	(7.1)
t=0-4	-3,368**	-7,548**	-668	-.046	-4,085*	-12	3.3
	(1,566)	(3,315)	(1,072)	(.038)	(2,470)	(9)	(6.8)
t=5-9	-4,386**	-9,797**	-776	-.033	-8,126**	-28***	1.5
	(2,054)	(4,115)	(1,407)	(.048)	(4,020)	(10)	(8.6)
t=10-14	-3,406	-8,012*	-430	.0018	-7,922*	-19*	-2.5
	(2,388)	(4,657)	(1,963)	(.055)	(4,771)	(10)	(9.5)
Cons	29,058***	42,998***	17,322***	.82***	51,505***	342***	146***
	(498)	(1,050)	(336)	(.012)	(956)	(2.7)	(2.3)
Ind	788	366	422	366	337	322	322
N	11,768	5,458	6,310	5,458	4,458	3,800	3,800

The table presents the effects of testing positive for the suspected Lynch syndrome gene mutation on labor income. *Labor income* (columns 1 to 3) equals to the pre-tax salary if an individual is working and to zero otherwise. *Working* (column 4) is an indicator whether the individual had non-zero pre-tax salary in the given year. *Salary* (column 5) stands for pre-tax salary (set to missing if zero). *FTE days* (column 6) are the number of full-time equivalent days the individual worked in the given year (set to missing if zero). Daily wage (column 7) is *Salary* divided by *FTE days*. The row *DiD* reports the coefficient β from Model 2a, which is the average treatment effect after genetic testing. The rows *t=0-4*, *t=5-9*, and *t=10-14* report the coefficients β_s, β_m, β_l from Model 3a, receptively. These coefficients represent the treatment effects in different years after genetic testing. *Cons* reports the constant, *N* stands for the number of individual-year observations, while *Ind* represents the number of unique individuals in the sample. All samples include individuals who underwent genetic testing at the age of 60 or younger and individual-year observations when the individual is between 25 and 64 years old. Standard errors clustered at the level of the individual are reported in parentheses, * p < 0.1, ** p < 0.05, *** p < 0.01

differences estimates based on Model 2a. In each model, I also control for an indicator of having a partner to account for the previously documented negative treatment effect on the probability of having a partner.[30] Partner's labor income equals the labor income of the individual's partner if they have one, otherwise it equals zero. The table presents two sets of estimates. Columns (1) to (3) consider the whole sample, while columns (4) to (6) exclude person-year observations where disposable household income is above the 99^{th} percentile across the whole sample.[31] Both sets of estimates present evidence that the household income of males are more negatively affected than the household income of females. This is mostly due to the previously

[30] A further difference from Table 7 is that for each model I restrict my sample to individuals who are the household heads of their household or the partners thereof. This additional sample selection criterium, and controlling for having a partner, explain the slight differences in the treatment effect estimates on labor income in Tables 7 and 8.

[31] This exclusion mostly concern instances of high non-labor income (e.g. capital income) and/or the highest income households, and substantially reduces the volatility of the dependent variable (and the standard error of the estimates).

discussed larger negative effect on male labor income. The results also suggest that not only tested individuals' own labor income are negatively impacted but also the labor income of their partners. However, treatment effects on the partners' labor income are mostly not statistically significant. Restricting our attention to columns (4) to (6), Table 8 also illustrates that the drop in household income is approximately equal to the sum of the negative effects on own labor income and on the partner's labor income (while keeping in mind that a unit decrease in *gross* labor income will decrease *disposable* household income by less than a unit due to taxation).

Table 8: Treatment effects on household income and its components

	Full sample			$< 99^{pctl}$ household income		
	(1)	(2)	(3)	(4)	(5)	(6)
	All	Male	Female	All	Male	Female
Household income (EUR)	-9,770**	-12,733**	-8,262	-3,821**	-6,068**	-2,459
	(4,184)	(6,201)	(6,226)	(1,542)	(2,734)	(1,839)
Own labor income (EUR)	-3,402**	-7,706**	-847	-3,446*	-7,128*	-1,456
	(1,671)	(3,478)	(1,196)	(1,953)	(4,203)	(1,349)
Partner's labor income (EUR)	-5,129*	-873	-7,805	-2,039	-752	-2,346
	(2,961)	(1,472)	(4,945)	(1,580)	(1,824)	(2,378)
Ind	761	350	410	761	350	410
N	9,784	4,468	5,316	9,684	4,436	5,248

The table presents the effects of testing positive for the suspected Lynch syndrome gene mutation on household income, the tested person's labor income, and the labor income of the tested person's partner. *Household income* refers to disposable income, which is the sum of all labor, and non-labor income (including transfers and capital income) of the household minus taxes paid. *Labor income* equals to the pre-tax salary if the individual is working and to zero otherwise. *Partner's labor income* is recorded as zero if the tested person had no partner in the given year or if the partner was not working. All cells report the coefficient β, which is the average treatment effect after genetic testing, from independent regression models 2a. *N* stands for the number of individual-year observations, while *Ind* presents the number of unique individuals in the sample. Both values refer to the row *Household income*. The samples of columns (1) to (3) include individuals who underwent genetic testing at the age of 60 or younger and individual-year observations when the individual was between 25 and 64 years old and was the household head or partner. The samples of columns (4) to (6) exclude individual-year observations when the tested person's household income was in the top 1 percentile across the whole sample. Standard errors clustered at the level of the individual are reported in parentheses, * p < 0.1, ** p < 0.05, *** p < 0.01

Effects on wealth accumulation – The estimated negative effects on disposable household income are far from negligible. In relative terms, they are equal to an 18.5% (whole sample) or an 8% (restricted sample) reduction in annual disposable household income. On the other hand, it is unlikely that this lower household income, ceteris paribus, substantially contributed to the reduced financial wealth accumulation documented in Section 4. This is because Dutch households save a low share of their disposable household income: The Dutch Bureau for Economic Policy Analysis estimates that between 2006 and 2017 Dutch household saved on average

-0.3% of their annual household income (this refers to private savings, households saved a much larger share of their income directly in the pension system).[32] I estimate similarly low savings rates in my sample, with a mean of 0% and a median of 2%. Finally, I also estimate a regression model where I augment the baseline model in column (1) of Table 2 with the cumulative disposable household income of the individual's household since 1995.[33] The results (available upon request) reveal that cumulative household income is a highly statistically significant determinant of financial assets (with a coefficient of 0.23); however, the treatment effect estimate is hardly affected (EUR -58,000).

5.3 Financial Investments and Savings

Besides changing household composition and reduced household income, lower financial wealth accumulation may also be explained by a more conservative portfolio allocation and by a lower savings rate.

Portfolio allocation – Panel (a) of Figure 7 presents the dynamic effects of testing positive on the risky share of financial assets. The risky share equals Financial Securities divided by total Financial Assets.[34] As the figure illustrates, the treatment effects on the risky share are immediate and rather persistent. In the year after testing, households of positive-tested individuals hold an about 6 pp. lower share of their financial assets in financial securities compared to households of negative-tested individuals and the year before testing. This is a substantial reduction in comparison with the mean risky share of 12% in the sample. The negative effect largely persists in the first 15 years after genetic testing.

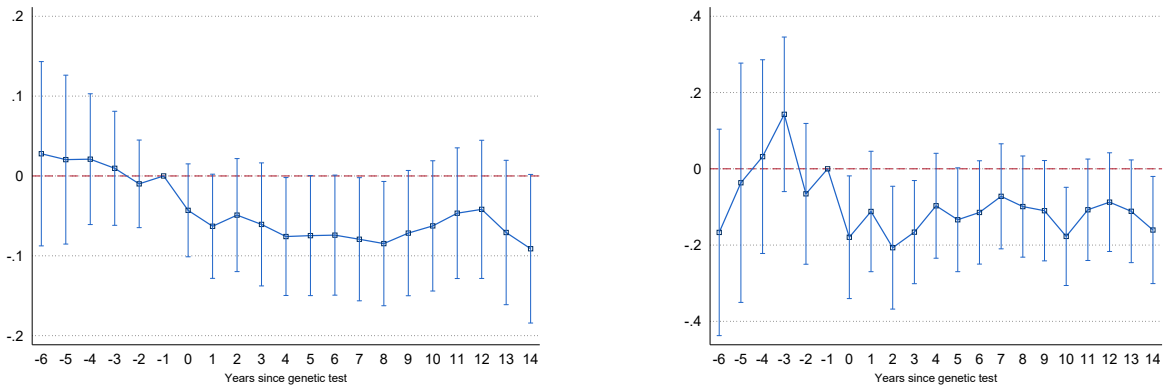
Table 9 further studies the treatment effects on the risky share. Column (1) summarizes the effects presented in panel (a) of Figure 7, based on Models 2b and 3b. The results show that households of positive-tested individuals hold on average a 9 pp. lower share of their financial assets in financial securities in the period after testing, compared to households of negative-tested individuals and the period before testing. Columns (2) and (3) split the treatment effect into an extensive margin (having any financial securities) and an intensive margin (risky share conditional on having financial securities) component, respectively. The estimates suggest that the negative effect on the risky share arises from a large negative effect on stock market participation (not having any financial securities).[35]

[32] Kerngegevens voor Nederland, 1970-2017, Centraal Planbureau.

[33] I replace household income with 0 in the years when it is missing or if the individuals is not classified as the household head or the partner thereof.

[34] Financial securities might also include direct bond holdings and investment in mutual funds that partially invest in safe assets, although the share of these assets are likely very limited. See the discussion in footnote 19 in Section 3.

[35] Table A7 in Appendix A presents various robustness tests on the treatment effect on the risky share.



(a) Risky share of financial assets

(b) Savings rate out of disposable income

The figure shows the dynamic effects of testing positive on the risky share of financial assets (panel a) and on the savings rate out of disposable income (panel b). Coefficient estimates from Model 1b are presented. The x-axis shows the year relative to the year of the genetic test. The figure presents 95% confidence intervals based on standard errors clustered at the individual level.

Figure 7: Dynamic treatment effects on the risky share of financial assets and on the savings rate

Notwithstanding the large negative treatment effect on the risky share, a back-of-the-envelope calculation suggests that a reduced risky share can only account for a small part of the lower financial asset accumulation documented in Section 4. Multiplying the average financial assets of households in my sample (EUR 77,000) by the negative treatment effect on the risky share (9 pp.) and by an equity risk premium of 6% yields an estimate of EUR 415 annual returns foregone due to the lower risky share. This is a small amount compared to the total negative effects on financial assets (about EUR 50,000 five years after genetic testing).

Savings rate – The previous analyses suggest that although changing household composition, reduced household income, and a lower risky share may all contribute to the documented negative effects on financial asset accumulation, the role of these channels is likely limited. This suggests that the most important channel through which genetic testing affects the wealth accumulation of tested individuals is altered consumption and savings behavior. In this sub-section, I present some direct evidence that households of positive-tested individuals indeed save a lower share of their disposable income in the period after genetic testing compared to households of negative-tested individuals and the period before testing.

Panel (b) of Figure 7 presents the dynamic effects of testing positive on the savings rate out of disposable household income. I define the savings rate as the ratio of savings and household disposable income. Savings refer to active savings, i.e., change in net wealth less unrealized capital gains or losses. Disposable income does not include unrealized capital gains. As I do not directly observe unrealized capital gains or losses, I need to make some (possibly

Table 9: Treatment effects on the risky share of financial assets and on the savings rate

	Risky share			Savings rate
	(1)	(2)	(3)	(4)
	All	Extensive margin	Intensive margin	All
DiD	-.09**	-.15**	-.02	-.12***
	(.038)	(.077)	(.061)	(.039)
t=0-4	-.083**	-.12	-.037	-.15***
	(.036)	(.073)	(.062)	(.042)
t=5-9	-.1**	-.16**	-.041	-.1**
	(.039)	(.08)	(.064)	(.043)
t=10-14	-.084**	-.17**	.021	-.13***
	(.042)	(.085)	(.071)	(.042)
Cons	.14***	.37***	.37***	.034***
	(.013)	(.027)	(.022)	(.012)
Ind	807	807	368	690
N	7,723	7,723	2,500	5,703

The table presents the effects of testing positive for the suspected Lynch syndrome gene mutation on risky financial investments and on savings out of disposable household income. *Risky share* (columns 1 to 3) is risky financial securities divided by total financial assets. Risky financial securities comprise mostly stocks and investments in mutual funds but might also include bonds. In column (1) the dependent variable is the risky share. In column (2) the dependent variable is an indicator of having a non-zero risky share. In column (3) the dependent variable is the risky share conditional that it is non-zero. *Savings rate* (4) is defined as savings divided by disposable household income. Savings is imputed from year-on-year changes in household wealth corrected for capital gains on housing and financial investments. Disposable household income is the sum of all labor and non-labor income (including transfers and capital income) of the household minus taxes paid. The row *DiD* reports the coefficient β from Model 2b, which is the average treatment effect after genetic testing. The rows *t=0-4*, *t=5-9*, and *t=10-14* report the coefficients β_s, β_m, β_l from Model 3b, receptively. These coefficients represent the treatment effects in different years after genetic testing. *Cons* reports the constant, *N* stands for the number of individual-year observations, while *Ind* represents the number of unique individuals in the sample. The sample includes individual-year observations when the individual was at least 25 years old and when they were classified by Statistics Netherlands as the household head or the partner thereof. Columns (1) to (3) include only observations when the individual had at least EUR 2,500 in bank deposits or savings. Column (3) include only observations where the risky share is non-zero. Standard errors clustered at the level of the individual are reported in parentheses, * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

strong) assumptions to calculate my savings rate measure, the details of which are presented in Section 3 and in Appendix B. Consequently, the results presented in this sub-section should be interpreted with caution. As the figure illustrates, the treatment effects on the savings rate are immediate and rather persistent, although in many cases long-term effects cannot be statistically distinguished from zero. The strongest effects appear to take place during the first four years after genetic testing. During this period, households of positive-tested individuals save on average a 15 to 20 pp. lower share of their disposable income compared to households of negative-tested individuals and the year before testing. This is a very large reduction in saving (or increase in dis-saving) given the mean savings rate in the sample (0%). Column (4) of Table 9 summarizes the effects presented in panel (b) of Figure 7. The results suggest that in the period after genetic testing, households of positive-tested individuals save on average a 12 pp. lower share of their disposable income compared to households of negative-tested individuals and the period before testing. This is a very substantial effect, although the magnitude of this estimate should be treated with caution as it is sensitive to the trimming applied when constructing the savings rate. For example, trimming the savings rate below at -100% (instead of the baseline -150%) reduces the estimate to -8 pp. (statistically significant at the 5% level).[36]

5.4 Mental Health

Although Lynch Syndrome has no negative impact on the physical health of positive tested individuals before developing cancer, a positive test result might still cause mental distress. Aktan-Collan et al. (2013) evaluate the long-term psychosocial consequences of predictive genetic testing in LS and report that 7 years following testing, most of the psychosocial variables remain unchanged, regardless of mutation status. Nevertheless, the authors find a moderate increase in fear of death among positive tested individuals relative to those who tested negative. Galiatsatos et al. (2015) conduct a literature review on the psychosocial impact of LS testing and conclude that LS mutation carriers suffer a transient increase in depression and anxiety scores post-disclosure, which seem to normalize by 6-12 months. I also study the effects of testing positive for a Lynch Syndrome mutation on the mental health of the individuals in my sample (Table A1 in Appendix A). As the results in columns (1) and (2) of Table A1 show, positive-tested individuals if anything are slightly less likely to consume antidepressants and anti-anxiety medication following genetic testing.

Despite the evidence on the limited prevalence of mental health problems among LS-affected people, it is well possible that genetic testing for LS leads to some forms of mental distress. It is

[36] Table A6 in Appendix A presents various robustness tests on the treatment effect on the savings rate.

unlikely that people update their beliefs on mortality and cancer risks without being emotionally affected. However, the cause of such mental distress would likely be the increased risk of cancer and the reduced life expectancy in Lynch Syndrome. In other words, while mental health problems may moderate the responses that individuals give to genetic testing, these mental problems, if present, are likely an integral part of the channel of changing life expectancy.

6 The Effects of Genetic Testing

Finally, I study how positive- and negative-tested individuals react to learning their mutation carrier status compared to a counterfactual where they do not receive this information. Estimating the effects of undergoing genetic testing is important to determine the costs and benefits of testing. Nevertheless, this exercise is hindered by endogeneity problems. Genetic testing is a choice: demographic factors (age, gender, parenthood, level of education, employment, participation in medical studies), psychological factors (lack of depressive symptoms), and family history (greater number of relatives with cancer) are positively associated with the uptake of genetic testing (Hampel, 2016).

Contrary to my baseline study, I lack a natural experiment that randomizes people into tested and non-tested groups. Instead, I apply a matching strategy and use individuals from the general Dutch population as a control group of untested individuals. Although matching on observables cannot alleviate all endogeneity concerns, the rich administrative data enables me to match on a broad range of characteristics, including the year of birth, gender, having a partner, the number of children, and homeownership.[37] As for the main analysis, I also include individual (or group) fixed effects in my regression models, which control for time-invariant unobservable differences between positive-tested, negative-tested, and untested individuals.

Results in Table 10 reveal that compared to the benchmark of the untested general Dutch population, both positive- and negative-tested individuals change their behavior following genetic testing. For example, the previously documented negative treatment effects on the probability of having a partner and having any children arise as a difference between positive effects on those who test negative and negative effects on those who test positive (columns 1 and 2).

[37] Table A2 in Appendix A compares tested individuals in my sample to the Dutch general population. Panel A only matches on birth year and gender. As discussed in footnote 8, tested individuals are more likely to have children and a partner, earn a somewhat higher household income, and have higher financial and non-financial wealth. Also, tested individuals are much more likely to have at least one parent who has already passed away. Panel B further matches on having a partner, the number of children, and homeownership in the year before testing. After matching, several characteristics that I do not match on also appear to be better balanced, such as financial assets, household income, or the indicator for working. On the other hand, tested individuals are still substantially more likely to have at least one parent who has already passed away. Given this, and potentially other, imbalances, the results in this section should be interpreted cautiously.

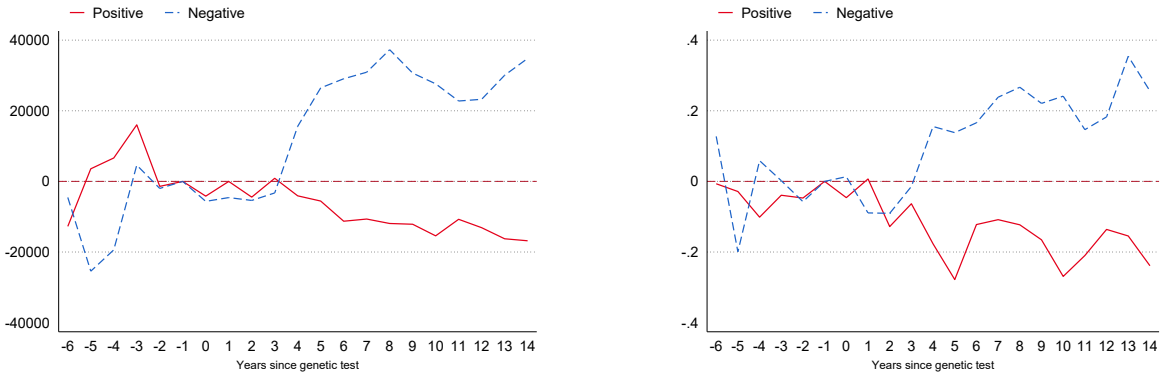
Table 10: Treatment effects compared to a matched sample of the Dutch general population

	(1) Has partner (binary)	(2) Has children (binary)	(3) Male labor inc. (EUR)	(4) Financial assets (EUR)	(5) Financial assets (logs)	(6) Risky share (ratio)	(7) Savings rate (ratio)
Positive	-.02	-.031	-3,124	-12,760	-.12	-.023	-.071**
	(.02)	(.031)	(2,711)	(9,420)	(.17)	(.029)	(.03)
Negative	.023	.045	1,060	28,153*	.19	.046**	.028
	(.016)	(.03)	(2,108)	(15,069)	(.14)	(.018)	(.027)
Cons	.77***	.23***	38,610***	59,912***	9.8***	.1***	.0058***
	(.00043)	(.00073)	(62)	(903)	(.013)	(.0017)	(.0014)
Ind	11,301	4,328	7,500	17,460	17,385	16,581	14,822
N	209,803	81,959	117,022	186,741	180,720	154,581	121,710

The table presents the effects of testing positive or negative for the suspected Lynch syndrome gene mutation compared to a baseline formed by a matched sample of the general Dutch population. Difference-in-differences estimates from models 2a or 2b are presented, with the models augmented to include two levels of treatment (positive-tested and negative-tested). *Has partner* (1) is a binary indicator if the individual had a (married or non-married) partner. *Has children* (2) is a binary indicator if the individual had any children. *Male labor income* (3) represents labor income, which equals the pre-tax salary if the individual was working and zero otherwise. *Financial assets* (4-5) refers to financial assets, which are the sum of bank deposits and savings, and risky financial securities (stocks, investments in mutual funds, and rarely bonds). *Risky share* (6) stands for the share of risky financial securities among total financial assets. *Savings rate* (7) is the savings rate, defined as savings divided by disposable household income. *Cons* reports the constant, *N* stands for the number of individual-year observations, while *Ind* presents the number of unique individuals in the sample. The sample inclusion criteria are the same as for previous tables where these variables appear. Standard errors clustered at the level of the individual are reported in parentheses, * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

In some cases negative-tested individuals appear to react stronger (financial assets, risky share, having children), whereas for other outcomes positive-tested individuals might be more affected (male labor income, savings rate). However, most estimates are not statistically significantly different from zero (from the outcomes of the benchmark group), therefore it is difficult to judge the magnitude of the relative effects.

Figure 8 illustrates the dynamic effects of genetic testing on financial assets (panel a) and log financial assets (panel b). Before testing, the financial assets of both positive- and negative-tested individuals evolve similarly to those of the benchmark group. On the other hand, following testing positive-tested (negative-tested) individuals accumulate lower (higher) financial assets. These dynamics are as expected: Positive-tested individuals receive bad news about their life expectancy, while negative-tested individuals receive good news.



(a) Financial assets (EUR)

(b) Log financial assets

The figure shows the dynamic effects of testing positive (solid red line) or negative (dashed blue line) on financial assets (panel a) and log financial assets (panel b), compared to a benchmark formed by a matched sample of the untested Dutch population. Coefficient estimates from Model 1b are presented. The x-axis shows the year relative to the year of the genetic test. The figure presents 95% confidence intervals based on standard errors clustered at the individual level.

Figure 8: Effects of genetic testing on financial assets

7 Conclusion

I merge genetic testing data and rich administrative data from the Netherlands to study the causal effects of life expectancy on individuals' financial, economic, and demographic decisions. My sample consists of people who start their life at a 50% risk of having inherited a gene mutation that causes Lynch Syndrome in their families. Lynch Syndrome is a hereditary disorder that substantially reduces life expectancy by increasing lifetime cancer risks. Individuals in my sample decide to undergo genetic testing to learn if they have indeed inherited the bad gene. Genetic testing randomizes tested persons into two groups. Those who test positive learn that they face a high risk of cancer and a shorter life expectancy. Those who test negative learn that their cancer risks are not elevated. Both groups may react to genetic testing: The differences in their reactions identify the causal effects of the life expectancy reduction in LS.

I find that reduced life expectancy has a negative effect on wealth accumulation: in the decades following testing, individuals who turn out to carry the gene mutation accumulate lower financial assets than those who learn that they are not affected by Lynch Syndrome. These findings are consistent with standard life-cycle models of savings and consumption, and provide evidence that lower (higher) life expectancy does prompt less (more) savings. Lower labor supply, changing household composition, and more conservative financial portfolios explain part of the negative effect on financial wealth accumulation, although the majority of the effect is due to lower savings rates.

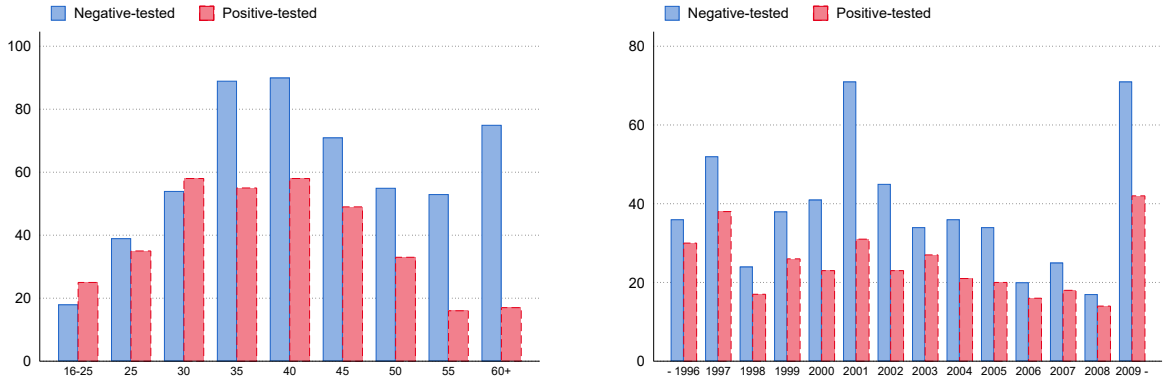
In a supplementary analysis, I find that both positive- and negative-tested individuals react to genetic testing. For example, those who test positive and experience a drop in life expectancy start to accumulate fewer financial assets. On the contrary, those who test negative and experience an increase in life expectancy start to accumulate more financial assets. I observe similar reactions for most of the other outcomes that I study, including having a partner and having children. These findings provide evidence that both bad and good news of life expectancy affect economic decisions.

The primary goal and contribution of my work is to understand the effects of life expectancy on economic behavior. However, I argue that my findings also contribute to the medical literature and can have direct policy implications. Understanding how people react to the results of genetic testing may be an important consideration for clinical geneticists and other medical professionals. This is especially true since predictive genetic testing might soon be offered for the general population[38], and many private providers (e.g., 23andMe) already offer testing for some of the more frequent single-gene genetic disorders (e.g., hereditary breast cancer). My results also highlight the potential benefits of genetic testing on negative-tested individuals: by alleviating health and mortality risks, genetic testing can help improve the socio-economic outcomes of these people.

My work also faces limitations. First, my results on the negative effects of reduced life expectancy on wealth accumulation provide qualitative but not necessarily quantitative support for standard life-cycle models. I am working on a realistically calibrated life-cycle model to compare the magnitude of my estimates to the model-implied ones. Second, due to data limitations, I cannot observe the effect of genetic testing on tested individuals' subjective mortality beliefs. Changes in mortality beliefs could help to estimate how tested individuals perceive the risks in Lynch Syndrome. This is an important input for any life-cycle model. In May 2023, I will conduct a survey among 1,500 people affected by Lynch Syndrome in collaboration with the Netherlands Foundation for the Detection of Hereditary Tumors. This survey may help shed light on how genetic testing affects the subjective beliefs of tested individuals.

[38] In late 2022, a nationally collaborative project was launched in Australia that will screen at least 10,000 people aged 18-40 for genes that increase risk of certain types of cancers (including Lynch Syndrome) and heart disease. Source: https://www.monash.edu/news/articles/world-first-preventative-dna-screening-for-cancer-and-heart-disease-risk2

A Additional Figures and Tables



(a) Age at testing

(b) Year of testing

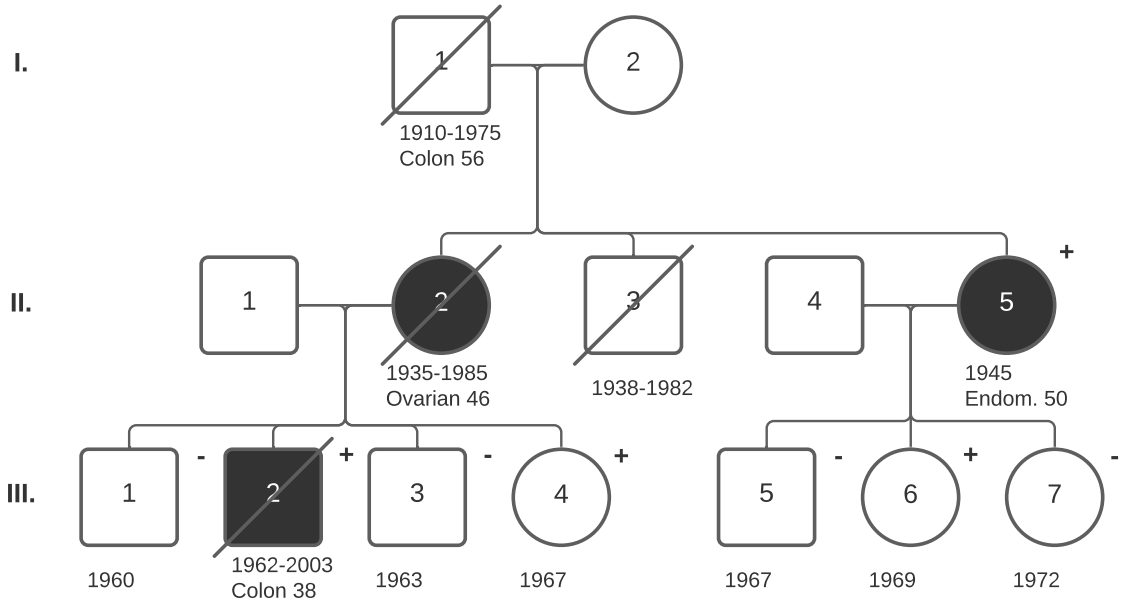
Figure A1: Distribution of positive- and negative-tested individuals by age at testing and year of testing

RESULT: The pathogenic MSH2 gene mutation (variant:...) documented in this family was excluded by two independent sequence analyses.
CONCLUSION: The tested individual is not a carrier of the family mutation in the MSH2 gene. As a result, the risk of colon and endometrial cancer of the person seeking advice and her progeny (descendants) has been reduced to that of the general population

Figure A2: Extract from a letter of a clinical geneticist to a tested individual explaining the *negative test result*

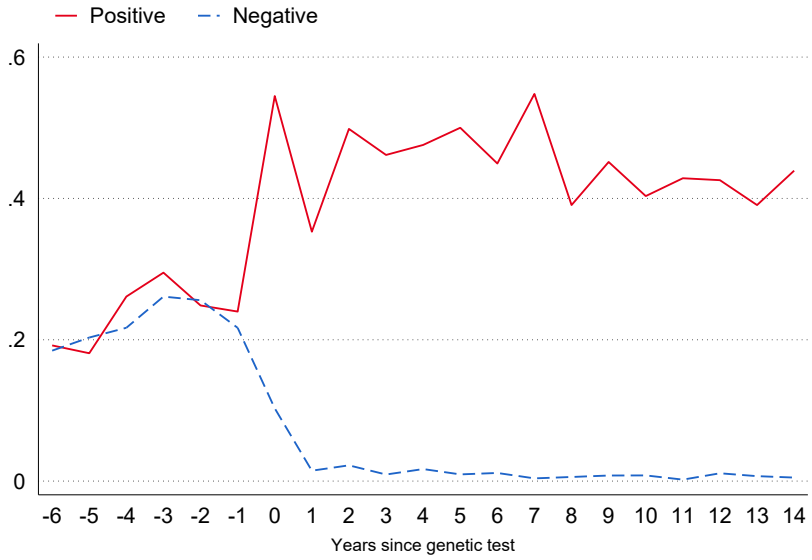
You were referred for genetic advice because several of your family members had colon cancer that DNA testing had shown is of a hereditary form. The DNA test shows that you also have this predisposition for HNPCC (also referred to as Lynch syndrome). People who have the predisposition (= the gene) for HNPCC have a high chance of developing colon cancer between the ages of 20 and 70. The age of cancer occurrence varies. Some people get colon cancer more than once. In rare cases, someone who has the HNPCC gene may still not have developed colon cancer by age 70. However, this chance is small, estimated at 5% (= 1 in 20). The other 95% of people with the gene will develop colon cancer sooner or later.
This means that it is necessary to regularly examine the intestine of people with this predisposition. This is can be done with a colon photo, or with a viewing device that is mounted in a flexible tube (colonoscopy). We recommend performing this examination once every 2 years...
The risk of uterine (endometrial) cancer is clearly increased in women. This risk is not known exactly, but it is estimated at 30%. Women with a predisposition to HNPCC are therefore advised to have an annual gynaecological examination with an ultrasound examination of the uterus...
The predisposition to HNPCC is inherited in an autosomal dominant manner. Autosomal means that both boys and girls can develop this condition. Dominant means that the altered aptitude is stronger than the normal aptitude... if one of the parents has the altered predisposition, they have a 50% (1 in 2) chance of having a child with this condition with each pregnancy. We therefore advise that children also have a DNA test after their 20th year to examine whether they have the predisposition.

Figure A3: Extract from a letter of a clinical geneticist to a tested individual explaining the *positive test result*



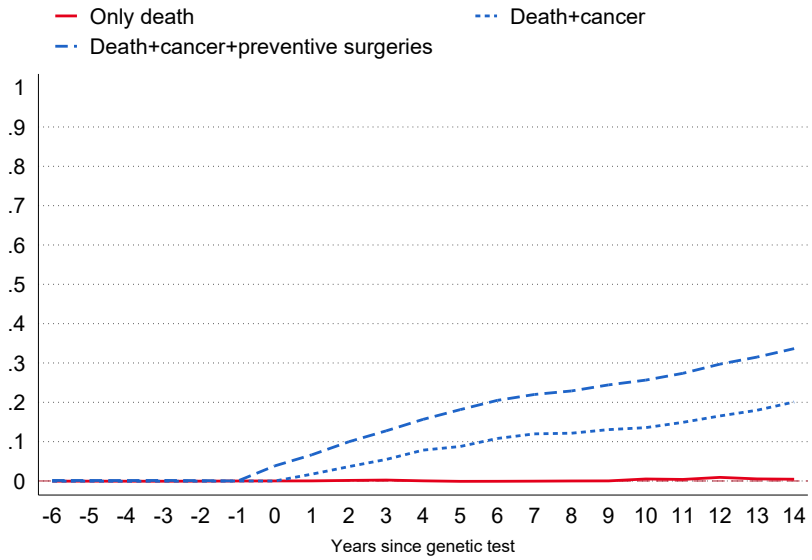
Legend: squares represent male, circles female family members; diagonal lines mark individuals who have passed away; the plus (+) signs identify tested mutation carriers, the minus (-) signs tested non-carriers; birth (and death) years, and the age of cancer diagnosis/cancer type are marked below the squares/circles. The roman numbers (I., II., and III.) refer to generations, the Arabic numbers identify individuals.
Notes: The figure presents cancer and genetic testing status in an actual family with some details altered to preserve privacy. In 1998, the gynecologist of family member III/7 referred the family to the attention of the DHCR due to suspected hereditary cancer in the family based on family history of cancer. The mother of the individual (II/5) had been previously diagnosed with endometrial cancer, her aunt (II/2) passed away following ovarian cancer, and her grandfather had colon cancer. Family members II/5, III/1, III/3, III/4 (but not III/2), and III/5-6-7 opted to register with the DHCR and undergo regular colonoscopy but decided not to undergo genetic testing (yet). In 2000, III/2 was diagnosed with colon cancer at the age of 38. Genetic testing identified a pathological MSH2 mutation, and this finding prompted other family members to undergo genetic testing. The three siblings of III/2 faced a 50% risk of inheriting the mutation as their brother was a proven mutation carrier, and eventually one of them tested positive (III/4). The surviving aunt of III/2 (II/5) faced a close to 100% probability of carrying the mutation given that III/2 most probably inherited it from the maternal side and II/5 had been diagnosed with endometrial cancer at a relatively young age. Following the positive test outcome of II/5, her children (III/5-6-7), who were now at 50% risk, were also offered the opportunity of genetic testing, and one of them (III/6) proved to carry the MSH2 mutation present in the family. Both positive and negative genetic test results were retained by the DHCR. The Lynch-affected individuals of the third generation, III/4, and III/6, undergo regular cancer screening and have not (yet) developed cancer.

Figure A4: Pedigree (family tree) of a Lynch-affected family



The figure shows the average annual value of a binary indicator of undergoing cancer screening (colonoscopy or gynaecological check-up) for positive-tested (solid red line) and negative-tested (dashed blue line) individuals.

Figure A5: Probability of undergoing colonoscopy or gynaecological cancer screening



The figure shows the excess probability that positive-tested individuals are excluded from the sample compared to negative-tested individuals. Regression coefficients are plotted based on Model 1a, where the dependent variable is an indicator of exclusion for death (solid red line), death or cancer (short-dashed blue line), and death, cancer or preventive surgeries (long-dashed blue line).

Figure A6: Excess attrition among positive-tested people due to death, cancer diagnosis and preventive surgeries

Table A1: Treatment effects on health outcomes

	(1) Antidepr.	(2) Tranq.	(3) Disabled	(4) Cancer screening
DiD	-.031	-.023	.019	.41***
	(.038)	(.028)	(.017)	(.015)
Cons	.095***	.062***	.059***	.09***
	(.015)	(.011)	(.0044)	(.0035)
Ind	818	818	854	889
N	8,206	8,206	12,894	16,499

The table presents the effects of testing positive for the suspected Lynch syndrome gene mutation on health outcomes. In column (1) the dependent variable is a binary indicator of being reimbursed for any antidepressants under the mandatory Dutch health insurance scheme. In column (2) the dependent variable is being reimbursed for any anti-anxiety medications. In column (3) the dependent variable is an indicator of receiving disability benefits. In column (4) the dependent variable is an indicator of participating in any cancer screenings (colonoscopies or gynaecological check-ups). The row *DiD* reports the coefficient β from Model 2b, which is the average treatment effect after genetic testing. *Cons* reports the constant, *N* stands for the number of individual-year observations, while *Ind* represents the number of unique individuals in the sample. Standard errors clustered at the level of the individual are reported in parentheses, * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A2: Tested individuals and the Dutch population

Panel A: Compared to a population matched on birth year and gender

Variable	Tested	S.e.	Mean	Tested	Population
Has child	0.04	0.01	0.68	875	42,002
Number of children	0.19	0.04	1.49	871	41,841
At least 1 parent died before testing	0.14	0.01	0.48	877	42,146
Has partner	0.06	0.01	0.73	866	41,557
Personal income (EUR)	313	1,051	31,188	397	18,345
Working	0.05	0.02	0.74	589	28,067
Disposable household income (EUR)	2,035	1,183	44,246	393	18,208
Financial assets (EUR)	9,098	7,399	51,136	204	9,420
Homeowner	0.09	0.02	0.65	551	25,976

Panel B: Additionally matched on having a partner, children and homeownership

Variable	Tested	S.e.	Mean	Tested	Population
Has child*	0.00	0.00	0.72	873	16,925
Number of children*	-0.02	0.02	1.71	870	16,907
At least 1 parent died before testing	0.14	0.01	0.48	874	16,954
Has partner*	0.00	0.00	0.80	865	16,838
Personal income (EUR)	-361	1,050	31,814	395	7,536
Working	0.03	0.02	0.75	589	11,270
Disposable household income (EUR)	-679	1,173	46,613	391	7,485
Financial assets (EUR)	2,091	7,357	60,372	205	3,883
Homeowner*	0.00	0.00	0.75	551	10,561

The table compares the pre-testing characteristics of tested individuals and two matched samples of the general Dutch population. In panel A, tested individuals are (exact) matched to similar individuals from the Dutch population based on birth year and gender. In panel B, (exact) matching also incorporates additional characteristics, having a partner, the number children and a binary indicator of homeownership. Both panels report coefficient estimates of regression models where individual and household characteristics (measured in the year before genetic testing) are regressed on an indicator of being in the tested sample. The regression controls for 'pair fixed effects', i.e., it compares tested individuals with their matched pairs. Robust standard errors are presented in the column 'S.e.'. The unconditional mean in the sample is presented in the column 'Mean'. 'Tested' refers to the number of tested individuals, which varies between variables due to the different sample periods (e.g., wealth variables for most individuals are only available from 2006) and the differences in the sample selection criteria. The same sample selection criteria apply as for Table 1. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A3: Main treatment effects under different sample selection criteria

	Financial assets (EUR)	Financial assets (logs)	Has partner (under 46) (binary)	Has child (under 46) (binary)	Household income (EUR)	Male labor inc. (EUR)	Savings rate (ratio)	Risky share (ratio)
Panel A: Baseline sample								
DiD	-60,126***	-.52**	-.048*	-.064	-10,463**	-8,412**	-.12***	-.09**
	(19,829)	(.21)	(.027)	(.044)	(4,221)	(3,513)	(.039)	(.038)
Panel B: No attrition due to preventive surgeries								
DiD	-59,356***	-.5**	-.046*	-.062	-8,996**	-7,802**	-.13***	-.089**
	(19,794)	(.21)	(.027)	(.044)	(4,095)	(3,464)	(.039)	(.038)
Panel C: No attrition due to cancer or preventive surgeries								
DiD	-56,061***	-.51**	-.048*	-.061	-8,421**	-7,501**	-.12***	-.083**
	(19,325)	(.21)	(.027)	(.044)	(4,005)	(3,500)	(.039)	(.036)
Panel D: Individuals certainly at 50% risk of mutation inheritance at birth								
DiD	-55,775***	-.6**	-.047*	-.06	-9,320**	-9,745***	-.11***	-.096**
	(21,077)	(.26)	(.027)	(.045)	(4,212)	(3,544)	(.04)	(.039)

The table presents the main treatment effect estimates of the paper under different sample selection criteria. Panel A repeats the baseline estimates. The samples in panel B do not exclude observations in the year of preventive surgeries and thereafter. The samples in panel C extend the samples in Panel B, as they also do not drop observations in the year of cancer diagnoses and thereafter. Panel D shows treatment effect estimates in a sample of individuals who were almost certainty at a 50% risk of inheriting Lynch Syndrome at birth. See footnote 9 for a discussion. *DiD* reports the coefficient β from Model 2a or 2b, which is the average treatment effect after genetic testing. *Cons* reports the constant, *N* stands for the number of individual-year observations, while *Ind* represents the number of unique individuals in the sample. The same sample restrictions apply as in the relevant tables in the main text. Standard errors clustered at the level of the individual are reported in parentheses, * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A4: Treatment effects on female labor income and its components

	(1) Labor income (EUR)	(2) Working (binary)	(3) Salary (EUR)	(4) FTE days (days)	(5) Daily wage (EUR)
DiD	-654	-.057*	-91	3.5	6.2
	(1,166)	(.032)	(1,416)	(11)	(4.8)
t=0-4	-668	-.046	-60	2.4	7.7*
	(1,072)	(.032)	(1,264)	(11)	(4.7)
t=5-9	-776	-.085**	-279	2.3	3.7
	(1,407)	(.039)	(1,679)	(12)	(5.7)
t=10-14	-430	-.037	149	9.4	5.8
	(1,963)	(.049)	(2,293)	(15)	(6.6)
Cons	17,322***	.71***	24,683***	229***	104***
	(336)	(.0092)	(424)	(3.5)	(1.6)
Ind	421	421	345	333	333
N	6,310	6,310	4,393	3,790	3,790

The table presents the effects of testing positive for the suspected Lynch syndrome gene mutation on labor income for females. *Labor income* (columns 1) equals to the pre-tax salary if an individual is working and to zero otherwise. *Working* (column 2) is an indicator whether the individual had non-zero pre-tax salary in the given year. *Salary* (column 3) stands for pre-tax salary (set to missing if zero). *FTE days* (column 4) are the number of full-time equivalent days the individual worked in the given year (set to missing if zero). Daily wage (column 5) is *Salary* divided by *FTE days*. The row *DiD* reports the coefficient β from Model 2a, which is the average treatment effect after genetic testing. The rows *t=0-4*, *t=5-9*, and *t=10-14* report the coefficients β_s, β_m, β_l from Model 3a, receptively. These coefficients represent the treatment effects in different years after genetic testing. *Cons* reports the constant, *N* stands for the number of individual-year observations, while *Ind* represents the number of unique individuals in the sample. All samples include individuals who underwent genetic testing at the age of 60 or younger and individual-year observations when the individual is between 25 and 64 years old. Standard errors clustered at the level of the individual are reported in parentheses, * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A5: Treatment effects on male labor income by age groups

	(1) Labor income (EUR)	(2) Working (binary)	(3) Salary (EUR)	(4) FTE day (days)
20-34	-2,762	-.069	2,069	4
	(4,268)	(.055)	(3,703)	(14)
35-49	-9,419**	-.037	-7,150*	-20**
	(4,358)	(.041)	(4,023)	(10)
50-59	-7,203*	-.024	-5,297	-18*
	(3,831)	(.055)	(3,329)	(9.8)
60-64	-10,509*	-.0082	-9,560*	-42**
	(6,124)	(.085)	(5,467)	(20)
Cons	42,801***	.83***	51,088***	341***
	(1,033)	(.011)	(908)	(2.8)
Ind	365	365	336	320
N	5,458	5,458	4,458	3,800

The table presents the effects of testing positive for the suspected Lynch mutation on male labor income and its constituents over the life-cycle, based on Model 2a where the difference-in-differences indicator is interacted with age groups. *Labor income* (columns 1) equals to the pre-tax salary if an individual is working and to zero otherwise. *Working* (column 2) is an indicator whether the individual had non-zero pre-tax salary in the given year. *Salary* (column 3) stands for pre-tax salary (set to missing if zero). *FTE days* (column 4) are the number of full-time equivalent days the individual worked in the given year (set to missing if zero). *Cons* reports the constant, *N* stands for the number of individual-year observations, while *Ind* represents the number of unique individuals in the sample. All samples include individuals who underwent genetic testing at the age of 60 or younger and individual-year observations when the individual is between 25 and 64 years old. Standard errors clustered at the level of the individual are reported in parentheses, * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A6: Robustness tests on the treatment effects on the savings rate

	(1) Baseline	(2) Partner control	(3) Including inheritance	(4) Individual f.e.
DiD	-.12***	-.12***	-.1***	-.09**
	(.039)	(.039)	(.038)	(.044)
t=0-4	-.15***	-.14***	-.12***	-.11**
	(.042)	(.042)	(.041)	(.045)
t=5-9	-.1**	-.1**	-.078*	-.058
	(.043)	(.042)	(.041)	(.049)
t=10-14	-.13***	-.13***	-.11***	-.088*
	(.042)	(.042)	(.041)	(.052)
Cons	.034***	.033***	.022*	.022
	(.012)	(.012)	(.012)	(.013)
Ind	690	690	691	679
N	5,703	5,702	5,719	5,703

The table presents robustness tests on the effects of testing positive for the suspected Lynch syndrome gene mutation on savings out of disposable household income. The dependent variable in all columns is the *Savings rate*, which is defined as savings divided by disposable household income. Savings is imputed from year-on-year changes in household wealth corrected for capital gains on housing and financial investments. Disposable household income is the sum of all labor and non-labor income (including transfers and capital income) of the household minus taxes paid. Column (1) repeats the baseline estimate from Table 9. Column (2) in addition controls for an indicator of having any children and an indicator of having a partner. Column (3) deducts gifts and inheritances received from the calculated savings. Data on gifts and inheritance are only available from 2007 on. Column (4) repeats the baseline but controls for individual fixed effects instead of group (positive-tested) fixed effects. The row *DiD* reports the coefficient β from Model 2b, which is the average treatment effect after genetic testing. The rows *t=0-4*, *t=5-9*, and *t=10-14* report the coefficients β_s, β_m, β_l from Model 3b, receptively. These coefficients represent the treatment effects in different years after genetic testing. *Cons* reports the constant, *N* stands for the number of individual-year observations, while *Ind* represents the number of unique individuals in the sample. The sample includes individual-year observations when the individual was at least 25 years old and when they were classified by Statistics Netherlands as the household head or the partner thereof. Standard errors clustered at the level of the individual are reported in parentheses, * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A7: Robustness tests on the treatment effects on the risky share

	(1) Baseline	(2) Individual f.e.	(3) Partner control	(4) No minimum deposits
DiD	-.09**	-.085***	-.083**	-.072**
	(.038)	(.031)	(.037)	(.035)
t=0-4	-.083**	-.083***	-.087**	-.066**
	(.036)	(.031)	(.035)	(.033)
t=5-9	-.1**	-.091***	-.086**	-.082**
	(.039)	(.033)	(.038)	(.037)
t=10-14	-.084**	-.073**	-.077*	-.065
	(.042)	(.035)	(.041)	(.04)
Cons	.14***	.14***	.14***	.14***
	(.013)	(.009)	(.013)	(.013)
Ind	806	778	823	806
N	7,723	7,723	8,809	7,675

The table presents robustness tests on the effects of testing positive for the suspected Lynch syndrome gene mutation on the risky share of financial assets. The dependent variable in all columns is the *Risky share*, which equals risky financial securities divided by total financial assets. Risky financial securities comprise mostly stocks and investments in mutual funds but might also include bonds. Column (1) repeats the baseline estimate from Table 9. Column (2) repeats the baseline but controls for individual fixed effects instead of group (positive-tested) fixed effects. Column (3) in addition controls for an indicator of having any children and an indicator of having a partner. Column (4) repeats the baseline but does not impose the requirement of having at least EUR 2,500 in bank deposits or savings. The row *DiD* reports the coefficient β from Model 2b, which is the average treatment effect after genetic testing. The rows *t=0-4*, *t=5-9*, and *t=10-14* report the coefficients β_s, β_m, β_l from Model 3b, receptively. These coefficients represent the treatment effects in different years after genetic testing. *Cons* reports the constant, *N* stands for the number of individual-year observations, while *Ind* represents the number of unique individuals in the sample. The sample includes individual-year observations when the individual was at least 25 years old and when they were classified by Statistics Netherlands as the household head or the partner thereof. Standard errors clustered at the level of the individual are reported in parentheses, * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

B Summary Statistics, Data Sources, and Variable Definitions

Variable	Age range	Age at test	Other sample criteria	N	Mean	S.d.	10^{th} pctl.	Median	90^{th} pctl.
Has a partner (0/1)	20≤			9,816	0.78	0.41	0.00	1.00	1.00
Has any children (0/1)	20≤			10,419	0.69	0.46	0.00	1.00	1.00
Number of children	20≤			10,327	1.48	1.22	0.00	2.00	3.00
Working (0/1)	25-64	≤60		11,768	0.75	0.43	0.00	1.00	1.00
Labor income (EUR)	25-64	≤60		11,768	27,993	30,049	0	24,196	59,706
Salary (labor income) (EUR)	25-64	≤60	working	8,851	37,218	29,278	9,114	33,406	65,043
Fulltime equivalent (FTE) days worked	25-64	≤60	working	7,590	283	103	120	338	366
Wage (EUR)	25-64	≤60	working	7,590	126	70	66	109	200
Patner labor earnings (0 if no partner) (EUR)	25-64	≤60	adult*	11,387	24,125	55,054	0	15,326	54,756
Disposable household income (EUR)	25-64	≤60	adult	9,794	52,646	52,368	23,625	45,108	79,552
Financial assets	25≤		adult	8,752	76,771	196,046	2,050	23,856	166,721
Deposits	25≤		adult	8,752	48,080	84,557	1,777	20,170	113,418
Financial securities	25≤		adult	8,752	24,530	110,710	0	0	33,458
Primary residence	25≤		adult	8,752	256,871	205,202	0	237,792	488,678
Other real estate	25≤		adult	8,752	24,180	109,421	0	0	0
Financial assets scaled by disposable household income	25≤		adult	8,676	1.22	2.02	0.05	0.52	3.06
Homeowner (0/1)	25≤		adult	12,501	0.78	0.42	0.00	1.00	1.00
Share of risky financial assets	25≤		adult	7,565	0.12	0.23	0.00	0.00	0.50
			≥EUR 2500 in deposits						
Has any risky financial assets (0/1)	25≤		adult	7,565	0.32	0.47	0.00	0.00	1.00
			≥EUR 2500 in deposits						
Savings rate (active)	25≤		adult**	5,703	0.00	0.35	-0.38	0.02	0.37

Table B1: Summary statistics of the main dependent variables

* Adult refers to being a household head or partner as defined in table Table B3

** Additional sample selection criteria apply, see Table B3

Name in English	SN dataset	Description
Dutch Hereditary Cancer Registry	External dataset	Data on genetic testing, cancer diagnoses, and preventive surgeries for individuals in Lynch syndrome-affected families.
Qualitative characteristics of employment relationships	BAANKENMERKENBUS	Qualitative data on jobs and wages of employees at Dutch companies for a specific reporting year or part of a reporting year, including the start and end date of the employment relationship, type of employment (e.g., regular employee, on-call, outsourcing, manager-large shareholder), social security insurance indicators (e.g., insured for unemployment benefits).
Quantitative characteristics of employment relationships	BAANSOMMENTAB	Qualitative data on jobs and wages of employees at Dutch companies for a specific reporting year or part of a reporting year, including taxable salary, calendar days worked, and payroll tax withheld.
Jobs and wages according to the administration of the Employee Insurance Agency	S/POLISBUS	Quantitative and qualitative data on jobs and wages of employees at Dutch companies for a specific reporting year or part of a reporting year.
Regional Income Distributions	RIO	Annual data on the income of persons and households for a sub-sample of the Dutch population including about 2 million households.
Income of People / Households	IPI / IHI	Annual income components (such as labor income, subsidies, income from entrepreneurship) of people resident in the Netherlands on the 1st of January of the statistical year. Information on the position of the person within the household with respect to the head of the household.
Income of People / Households	INPATAB / INHATAB	Revised version of IPI/IHI
Income Panel Cohort	IPOREVE	Cohort study of approximately 90 thousand households, annual data on income and wealth components, harmonized with VEHTAB and INPATAB/INHATAB definitions.
Wealth of households	VEHTAB	Annual wealth components (assets and liabilities) of households in the Netherlands on the 1st of January of the statistical year. SN compiles this dataset from a broad range of sources, including income tax returns (tax on the primary residence/box 1, substantial interests/box 2, wealth tax/box 3), information directly supplied by financial institutions on asset holdings and loans, data on house values estimated for municipal taxation (WOZ-value), student loans, etc.
Extract from the Municipal Personal Records Database	GBAPERSOONTAB	Demographic background data (that do not or hardly change) of all persons who appear in the Municipal Personal Records Database from 1 January 1995 (e.g., gender, year of birth, migration background).
Date of death of persons registered in the Municipal Personal Records Database	GBAOVERLIJDENTAB	Contains the date of death of all persons who have died since 1 October 1994 and who were registered in the Personal Records Database (BRP) on the date of death.

Name in English	SN dataset	Description
Persons with a partner with an address	PARTNERBUS	Contains all persons registered in the Personal Records Database (BRP) from 1 October 1994 who (ever) formed a cohabiting couple at one address for a continuous period. A cohabiting couple includes both married (or in registered partnerships) and unmarried couples. Non-married couples form a cohabiting couple if they have a child in common, ever move to a new address together, or are considered as partners for taxation or social subsidies.
Persons and their legal parents	KINDOUDERTAB	Contains all persons registered in the Municipal Personal Records Database (BRP) and the identifying numbers of their parents insofar as the parent(s) could be identified.
Annual dispensations of medicines per ATC-4 code per person	MEDICIJNTAB	All dispensed medicines that are reimbursed under the basic health insurance policy to persons who are registered in the Municipal Personal Records Database (GBA). No quantities are recorded; merely the 4-digit ATC codes (e.g., N06A) are listed that were dispensed for a given person in the statistical year.
Address of people	GBAADRESOBJECTBUS	(Encrypted) address of people registered in the Municipal Personal Records Database (BRP) with starting and ending date of validity

Table B2: Data sources

Variable name	Description	Data sources / variables
Lynch Syndrome-related		
Year of genetic test	Year when a person underwent genetic testing for Lynch syndrome. For about 50 individuals, the DHCR could not obtain the details of the genetic test from the clinical geneticist. However, the test outcome (positive/negative) is recorded, as this was shared with the DHCR by the tested person (or relatives) orally, or the test outcome was recorded in medical documents shared with the DHCR (e.g., colonoscopy results). In these cases, I impute the year of DNA test as the median year of DNA tests of the siblings of the concerned person, and set the suspected gene mutation the same as the mutation of the siblings.	DHCR
Suspected gene mutation	The gene mutation which leads to Lynch Syndrome in the individual's family, the gene the individual is tested for. One of MLH1, MSH2, MSH6, EPCAM, or PMS2. The MLH1/MSH2 genes are responsible for about 80% of the mutations in the sample	DHCR
Genetic test outcome	Mutation carrier (positive-tested) or non-carrier (negative-tested)	DHCR
Prophylactic (preventive) surgeries	Prophylactic surgeries listed with the date of operation and type of the operation (e.g., colectomy, hysterectomy)	DHCR
Cancer diagnoses	Cancer diagnoses listed with the diagnosis date and the International Classification of Diseases (ICD) code	DHCR
Labor market and income		
Pre-tax labor income (salary)	The salary that serves as a base for payroll taxes and national insurance premia; aggregated over all jobs of a person in a given year. Includes overtime pay, pay in nature (e.g., company car's tax value), and bonuses as well	1995-1998: RIO/LOONFIB 1999-2016: BAANS./FISCLOON 2017-2019: S/POLISBUS/LNLBPH
Labor income	Equals to *Pre-tax labor income* if an individual is working, otherwise 0	derived
Days worked	Number of calendar days that a person was employed in a given year. Overlapping periods of employment are aggregated, maximum 365 (366) days per year	1999-2016: BAANS./KALDG 2017-2019: S/POLISBUS/BAANDAGEN
FTE days worked	The number of days worked corrected for part-time employment. For the 2001-2016 period (BAANSOMMENTAB), full-time equivalent days are calculated by multiplying the part-time factor with the number of days worked. Winsorized at 366 per year	2001-2016: BAANS./DEELTIJDFACT. 2017-2019: SPOLIS./VOLTIJDDAGEN
Wage	= Pre-tax labor income / Full-time equivalent days worked. Winsorized at the 99^{th} percentile	derived
Working (indicator)	= Pre-tax labor income > 0	derived
Partner's labor income	Defined as 'Labor income' but for the partner. In case the individual has no partner it takes the value 0	as above

Variable name	Description	Data sources / variables
Disposable household income	= Gross personal income (pre-tax labor income, entrepreneurial income, transfers such as unemployment, sickness, disability insurance benefits, pension benefits, social security benefits, housing allowance, alimony) of all household members (-) income insurance premia (paid by employer or employee) (+) household-level income (income from wealth, and some subsidies received at the household level such as child-related subsidies) (+/-) alimony and other transfers paid/received at the household level (-) taxes on income and wealth. Winsorized at EUR -500,000 and EUR 1,000,000, following the winsorization in the IHI dataset.	1992-2002: IPOREV/INHBESTINKH 1995-2000: RIO/BIHH94E 2001-2002: RIO/BESTINKH 2003-2010: IHI/BVRBESTINKH 2011-2019: INHATAB/INHBESTINKH
Wealth		
Net wealth	Balance of assets and liabilities	1992-2005: IPOREV/VEHW1000VERH 2006-2020: VEHTAB/VEHW1000VERH
Assets	Bank deposits/savings, financial securities, real estate, enterprise capital, substantial interest, and other assets	1992-2005: IPOREV/VEHW1100BEZH 2006-2020: VEHTAB/VEHW1100BEZH
Financial assets	Sum of bank deposits/savings and financial securities. In my baseline specification, I winsorize financial assets at the 1^{st} and 99^{th} percentiles.	1992-2005: IPOREV/VEHW1110FINH 2006-2020: VEHTAB/VEHW1110FINH
Bank deposits/savings	All money kept in a bank account, including foreign deposits. Winsorized variable at the 1^{st} and 99^{th} percentiles	1992-2005: IPOREV/VEHW1111BANH 2006-2020: VEHTAB/VEHW1111BANH
Financial securities	Sum of bonds and shares (excluding substantial interest). Bonds relate to the market value of negotiable instruments serving as evidence for debt. Shares relate to the market value of shares in corporations, mutual funds, and other investment funds. Investments in risky financial securities (shares and equity mutual funds) dominate financial securities. Using detailed survey data, Gaudecker (2015) finds that only 5% of Dutch households with financial securities do not own any shares or mutual funds but instead own only bonds or options, and that the majority of mutual funds held are equity funds. Using data from Statistics Netherlands, I estimate that only 17% of households with financial securities in 2011 received any interest payments from bonds. Winsorized variable at the 1^{st} and 99^{th} percentiles	1992-2005: IPOREV/VEHW1112EFFH 2006-2020: VEHTAB/VEHW1112EFFH
Primary residence	Property owned and used as the main residence. Based on the WOZ value determined for municipal taxes	1992-2005: IPOREV/VEHW1121WONH 2006-2020: VEHTAB/VEHW1121WONH
Other real estate	Includes second homes, holiday homes, investment properties, and such. Based on the WOZ value determined for municipal taxes	1992-2005: IPOREV/VEHW1122OGOH 2006-2020: VEHTAB/VEHW1122OGOH

Variable name	Description	Data sources / variables
Enterprise capital	Balance of assets and liabilities belonging to the business of self-employed (own unincorporated enterprise)	1992-2005: IPOREV/VEHW1130ONDH 2006-2020: VEHTAB/VEHW1130ONDH
Substantial interests	Substantial share (>5%) in equity in incorporated businesses (e.g., family firms)	1992-2005: IPOREV/VEHW1140ABEH 2006-2020: VEHTAB/VEHW1140ABEH
Additional assets	Includes cash, movable property leased or used as investment, trust assets, shares in undivided estate, assets encumbered with usufruct, or limited ownership	1992-2005: IPOREV/VEHW1150OVEH 2006-2020: VEHTAB/VEHW1150OVEH
Liabilities	Sum of primary residence loans, education loans, and other loans	1992-2005: IPOREV/VEHW1200STOH 2006-2020: VEHTAB/VEHW1200STOH
Primary residence loans	Loans for the purpose of constructing, purchasing, or improving the primary residence. The saving money intended to repay the mortgage is partly included	1992-2005: IPOREV/VEHW1210SHYH 2006-2020: VEHTAB/VEHW1210SHYH
Education loans	Loans to cover study expenses. Only completely recorded since 2011, previously part of 'Other loans' (if declared on income tax form)	1992-2005: IPOREV/VEHW1220SSTH 2006-2020: VEHTAB/VEHW1220SSTH
Other loans	Includes bank account overdrafts, consumer durable loans, other real estate loans, financial asset loans, tax debts. Until 2011, other loans were only recorded for households who were obliged to pay a wealth tax (box 3)	1992-2005: IPOREV/VEHW1230SOVH 2006-2020: VEHTAB/VEHW1230SOVH
Net real estate	= Primary residence + Other real estate - Primary residence loans. Winsorized at the 1^{st} and 99^{th} percentiles	derived
Other assets	= Enterprise capital + Substantial interests + Additional assets. Winsorized variable at the 1^{st} and 99^{th} percentiles	derived
Other debt	= Education loans + Other loans. Winsorized variable at the 1^{st} and 99^{th} percentiles	derived
Financial assets scale to income	Financial assets divided by the mean disposable household income in the sample period. Winsorized variable at the 1^{st} and 99^{th} percentiles	derived
Share of risky financial assets	= Financial securities / Financial assets. Trimmed below 0 and above 1 (in very rare cases Bank deposits can take negative values).	derived
Stock market participation	= Financial securities > 0	derived

Variable name	Description	Data sources / variables
Savings rate	=1-(Consumption/Disposable household income) Household-level consumption is derived from the accounting identity that total household spending is equal to income plus capital gains minus the change in wealth over the period (Eika, Mogstad, and Vestad, 2020). I correct for capital gains on *Financial securities* using national account data on the mutation in financial securities due to financial transactions and due to changing prices, following Ji, Teulings, and Wouterse (2019). For the *Primary residence*, if a homeowner household does not change address and continues to own its home, I assume that all value changes are from capital gains. In case a homeowner household moves but stays a homeowner, I assume that capital gains for the whole year are proportional to the growth rate of home values in the municipality of origin. If a homeowner household becomes a renter or a renter household becomes a homeowner, I assume that it earns capital gains for the fraction of the year it was a homeowner based on the growth rate of home values in the municipality of origin. For *Other real estate*, I assume zero capital gains if the households moves from not owning any other real estate to owning any, or vice-versa. If the household continues to own other real estate, I assume all year-on-year value changes up to 15% of the base year value to be capital gains, following Ji, Teulings, and Wouterse (2019). I assume that capital gains on savings accounts, entrepreneurial wealth, substantial interests, and other assets can be neglected. Following Ji, Teulings, and Wouterse (2019), I exclude individual-year observations if (1) the household composition (household head or partner) changes, (2) disposable household income is below 75% of the yearly social welfare level for a single household in 2009 (EUR 5760), (3) consumption is negative or average annual consumption over time is lower than EUR 5760, (4) average annual consumption over time is at least EUR 120,000 higher than average household disposable income. Following Ji, Teulings, and Wouterse (2019), I also winsorize consumption at the bottom, at EUR 5760, and at the top, at one million. Furthermore, I exclude all 2010 observations where the household had *Education loans* in 2011 (due to a break in the *Education loans* series) and all 2010 observations where the household had no *Other loans* in 2010 but had *Other loans* in 2011 (due to a break in the series). I also exclude (7) individuals who ever had *Substantial interests* due to changing coverage of these assets in the wealth statistics. In the baseline specification, I trim the resulting savings rate at the lower limit of -1.5, but I also implement robustness tests with other trimming thresholds.	derived

Variable name	Description	Data sources / variables
Home owner	Binary indicator of home ownership	1992-2002: IPOREV/INPPERSBRUT 1999-2002: OBJECTW./HUURKOOP 2003-2010: IPI/PERSBRUT 2011-2019: INPATAB/INPPERSBRUT
Demographic / other		
Has partner	Has partner, including married and non-married partnerships, on the 1st of January of a given year. Statistics Netherlands considers all couples within an 'official' partnership (e.g., marriage, civil partnership and cohabitation agreements) as partners. Furthermore if two people change addresses together they are also considered as partners	PARTNERBUS
Adult	Household head or partner of the household head. The position within the household is determined relative to the household head (the household head is the person with the most important socio-economic position, largely determined by personal income and the source of income)	1992-2002: IPOREV/INPPOSHHK 2001-2002: RIO/POSHH 2003-2010: IPI/POSHHK 2011-2019: INPATAB/INPPOSHHK
Number of children	Number of children that were born in or before a given year	1985 - 2020: KINDOUDERTAB
Year of birth, gender	Year of birth and gender for people registered in the Municipal Personal Records Database	1995 - 2020: GBAPERSOONTAB
Address on 1st of January	(Anonymized) address on the 1st of January	1995- 2020: GBAADRESOBJECTBUS
Number of siblings	Number of siblings on the maternal side (if the mother of the individual is known)	1985 - 2020: KINDOUDERTAB

Table B3: Variable definitions

C Data Cleaning of the Dutch Hereditary Cancer Registry

The following describes the data collection and cleaning steps I undertook at the Leiden site of the Dutch Hereditary Cancer Registry before importing the DHCR data to the secure environment of Statistics Netherlands.

Most of the information on Lynch families in the DHCR is stored digitally in a relational database. These include data on demographic characteristics (date of birth, sex, family relations), genetic test results (type of mutation tested for, test result, date of test), cancer history (type of tumor, diagnosis date), and preventive surgeries (type of operation, date). On the other hand, some information had to be hand-collected from the scanned dossiers of registered individuals. The dossier of an individual always includes the registration (consent) form, and all relevant correspondence (letters, emails) between the DHCR and the registered individual, their general practitioners, medical specialists, and clinical geneticists. The dossier also includes the medical documents on genetic tests, preventive surgeries, cancer diagnoses, and medical screenings (e.g., colonoscopy results). These documents are collected from the registered individuals and/or their physicians.

First, to be able to match registered individuals to the microdata files offered by Statistics Netherlands, I collected identifying information from the dossiers, including social security numbers (BSN numbers) and if the social security number was not available, address information (latest address and year of validity of that address). Identifying information is stored at the computers of the DHCR and was never shared with me outside the Leiden offices of the DHCR. Second, I have also collected information on the year of registering with the DHCR, and whether the individual had registered before undergoing genetic testing. Finally, I have cross-checked the genetic testing data stored in the DHCR's database with the medical dossiers. This resulted in several updates to the DHCR's database. In some cases, the database indicated that a genetic test took place, but it did not record the details of the test. These details I could often locate in the dossiers. In other cases, the test date was missing or incorrectly filled. Seldom, the test result was incorrectly recorded.

C.1 Individuals almost certainly at 50% risk of inheriting LS

I collect data on the cancer history of tested individuals' parents from the family trees recorded by the DHCR. Family trees contain family linkages (parents), birth and death years (with several missing observations), and information on cancer diagnoses (cancer type, age at diagnosis). First, I determine if a person is suspected to having inherited the LS gene mutation from their mother (maternal side) or father (paternal side). I do so by verifying the presence of the maternal and paternal grandparents in the family tree (DHCR family trees do not record the non-affected side of the family). Next, I link to each individual the birth year, death year, and age at first recorded cancer diagnosis of both of their parents. Besides information on family trees, for registered patients the DHCR contains data on cancer histories and DNA testing histories. For each person in my sample, I use this dataset and merge information on DNA testing and cancer diagnoses of their siblings and parents. I consider an individual to have 50%

at-birth risk of inheriting LS if any of the following criteria is met:

- maternal (paternal) inheritance is suspected and the person's mother (father) was still alive in the DNA test year,
- maternal (paternal) inheritance is suspected and the person's mother (father) had been diagnosed with cancer before the DNA test year, at an age not older than 65 years,
- maternal (paternal) inheritance is suspected and the person's mother (father) had passed away before the DNA test year, at an age not older than 60 years,
- the side of inheritance cannot be determined (from the DHCR's records) but both parents were still alive in the DNA test year,
- the side of inheritance cannot be determined (from the DHCR's records) but one of the parents had been diagnosed with cancer before the DNA test year, at an age not older than 55 years,
- any siblings of the tested person had a DHCR-registered colorectal or endometrial cancer, or had tested positively for LS before the DNA test year,
- one of the tested person's parents had tested positively for LS before the DNA test year.

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