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Household Finance and Life-Cycle Economic Decisions under the Shadow of Cancer

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Household Finance and Life-Cycle Economic Decisions under the Shadow of Cancer

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Abstract

I study the causal effects of life expectancy on financial, economic, and demographic 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 similar to that of the general population. 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
Keywords: Household finance, life expectancy, savings, labor supply, genetics

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1 Introduction

Life-cycle economic models often presume that life expectancy affects individuals' economic choices, including savings (De Nardi, French, and Jones, 2009), labor supply (Aísa, Pueyo, and Sanso, 2012), and human capital accumulation (Soares, 2005). On the other hand, empirical evidence on the causal effects of life expectancy is still limited due to at least two 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 requires exogenous variation in the mortality risks individuals face. Second, such exogenous variation must happen independently from episodes of bad health. While bad health does reduce life expectancy, it may also impact other determinants of economic choices, such as labor productivity, the marginal utility of consumption, risk aversion, and other economic parameters (Decker and Schmitz, 2016; Finkelstein, Luttmer, and Notowidigdo, 2013; Moran, Short, and Hollenbeak, 2011).

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 which drastically increases the risk of colorectal (up to 50 to 80% lifetime risk), endometrial (25 to 60%), and ovarian cancer (5 to 10%).[1] I estimate that LS reduces median life expectancy by about 3 years, despite preventive care. Most of this decrease happens during old age: LS reduces median retirement length by about 10%.

Individuals in my sample decide to undergo genetic testing to learn if they have inherited the gene mutation that causes LS in their families. Crucially, tested individuals are still healthy, they have not yet developed cancer. Genetic testing randomizes tested persons into two groups. Those who test positive learn that they face an elevated risk of cancer and a shorter life expectancy. Those who test negative learn that their cancer risk is similar to that of the general population. My dataset contains a balanced sample of both positive- and negative-tested individuals. To identify the effects of the life expectancy shock, I merge genetic testing data with Dutch administrative panel data and study the differences in outcomes between these two groups.

I start by estimating the effects of testing positive for LS on household wealth accumulation. I find that following genetic testing, the financial wealth (incl. bank deposits and financial securities) of positive-tested households exhibit a gradual decline. In the first five years following testing, households of Lynch-positive individuals have on average EUR 27,000 lower financial wealth than household of negative-tested people. During the next five years the difference increases to EUR 70,000, where it stabilizes. On average, during the first 15 years after testing, Lynch-positive individuals have EUR 57,000 lower financial assets than those who tested negative. This is an economically significant difference equal to about 1.25 year median disposable household income in my sample. Individuals who had no children before testing and those whose parents had been previously diagnosed with cancer exhibit a larger drop in financial assets, although these differences are not statistically significant. Testing positive for LS does

[1] 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)

not affect the real estate wealth (home ownership) of tested individuals.

Next, I study four channels that could give rise to the negative effect on financial wealth accumulation. These are changing household composition, decreasing labor income, lower savings rates, and lower portfolio allocation to risky financial assets.

I turn to household composition first, and study the effects on two important early-life demographic outcomes, family formation, and childbearing. I find that 5 to 9 years after testing, individuals who were tested when not older than 45 years face an 8 pp lower probability of having a partner. This negative effect is particularly pronounced for people who had no children before testing (16 pp). Individuals who were tested after turning 45 years are on average not affected.

Being diagnosed with a Lynch mutation also influences reproductive choices among previously childless individuals who are still in the reproductive age. Among people who were tested not older than 30 years, positive-tested shift childbearing to a younger age but eventually have no fewer children than negative-tested. On the contrary, among individuals who were tested between 31 and 45 years old, positive-tested face an about 20 pp lower probability of ever having children.

Can changing household composition completely account for the lower wealth accumulation of households of Lynch-positive individuals? My results suggest otherwise. I employ two strategies to disentangle the effects of changing household composition from the effects of other economic channels. First, I control for partnership status and having children in my regression models, while keeping in mind that these controls may also be affected by the treatment. I estimate almost identical negative effects on financial wealth accumulation and the other main outcome variables that I study. Second, I consider a sub-sample of individuals, about two thirds of my sample, whose demographic outcomes are little affected. This group comprises people who were tested after turning 45 years old and those who already had at least one child before testing. I find a slightly lower but still economically and statistically significant negative effect on financial wealth accumulation (on average EUR -43,500 in the first 15 years following testing) in this sub-sample.

Economic theory suggests that decreasing life expectancy may lead to a lower labor supply (early retirement) as the need for retirement savings is lower (Bloom et al., 2003). I document a negative effect of testing positive on the labor income of men, who earn on average EUR 6,600 less in the first 15 years after testing. This drop in income is equal to 17% of the median annual labor income of males in my sample. On the contrary, the labor income of women is not affected, although positive-tested women exhibit a slightly lower labor participation (not statistically significant). Moreover, in the long run positive-tested women are estimated to be 7.5 pp less likely work as an entrepreneur.

The drop in male labor income can be explained both by a somewhat lower probability of working (not statistically significant) and by reducing labor supply along the intensive margin. In the first 15 years after testing, positive-tested working males work on average 20 fewer full-time equivalent days per year than negative-tested ones. The daily wage is not statistically significantly affected by the Lynch diagnosis. Considering the effects throughout the life-cycle,

I find that it is in the pre-retirement age (60-64) when working men reduce their labor earnings and labor supply the most.

Having studied tested individuals' labor response, I turn to estimating the effects on their partners' labor outcomes. This is important because most individuals in my sample (77%) have a partner when they undergo genetic testing, and partners may make labor market choices collectively (Blundell and MaCurdy, 1999). I find that only the labor income of positive-tested women's partners (98% male) are affected. Part of this negative effect is due to working less, while the other part can be explained by lower wages.

Lower probability of having a partner, lower own labor income (for men) and lower labor income of the partner (for women) suggest that positive-tested individuals would face a lower household income. Indeed, I estimate that positive-tested individuals have about 15% lower household income during the first 15 years following testing. I show that household composition can only account for a small share of this negative effect, which is mostly driven by lower household labor income.

Besides changing household composition, and reduced income, lower wealth accumulation may also be explained by lower savings rates and by more conservative financial asset allocation choices. My results indicate that positive-tested households indeed save a lower share of their disposable income, where savings is imputed from changes in household wealth corrected for capital gains. This is in line with theoretical models where a lower lifespan implies a lower savings rate (e.g., Bloom et al., 2003).

Regarding asset allocation, positive-tested households swiftly reduce their share of financial assets allocated to risky financial securities after learning about their higher predisposition to cancer. During the first 15 years of the post-testing period, positive-tested households invest on average 8 pp lower share of their financial assets into financial securities (mostly stocks and investment funds). This is an economically (and statistically) significant effect given the unconditional share of 12 pp in my sample. The observed drop in the risky share is in line with models where due to return predictability investors should allocate substantially more to stocks if their horizon is longer (Barberis, 2000).

In the final part of this paper, I estimate how positive- and negative-tested individuals react to learning their mutation carrier status compared to a counterfactual where they do not receive this information. For the lack of a natural experiment that randomizes individuals into a tested and non-tested group, I employ 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 these individuals on a broad range of characteristics, including the year of birth, gender, having a partner, the number of children, and home ownership.

I find that while some effects that I have previously documented are mainly the result of a negative effect on positive-tested individuals (e.g., probability of having children) or a positive effect on negative-tested ones (e.g., labor supply), most effects arise as the difference of opposite sign impacts. For example, compared to the general Dutch population, positive-tested individuals decrease their allocation to risky financial assets following genetic testing,

while negative-tested individuals increase it. These findings suggest that genetic testing can be beneficial for the socioeconomic outcomes of negative-tested individuals, possibly because it eliminates the fears of facing a high risk of cancer.

My work aims to contribute to our understanding of how individuals incorporate information on 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 particularly life expectancy on household financial and economic behavior. In macroeconomics, most work relies on cross-country samples, reporting a positive correlation between average life expectancy at birth and savings rates or human capital investment (e.g., Bloom et al., 2007). At the micro-level, most studies focus on the later years of life, and find 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). Spaenjers and Spira (2015) estimate a positive effect of subjective life expectancy 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. The literature relies mostly on instrumental variables estimation to overcome the endogeneity of (subjective) life expectancy and to address the problem of focal survey answers. My work adds to this field by studying how a well-defined information shock on a substantial future health risk affects decisions over the life-cycle. Genetic testing offers a clear way to identify the effects of shifting life expectancy among a set of individuals who have a strong awareness of cancer due to their family history.

This paper is also closely related to the small but growing field that studies the impact of predictive medical testing on individuals' economic decisions. Oster, Shoulson, and Dorsey (2013) and Oster et al. (2010) find that testing positive for Huntington's disease, a progressive and eventually fatal neurogenerative disease, lowers investments in human capital and increases the probability of taking out long-term care insurance, 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. I study the long-term effects of learning about genetic risks on hitherto under-investigated outcomes such as wealth accumulation, labor supply, and family formation.

Finally, this study 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 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 accumulate lower wealth.

The paper proceeds as follows. Section 2 describes the risks associated with Lynch syndrome, and discusses the possible economic reactions to these risks. Section 3 presents the data sources and the main variables, while Section 4 discusses the empirical strategy. Sections 5, 6, 7, and 8 present the effects of testing positive for LS on the household balance sheet, household composition, labor and household income, and savings and investments decisions, respectively. Section 9 studies the effects of undergoing genetic testing by comparing outcomes of negative- and positive-tested individuals to those of a matched sample from the general Dutch population. Section 10 concludes.

2 Background and incentives

Lynch syndrome (LS) is a hereditary condition that gives rise to a substantially increased lifetime risk of colorectal and endometrial cancer, and an increased risk of some other forms of cancer. LS is caused by a mutation in one of four genes (MLH1, MSH2, MSH6, PMS2) that play a crucial role in suppressing tumor growth, or in the EPCAM gene which may silence the neighbouring MSH2 gene. Mutations in these so-called mismatch repair genes cause DNA damage to go unrepaired, which leads to an increase in mutation frequency. The disease is inherited in an autosomal dominant manner: individuals with one parent who carries a Lynch mutation have a 50% probability of inheriting the faulty gene. Individuals who test negative for the mutation responsible for Lynch syndrome in their families face a lifetime cancer risk similar to that of the general population. Families affected by Lynch syndrome are most often painfully aware of their family history of cancer, as illustrated by the following testimony:

> *"I grew up surrounded by cancer. My grandma, two aunties and one great uncle all died from cancer when I was a girl. Then it was my mum's turn. Cancer. Again. More dreadful treatment but still the same sad outcome. My brother, sister and I become adults, expecting the worst."*
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The following section presents an overview of the medical and economic risks faced by individuals diagnosed with Lynch syndrome in the Netherlands, and discusses the possible economic reactions to these risks as predicted by economic theory.

2.1 Risks associated with Lynch syndrome

To contextualize the economic reaction to a Lynch syndrome diagnosis, it is important to understand the medical and economic 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

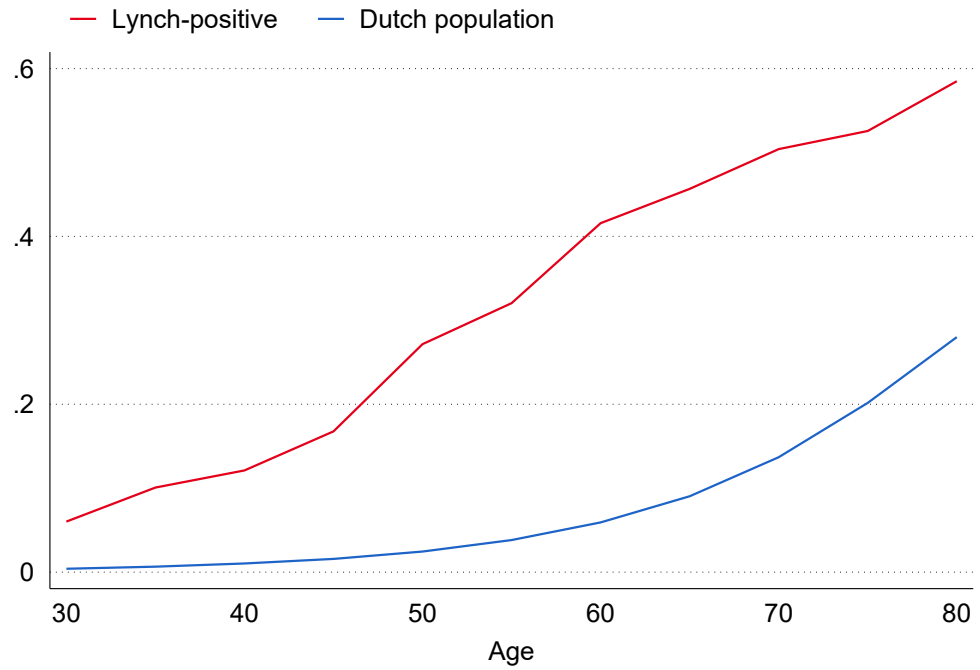
an additional 25 to 60% risk of endometrial cancer and a 5 to 10% risk of ovarian cancer. In comparison, the lifetime risk in the general population for colorectal, endometrial, and ovarian cancer are 5.5%, 2.7%, and 1.6%, respectively (Wolf, Buchanan, and Farkas, 2013). The risk of some other forms of cancer such as stomach, small bowel, upper urological tract, biliary tract, and brain cancer is also elevated for Lynch mutation carriers.

As letters from clinical geneticists presented in Appendix A (Figures A2 and A3) reveal, these baseline risks are clearly communicated to those who undergo genetic testing. The risk of cancer and cancer mortality can be substantially reduced by periodic cancer screenings, and in some cases by preventive surgeries[2]; however, it cannot be eliminated, and LS-positive individuals continue to face a substantially elevated risk (Møller et al., 2017). I estimate that the positive-tested individuals in my sample, who mostly carry the MLH1 or MSH2 mutation and who were tested before developing any cancer, face an about 50% cumulative incidence of cancer by the age of 70 (Figure 1). The risk of cancer is slightly higher for men than for women. This cancer exposure, which is mostly driven by increased colorectal and endometrial cancer risk, is considerably higher than the about 15% cumulative incidence that the general population faces by the same age.

Cancer screening also has a positive effect on cancer survival conditional on cancer incidence (De Jong et al., 2006), as it enables early detection and treatment. In spite of this, the greater incidence of colorectal and endometrial cancer and the involvement of hard-to-prevent extra-colonic and extra-endometrial cancers (e.g. upper urological tract, pancreatic cancer, and brain cancer) result in an increased mortality in LS (Pylvänäinen et al., 2012). I estimate a 2.7-year reduction of median lifespan in my sample based on a parametric (Gompertz) survival model that controls for sex, the age at testing, and the suspected mutation type (MLH1/MSH2 or other mutation) (Figure 2). As the figure reveals, most of the increase in mortality happens in the post-retirement age. At the age of 60, the median life expectancy of positive-tested individuals is approximately 2.5 years or 10% lower (22.7 years) than the median life expectancy of negative-tested people (25.2 years). The health risks presented by LS are also reflected by the fact that individuals correctly recall their test results, exhibit high compliance with recommended cancer screening protocols, and in general report their cancer risks as high (Aktan-Collan et al., 2001; Järvinen et al., 2009).

Cancers associated with Lynch syndrome may not only increase mortality but might also impose high medical costs and disability on affected individuals. Nevertheless, 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 (in 2021, maximum EUR 385). The Netherlands also offers a generous and comprehensive public long–

[2]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 greatly 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 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.



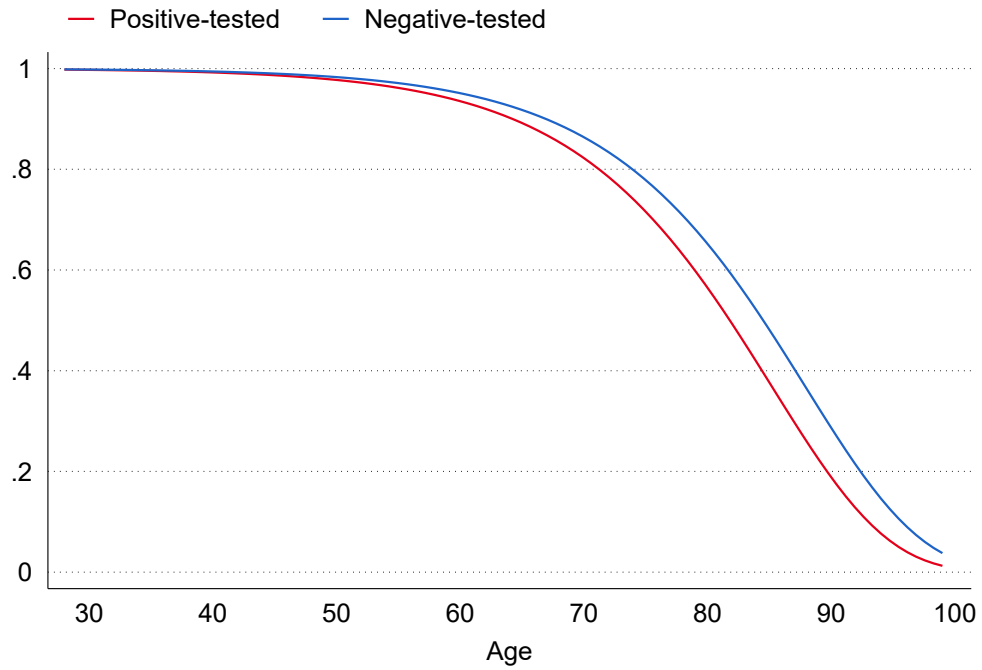
The line *Lynch-positive* presents non-parametric Kaplan-Meier failure estimates in my sample. The start of the observation time is the year of genetic testing, failure is defined as developing cancer for the first time, and observations are censored at the year of death or 2018 at the latest. Observations 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).

Figure 1: Cumulative cancer incidence by age among LS mutation carriers and the general population

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).

Disability and income risks imposed by LS are also limited by the nature of the disorder, and by the generous welfare state of the Netherlands. My estimations show that positive-tested individuals face only a slightly elevated risk of being disabled than negative-tested ones.[3] A possible explanation is that certain extra-colonic, extra-endometrial cancers (e.g., pancreas or biliary tract, brain) that entail the worse prognosis, and which are responsible for about 60% of cancer death among LS-positive individuals (Pylvänäinen et al., 2012), manifest mostly at a later age. Even in case of disability, earnings loss is limited: The Netherlands offers a very generous public disability insurance scheme, which also covers non-work-related disability, and on average replaces 70 percent of lost gross earnings. 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). See Appendix C for details.

[3]In a non-parametric Kaplan-Meier survival analysis, by the age of 40, 5.1% (5.7%) of positive-tested (negative-tested) individuals have ever entered disability, by the age of 50, 8.0% (10.0%), by the age of 60, 13.7% (12.4%). In a difference-in-differences regression, where the sample period is not restricted to pre-cancer years, positive-tested are 1.7 pp (t-stat=1.11) more likely to be disabled.



The figure presents parametric (Gompertz) survival estimates where the start of the observation time is the year of genetic testing. The model controls for sex, age at test (in groups of 10 years), and the mutation type (MLH1/MSH2 or other mutation)

Figure 2: Estimated survival function of positive- and negative-tested individuals

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. In a difference-in-differences regression, I estimate that positive-tested people are slightly less likely to use antidepressants ($\beta = -0.017$, t-stat=-0.57) and tranquilizers ($\beta = -0.03$, t-stat=-1.25) than negative-tested ones.

2.2 Lynch syndrome health risks and economic decisions: a theoretical overview

The purpose of this section is to derive implications for the empirical analysis based on economic theory and the risks associated with Lynch syndrome. Learning about future health risks may affect a broad range of economic decisions over the life-cycle, including choices related to consumption/savings, portfolio allocation, labor supply/retirement, marriage and fertility, human capital formation, and health capital formation. Theoretically, these changes may be mediated by shifts in a variety of economic parameters and expectations thereof, including

mortality expectations, the income process, medical costs, demographic shifters (household composition), and preference parameters (risk aversion, health preference shifters). I argue that the most important channels through which testing positive for a Lynch syndrome mutation affects economic behavior in the Netherlands are altered mortality expectations and changes in household composition.

As Figure 2 illustrates, despite frequent cancer screening, individuals affected by Lynch syndrome are expected to have a shorter lifespan, with most excess mortality presenting after the sixth decade of life. The length of the lifespan and mortality expectations are important theoretical determinants of individuals' life-cycle saving and consumption choices. In a model of deterministic lifespan and endogenous retirement age, Bloom et al. (2003) show that a decrease in life expectancy decreases the optimal length of life spent working but not enough to offset the lower need for retirement income, and consequently savings rates decrease at every age. De Nardi, French, and Jones (2009) investigate the effect of uncertain life expectancy on the net wealth decumulation for the elderly. Although reduced life expectancy leads to a faster decumulation of assets, uncertainty dampens this effect as individuals prefer to avoid outliving their net worth and keep part of their net worth for precautionary reasons. Heimer, Myrseth, and Schoenle (2019) elicit subjective survival probabilities using a novel survey instrument and show that survival is underestimated by the young while it is overestimated by the old. In a calibrated life-cycle model, the authors estimate that this pattern of survival beliefs leads to higher consumption rates and lower wealth accumulation during the working years.

Lifespan and mortality expectations also affect retirement decisions, as illustrated by the model of Bloom et al. (2003), but higher expected mortality may also reduce labor supply at the intensive margin as a result of a lower need for retirement savings.

Life expectancy can also have a meaningful impact on individuals' portfolio allocation. Higher mortality expectations reduce the planning horizon, which may in turn decrease the optimal allocation to risky assets (Barberis, 2000). In the life-cycle model of Heimer, Myrseth, and Schoenle (2019) mortality beliefs influence portfolio allocations indirectly through their effect on life-cycle wealth accumulation. Higher than actuarial mortality beliefs when young and lower than actuarial mortality beliefs when old result in lower wealth accumulation and a higher risky share during the life-cycle.

Changes in household composition might also impact the outcomes that I study, especially in the long run. Learning about carrying a LS 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, and I present corroborative evidence of this hypothesis in Section 6. Second, childbearing might also be affected if positive-tested individuals are afraid of passing down the faulty gene to their children, or because female mutation carriers are recommended to undergo preventive surgeries at an early age, which might shorten the reproductive period.

Most economic decisions might 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, as my

wealth data is aggregated at the household level, family composition also mechanically affects wealth levels. I aim to mitigate the effects of changing household composition on my estimates using two empirical strategies. First, in robustness tests, I control for having a partner and having any children in my regression models, why keeping in mind that these outcomes may also be affected by the treatment. Second, I show that my results continue to hold in a sample in which LS has little effect on partnership formation or childbearing. This sub-sample covers around $2/3^{rd}$ of my full sample and consists of individuals who already had a child before genetic testing and/or who were older than 45 years upon testing.

Finally, besides life expectancy and changes in household composition, shifts in other economic parameters and expectations thereof might also give rise to the economic behavior I document. I argue that these channels are somewhat limited in my context, although I cannot exclude that they contribute to my estimates. A future cancer diagnosis might bring about decreased labor earnings and high medical costs. However, as previously discussed, the generous Dutch universal healthcare insurance, the public long-term care scheme, and the public disability insurance scheme alleviate the risk of excessive medical costs and income loss from disability. Preference parameters might also be influenced by future health shocks. Decker and Schmitz (2016) document that health shocks may lead to increased individual risk aversion, but it is not clear if an increase in future risks of cancer would also increase present-day risk aversion. The marginal utility of consumption may also depend on the future health status, with possible implications for optimal life-cycle savings. On the other hand, it is theoretically and empirically ambiguous if the marginal utility of consumption is higher, lower, or similar in bad health states compared to good health states (Finkelstein, Luttmer, and Notowidigdo, 2009).

3 Data

This section presents an overview of the main data sources and variables used in this study. For a detailed description of the data sources and variables see Appendix B.

3.1 Genetic testing data from the Dutch Hereditary Cancer Registry

My data on individuals 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 (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). Some people registered with the DHCR lack information on genetic testing. These individuals have either not (yet) undergone testing, or the registry could not obtain their test results despite their best efforts. For about 50 individuals in my sample, while the DHCR could not obtain the details of the genetic test it could collect information on the mutation carrier status. The source of such information is either the tested person (or relatives) or medical documents (e.g., colonoscopy results) shared with the DHCR. For these observations, I impute the tested gene from the tested gene of the person's family members. I impute the year of the DNA test as the median year of the DNA tests of the person's tested siblings if any.

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.

3.2 Administrative data from Statistics Netherlands

Statistics Netherlands offers a broad set of microdata files that can be matched at the individual level using pseudo-anonymized identifiers (so-called rin numbers). 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 and the number of calendar days worked within a year 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.

Entrepreneur - Indicator based on the SN-determined annual socio-economic category, available for 2003-2019 (whole population), 1995-2002 (1/3rd random sample). The annual socio-economic category of a person is determined in principle by the largest source of income, with some exceptions (e.g. students).

Homeowner - Indicator based on the household income files and ownership status files. Available for the whole Dutch population from 1999 to 2019.

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.

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[4]; 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 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 I assume that all value changes are due to capital gains unless the household changes address (or moves from being a renter to an owner or vice-versa) in which case I assume zero capital gains. 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 A5.

Appendix B Table A3 provides summary statistics for the main dependent variables used in this study.

[4]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.

4 Empirical strategy

My sample consists of individuals who underwent genetic testing for a Lynch syndrome mutation present in their family before developing any forms of cancer, and who were registered with the Dutch Hereditary Cancer Register before genetic testing. This section presents genetic testing as an ideal experiment, discusses the challenges we face when implementing this experiment and how these challenges can be addressed in my setting, and presents the empirical methodology used in the paper.

4.1 Genetic testing as an ideal experiment and related challenges

Conditional on the risk of carrying the tested Lynch mutation, the outcome of genetic testing is as good as randomly assigned. There are two principal empirical challenges to this ideal experiment presented by genetic testing. The first is to precisely establish the ex-ante risk of testing positive. Individuals whose parent is a mutation carrier have a 50% probability of inheriting the mutation (at-birth risk). Although I do not directly observe tested individuals' at-birth risk, the institutional settings of genetic testing among my sample of LS-affected families ensures that almost all tested individuals faced a 50% risk of inheriting the mutation at birth. This is because in LS-affected families genetic testing is carried out systematically, referred to as cascade testing. The process starts with the identification of one or multiple index patients who are suspected of LS based on their personal (and/or family) history of cancer. Index patients are tested extensively to determine the mismatch repair gene mutation, if any, responsible for their cancer. If and only if the index patient tests positive for a LS mutation are other family members also tested for the mutation. Testing starts with first-degree relatives (children, siblings, parents), who now face a 50% at-birth risk, and proceeds in the form of a cascade: a person is tested if one of their first-degree relatives had already tested positive. This procedure prevents unnecessarily testing, saves time and money, and crucially it mostly ensures that tested individuals face 50% at-birth risk. [5] [6] 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.

The ex-ante probability of testing positive for a LS gene mutation does not only depend on risk at birth but also information revealed during one's lifetime. Information arrives in the form of cancer diagnoses or the lack of thereof. If someone with a 50% at-birth risk of LS is diagnosed

[5] Rarely, parents are tested after their descendants has tested positive, for example if their child is the index patient in the family. To account for these cases, I exclude parents from my sample (9 individuals) who got tested following a positive, and DHCR-registered, test result of their offspring.

[6] In rare cases, individuals without a proven first-degree mutation-carrier relative might also be tested. This mostly occurs when an individual's parent who is suspected of LS cannot be tested because he or she had already passed away. If the parent had not developed a LS-associated tumour prior to their death, it is possible that they were not actually LS mutation carriers. This would mean that the tested individual had a 0% at-birth risk and would necessarily end up as a negative-tested person in my sample. To address this question, I collect data on the cancer history and DNA test results of tested individuals' parents and siblings from family trees and other registers available at the DHCR. Based on criteria discussed in Appendix D.1, I find that at least 93% of my sample faced almost certainly 50% risk of inheriting LS. This is a lower bound because cancer and DNA testing history of family members is incomplete for many families/individuals. In robustness tests (currently under tabulation), 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.

with colon cancer at the age of 40, LS is almost certain, while if the person stays cancer-free till the age of 75, LS can be almost completely excluded. Fortunately, the Dutch Hereditary Cancer Registry also contains information on the cancer history of registered individuals, which enables me to condition on past cancers. More precisely, I exclude individuals with past cancer diagnoses as I focus my study on people who test before developing any cancer.

The second empirical challenge is to collect the testing outcomes of all individuals who undergo genetic testing, irrespective of the test outcome. As Oster, Shoulson, and Dorsey (2013) highlight, the selection of individuals into medical cohorts may not be random. In our case, people who register with the DCHR despite having tested negative for Lynch syndrome might differ from people who register following a positive genetic test. This concern is addressed by the previously mentioned fact that the DHCR strives to obtain the genetic test results of all registered individuals, irrespective 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.

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) in my sample (*observed*) and the share that we would expect among cancer-free individuals with a 50% at-birth risk of LS (*expected*). This latter measure is calculated using Bayes' rule and cancer incidence estimates from the medical literature.[7] The two sets of estimates exhibit a close correspondence at all age groups, which gives strong evidence that my sample includes almost all individuals who had registered with the DHCR and subsequently underwent genetic testing.

While Figure 3 presents evidence that my sample includes a balanced number of positive and negative tested individuals, Table 1 reveals that these individuals are also similar as far as a broad range of characteristics are concerned (besides the conditioning variables age at testing and mutation type). The table presents results from linear regressions where the outcome variable is regressed on an indicator of a positive test result (mutation carrier) controlling for covariates that determine the ex-ante risk of testing positive (sex, age at test, type of mutation), and in case of time-variant outcomes year fixed effects.

4.2 Empirical methods

While the previous sub-section provides evidence that conditional on the ex-ante risk of testing positive, genetic test outcomes are close to randomly assigned, the availability of panel data enables me to further strengthen my identification strategy by controlling for time-invariant unobserved characteristics.

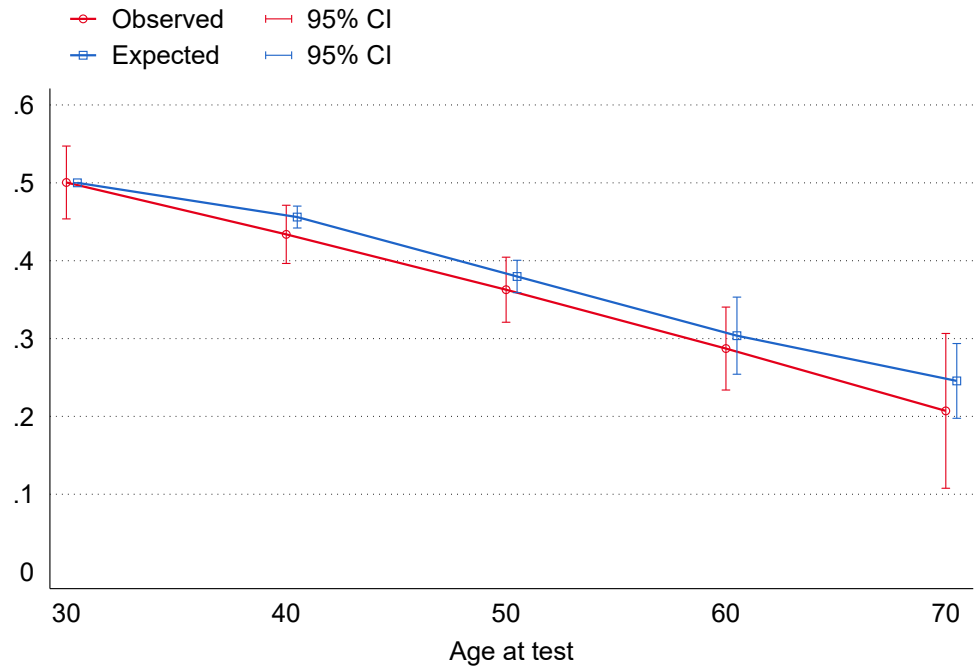
Controlling for individual fixed effects is beneficial for at least two reasons. First, it helps to

[7] Cumulative cancer incidence among individuals who carry the MLH1 Lynch mutation (the most frequent mutation in my sample) is estimated to reach 18%, 37%, 58% and 74% by the age of 40, 50, 60 and 70, respectively (Dominguez-Valentin et al., 2020, , Table S2, column 1). As these figures are based on cancer incidence in individuals who are participating in regular cancer screening and whose mutation status was confirmed, they might underestimate true cancer incidence for Lynch positive individuals, especially in older cohorts. Therefore, the *expected* share of positive-tested individuals on Figure 1 might be slightly overestimated.

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

	Variable	Positive	s.e.	Constant	s.e.	N
(1)	Age at test	-4.81***	(0.77)	44.90	(0.53)	939
(2)	MLH1/MSH2 mutation	-0.06**	(0.03)	0.86	(0.01)	939
(3)	Female	-0.03	(0.03)	0.55	(0.02)	939
(4)	Year of DNA test	0.11	(0.30)	2002.61	(0.18)	939
(5)	# siblings	-0.06	(0.16)	4.35	(0.11)	751
(6)	Maternal inheritance	0.01	(0.04)	0.44	(0.02)	827
(7)	Parent with cancer before 70	0.04	(0.03)	0.80	(0.02)	827
(8)	Has child	-0.03	(0.03)	0.73	(0.02)	922
(9)	# children	-0.07	(0.08)	1.71	(0.05)	918
(10)	Partner	0.03	(0.03)	0.77	(0.02)	915
(11)	Working	-0.03	(0.03)	0.80	(0.02)	619
(12)	Annual salary (EUR)	-30	(2077)	27281	(1102)	619
(13)	Household disposable income (EUR)	-47	(2059)	45181	(1495)	482
(14)	Financial assets (EUR)	-3904	(18606)	66296	(14266)	226
(15)	Homeowner	0.02	(0.04)	0.74	(0.02)	580

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.
The control variables are as follows. (1) Age at test: mutation type and gender; (2) MLH1/MSH2 mutation: age at test fixed effects (in groups of 10 years) and gender; (3) Female: mutation type and age at test fixed effects; (4) Year of DNA test: mutation type, age at test, and gender fixed effects; (5)-(15): mutation type, 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)-(10) at least 20 years old, (11)-(12) 25 to 64 years old and diagnosed before turning 61 years old, (13)-(15) at least 25 years old and classified as the household head or their partner. The varying number of observations is due to the different sample periods (e.g., wealth variables are only available from 2006 on) and the differences in sample selection criteria. Robust standard errors are presented in parentheses, * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$



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 2nd degree age polynomial. *Expected* is calculated using Bayes' rule assuming a 50% probability of carrying the mutation at birth, cumulative cancer incidence estimates for LS (Dominguez-Valentin et al., 2020), and cumulative cancer incidence estimates for the general population (as reported in Figure 1).

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

remove any remaining pre-testing differences in the level of outcome variables, which might arise due to the sample size or failures of randomization due to any of the reasons discussed above. The main identifying assumption becomes the parallel trends assumption: in the absence of treatment (testing positive and not negative), the difference between the positive-tested and the negative-tested group is constant over time.

Second, controlling for individual fixed effects can also address changes in the composition of positive-tested and negative-tested individuals over time. 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 follow individuals in my sample as long as their health is unimpaired, i.e., they do not develop cancer or do not undergo any preventive surgeries.[8]

Given this background, my baseline regression model is,

$$y_{i,t} = \alpha_i + \beta_{i,t} + \gamma_{K_{i,t},i} + \sum_{k=-6, k\neq -1}^{14} \delta_k \{K_{i,t} = k\} \cdot T_i + \epsilon_{i,t} \tag{1a}$$

where i indexes individuals, and t indexes calendar years. α_i are individual fixed effects,

[8] In general, my results are not sensitive to censoring observations after the first cancer diagnosis or preventive surgery. Robustness test results currently under tabulation.

while $\beta_{i,t}$ stand for year fixed effects and gender-age fixed effects. $K_{i,t}$ represents the relative years since the genetic test, where $K_{i,t} = 0$ is the year of the test. $\gamma_{K_{i,t},i}$ stand for relative year*gender, relative year*age at testing (in groups of 10 years), and relative year*mutation type (MLH1, MSH2, etc.) fixed effects. T_i is the treatment indicator that takes the value 1 for positive-tested and 0 for negative-tested individuals. δ_k are the coefficients of interest, they represent the differential time trend (in relative years) of positive-tested individuals. δ_{-1} (the difference between positive- and negative-tested in the year before testing) is normalized to 0 due to the individual fixed effects. $\epsilon_{i,t}$ are 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 is only available from end-2005, whereas 75% of individuals in my sample were tested before 2006. Using group fixed effects (a difference-in-differences estimator) instead of individual fixed effects enables me to exploit the information provided by individuals with observations only in the post-testing period. The resulting model is,

$$y_{i,t} = \alpha_{T_i} + \beta_{i,t} + \gamma_{K_{i,t},i} + \sum_{k=-6, k\neq -1}^{14} \delta_k \{K_{i,t} = k\} \cdot T_i + \epsilon_{i,t} \tag{1b}$$

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 yield similar results to the primary specification.

While models 1a and 1b make it possible to evaluate pre-trends by observing the estimated δ_k for all $k < 0$, s (Borusyak and Jaravel, 2017) note, it does not estimate the treatment effects efficiently, and given no pre-trends all δ_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 for positive-tested and negative-tested individuals,

$$y_{i,t} = \alpha_i + \beta_{i,t} + \gamma_{K_{i,t},i} + \delta\{K_{i,t} \geq 0\} \cdot T_i + \epsilon_{i,t} \tag{2a}$$

$$y_{i,t} = \alpha_{T_i} + \beta_{i,t} + \gamma_{K_{i,t},i} + \delta\{K_{i,t} \geq 0\} \cdot T_i + \epsilon_{i,t} \tag{2b}$$

where δ represents the average treatment effect for the first 15 years (0 to 14) of the treatment. 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),

$$y_{i,t} = \alpha_i + \beta_{i,t} + \gamma_{K_{i,t},i} + \delta_s\{K_{i,t} \in [0,4]\} \cdot T_i + \delta_m\{K_{i,t} \in [5,9]\} \cdot T_i + \delta_l\{K_{i,t} \in [10,14]\} \cdot T_i + \epsilon_{i,t} \tag{3a}$$

$$y_{i,t} = \alpha_{T_i} + \beta_{i,t} + \gamma_{K_{i,t},i} + \delta_s\{K_{i,t} \in [0,4]\} \cdot T_i + \delta_m\{K_{i,t} \in [5,9]\} \cdot T_i + \delta_l\{K_{i,t} \in [10,14]\} \cdot T_i + \epsilon_{i,t} \tag{3b}$$

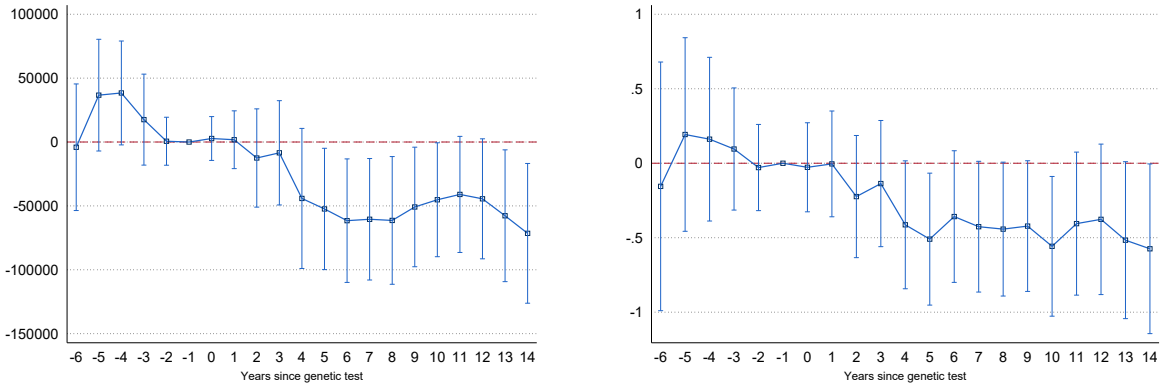
where δ_s, δ_m, and δ_l identify the short-, medium-, and long-term treatment effects, respectively.

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 $\gamma_{K_{i,t},i}$.

5 Wealth accumulation and portfolio composition

Keeping the medical risks associated with Lynch syndrome in mind, in the main part of the analysis, I study the effects of learning to be a Lynch mutation carrier on households' financial, economic and demographic decisions. I start by estimating the effects on household wealth accumulation. Figure 4 presents estimates from Model 1b. The figure reveals a strong decrease in household financial assets following genetic testing, measured either in euros (panel a) or in logs (panel b). Financial assets include both bank deposits/savings and risky financial securities.

Column 1 of Table 2 presents difference-in-differences treatment effect estimates from Model 2b based on the same observations as in panel (a) of Figure 4. In the first five years following testing, households of Lynch-positive individuals have on average EUR 27,000 lower financial wealth than household of negative-tested people. During the next five years the difference increases to EUR 70,000, where it stabilizes. On average, during the first 15 years after testing, Lynch-positive individuals have EUR 57,000 lower financial assets than those who tested negative. This is an economically significant difference equal to about 1.25 year median disposable household income in my sample. Appendix A Table A1 presents robustness tests.



(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 effects of testing positive on financial assets and log financial assets

Table 2: Effects of testing positive on financial wealth accumulation

	(1) All	(2) Male	(3) Female	(4) No child	(5) Had child
DiD	-57058***	-52959**	-51535	-113964***	-43295**
	(20839)	(25502)	(34417)	(43636)	(20651)
t=0-4	-27402	-34641	-13418	-81375*	-17252
	(17061)	(27660)	(22762)	(48865)	(15798)
t=5-9	-69587***	-60845*	-70662*	-141560***	-49568**
	(24038)	(31397)	(39384)	(52632)	(23590)
t=10-14	-63079***	-55267**	-57875	-103186**	-54895**
	(23431)	(24523)	(40680)	(40742)	(24830)
Cons	96244***	94791***	94697***	117815***	93274***
	(10785)	(14603)	(15456)	(28910)	(10856)
N	9093	4200	4893	2473	6619
Ind	858	399	458	239	616

The table presents the effects of testing positive for the suspected Lynch syndrome gene mutation on financial assets. Financial assets include bank deposits and risky financial securities (stocks, bonds, and investments in mutual funds). The row *DiD* reports the coefficient δ 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 δ_s, δ_m, δ_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. Column (1) includes all observations that meet these criteria. Columns (2) and (3) restrict the sample to males and females, respectively. Columns (4) and (5) restrict the sample to those who had no children and those who did have children before testing, respectively. Standard errors clustered at the level of the individual are reported in parentheses, * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Sub-sample analysis reveals that the financial wealth of males and females are similarly affected (columns 2 and 3). On the contrary, when I partition the sample to individuals who had no children before testing (column 4) and individuals who had at least one child (column 5), I find that childless individuals are affected to a much greater extent (albeit this difference is not statistically significant). This finding might be explained by stronger bequest motives among individuals with children, although having no children before testing does not exclude having children after testing; moreover, individuals without children can also exhibit bequest motives (Kopczuk and Lupton, 2007).

Table 3 studies treatment effects on other wealth components. As the table reveals, both bank deposits/savings and financial securities are negatively affected by a LS diagnosis (columns 1 and 2). As Section 8 will document, financial securities experience a larger relative decline than safe bank deposits/savings, which leads to a reduction in the risky share of financial assets. Column (3) shows a slight negative impact on home ownership; however, these effects are not statistically significant during any time period at any conventional level. Finally, column (4) reveals no effect on the value of other real estate, such as second homes, holiday homes, or investment properties.

Table 3: Effects of testing positive on household wealth components

	(1) Deposits	(2) Securities	(3) Home owner	(4) Other RE
DiD	-20603**	-29108**	-.042	-2335
	(8569)	(12517)	(.033)	(13530)
t=0-4	-7955	-15587	-.032	-5843
	(7681)	(10454)	(.033)	(12683)
t=5-9	-21745**	-36776**	-.046	-2461
	(9173)	(14419)	(.037)	(14607)
t=10-14	-27832***	-29683**	-.057	162
	(9983)	(13970)	(.041)	(14964)
Cons	54410***	34904***	.81***	24164***
	(4152)	(6354)	(.011)	(5295)
N	9093	9093	7949	9093
Ind	858	858	561	858

The table presents the effects of testing positive for the suspected Lynch syndrome gene mutation on household wealth components. The rows *t=0-4*, *t=5-9*, and *t=10-14* report the coefficients δ_s, δ_m, δ_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. Columns (1) and (2) examine the effects on the two components of financial assets, bank deposits/savings and financial securities, respectively. Financial securities comprise mostly stocks and investments in mutual funds but may also include bonds. The dependent variable in column (3) is a binary indicator of home ownership. Column (4) studies the effects on the value of other real estate owned, such as second homes, holiday homes, investment properties. Variables in columns (1), (2) and (4) are winsorized at the 99^{th} percentile. Standard errors clustered at the level of the individual are reported in parentheses, * p < 0.1, ** p < 0.05, *** p < 0.01

Having documented a negative effect on financial wealth accumulation, I turn to studying four channels that could give rise to these results. These are changing household composition, decreasing labor income, lower savings rates, and lower portfolio allocation to risky financial assets.

6 Household composition

In this section, I describe the impact of testing positive for Lynch syndrome on two important early-life demographic outcomes, partnership formation, and childbearing.

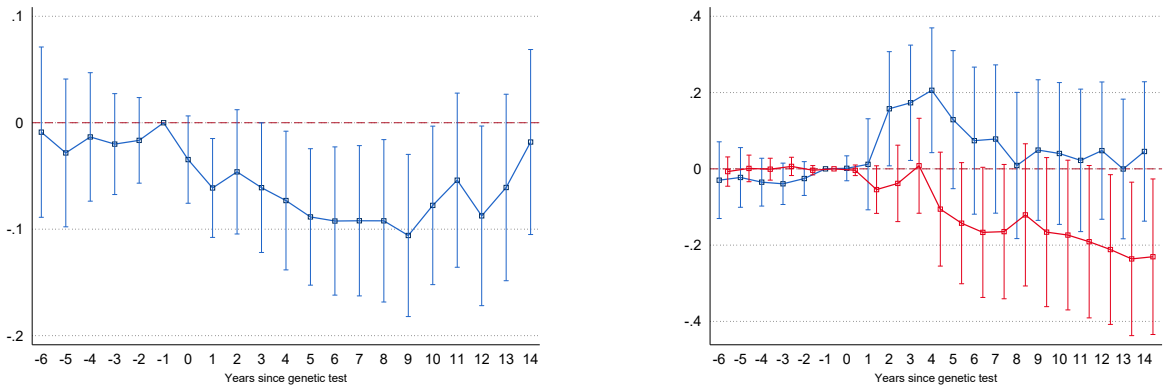
6.1 Partnership formation

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.

Figure 5 presents estimates from Model 1a of the dynamic effects of testing positive on having a partner (panel a) and having any children (panel b). Both samples are restricted to individuals who were tested when no older than 45 years old as it is mostly in the earlier life when individuals form partnerships and have children. Furthermore, the sample of panel (b) is restricted to individuals who had no children when undergoing genetic testing. As panel (a) illustrates, positive-tested individuals face an immediate reduction in the probability of having a partner following genetic testing.[9] The negative effects grow over time, reaching about 10 pp by year 10; however, we can observe some recovery in the long run.[10]

[9] Further analysis (currently under tabulation) reveals that the partnership probabilities of individuals both with and without a partner before genetic testing are negatively affected.

[10] I find a much milder recovery in case I do not censor my sample at the time of the first preventive surgery. This may suggest that the time-variant part of the propensity to have a partner (which is not absorbed by individual fixed effects) might be correlated with the decision to undertake preventive surgeries. Individuals with a low propensity to have a partner might dynamically select out of the sample of positive-tested people as they undertake preventive surgeries (it is mostly women who undertake preventive surgeries to prevent ovarian and endometrial cancer).



(a) Having a partner (binary)

(b) Having children (binary)

The figure shows the dynamic effects of testing positive on having a partner (panel a) and 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. Panel (a) includes only individuals who were not older than 45 years when undergoing genetic testing. Panel (b) considers only individuals who had no children prior to testing. Furthermore, the *blue line* on panel (b) shows estimates for a subsample who were tested not older than 30 years old, while the *red line* shows estimates for those who were tested between 31 and 45 years old. The figure presents 95% confidence intervals based on standard errors clustered at the individual level.

Figure 5: Dynamic effects of testing positive on having a partner and having children

Table 4 summarizes the treatment effects on partnership formation using Model 2a, 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 2) compared to those who tested when younger (column 3). 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. On the contrary, as columns (6) and (7) reveal, individuals who already had any children before genetic testing face a substantially smaller negative effect on partnership probabilities than those without children. Finally, column (8) combines the samples of columns (2) and (6), i.e. studies treatment effects in the sub-sample of individuals who were either tested at an older age or who had children before testing (about $2/3^{rd}$ of my total sample). As the results reveal, the partnership probabilities of these individuals are on average little affected. In subsequent robustness tests, I use this sample to verify that my results on wealth accumulation (and other outcomes) are not purely driven by changes in household composition.

6.2 Childbearing

Being diagnosed with a Lynch mutation may also influence the propensity to have children at least for three reasons. First, as discussed above, positive-tested individuals in the reproductive age (here defined as 45 years old or younger), face a lower probability of having a partner

Table 4: Effects of testing positive on having a partner

	(1) All	(2) > 45	(3) ≤ 45	(4) Female	(5) Male	(6) Child	(7) No child	(8) Unaffected
DiD	-.031	.028	-.056**	-.063*	-.044	-.03	-.1*	-.014
	(.02)	(.03)	(.026)	(.035)	(.04)	(.027)	(.054)	(.019)
t=0-4	-.015	.034	-.041*	-.046	-.037	-.051**	-.044	-.012
	(.018)	(.025)	(.024)	(.032)	(.037)	(.025)	(.053)	(.017)
t=5-9	-.05**	.022	-.08**	-.086**	-.062	-.058*	-.16**	-.023
	(.025)	(.04)	(.033)	(.042)	(.051)	(.035)	(.063)	(.024)
t=10-14	-.032	.021	-.047	-.063	-.031	-.0082	-.12*	-.0027
	(.029)	(.045)	(.037)	(.053)	(.054)	(.037)	(.075)	(.027)
Cons	.79***	.79***	.8***	.82***	.77***	.92***	.61***	.86***
	(.005)	(.0054)	(.0079)	(.01)	(.012)	(.0074)	(.019)	(.0043)
N	16270	6043	10227	5663	4564	6126	3885	12374
Ind	937	353	583	332	249	342	224	709

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 δ 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 δ_s, δ_m, δ_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 older than 45 years, and not 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 females and males. Columns (6) and (7) include individuals who had at least one children and those who had no children before testing, respectively. Column (8) combines the samples of column (2) and column (6). Standard errors clustered at the level of the individual are reported in parentheses, * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

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 Lynch syndrome 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. The figure illustrates two patterns based on the age at which individuals underwent genetic testing. Among people who were tested 30 years old or younger (*blue line*), positive-tested shift childbearing to a younger age but eventually have no fewer children than negative-tested. On the contrary, among individuals who were tested between 31 and 45 years old (*red line*), positive-tested face an about 20 pp lower probability of ever having children.

Columns (1) and (3) of Table 5 summarize the results presented in panel (b) of Figure 5 based on Models 2a and 2a. Columns (2) and (4) study the effects on the number of children instead of having any children. The 20 pp lower probability of ever having children that individuals tested between 31 and 45 years old face (column 3) is accompanied by 0.39 fewer children (column 4). Negative-tested individuals in the samples of columns (3) and (4) have on average 2.2 children conditional on having any children. If the effect on having any children would be independent of the number of children conditional on having any children, we would expect 0.44 fewer children among positive-tested individuals. The fact that the treatment effect on the number of children is smaller than 0.44 might suggest that testing positive for LS has a larger negative effect on the propensity of having children for individuals who would otherwise have fewer children. Finally, column (5) examines treatment effects on the number of children among individuals who already had at least one child before genetic testing. As the column shows, this intensive margin of childbearing is little affected.

Can the changing household composition documented in this section completely account for the lower wealth accumulation of Lynch-positive households? My results suggest otherwise. I employ two strategies to disentangle the effects of changing household composition from the effects of other economic channels. First, I control for partnership status and having children in my regression models, while keeping in mind that these controls may also be affected by the treatment. Second, I consider a sub-sample of individuals who were tested after turning 45 years old or who already had at least one child before testing. This sub-sample constitutes about $2/3^{rd}$ of my complete sample. As column (8) of Table 4 reveals, the probability of having a partner are little affected among these individuals. Neither is childbearing, given that individuals in my sample who are older than 45 years rarely have new children, and as column (5) of Table 5 shows the number of children among people who already had children before testing is barely affected. Table A2 in Appendix A presents robustness tests where I apply these two strategies to four of my main dependent variables. The results reveal that changing household composition only

Table 5: Effects of testing positive on having children

	20-30 yo childless		31-45 yo childless		Had child
	(1) Has child	(2) # children	(3) Has child	(4) # children	(5) # children
DiD	.096	.22	-.12*	-.2*	-.036
	(.068)	(.14)	(.06)	(.11)	(.056)
t=0-4	.13**	.16*	-.037	-.062	-.028
	(.061)	(.093)	(.035)	(.046)	(.047)
t=5-9	.091	.24	-.15*	-.24	-.045
	(.09)	(.18)	(.081)	(.15)	(.062)
t=10-14	.053	.28	-.2**	-.39**	-.037
	(.091)	(.22)	(.094)	(.2)	(.072)
Cons	.29***	.48***	.15***	.26***	2.1***
	(.026)	(.055)	(.018)	(.034)	(.014)
N	2179	2158	1892	1890	6705
Ind	121	121	101	101	354

The table presents the effects of testing positive for the suspected Lynch syndrome gene mutation on having children. *Has child* is a binary indicator of having any children, while *# children* refers to the number of children. .The row *DiD* reports the coefficient δ 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 δ_s, δ_m, δ_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 (4) include individuals who had no children before testing. Columns (1) and (2) include people who were tested between 20 and 30 years old, while columns (3) and (4) consider people tested between 31 and 45 years old. Column (5) include people who already had children before testing and who were tested not older than 45 years. Standard errors clustered at the level of the individual are reported in parentheses, * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

accounts for a minor share of the effects that I document.

7 Labor and household income

Next, I turn to estimate the impact of testing positive for LS on household income and its main constituents, the labor income of the tested individual and their partner.

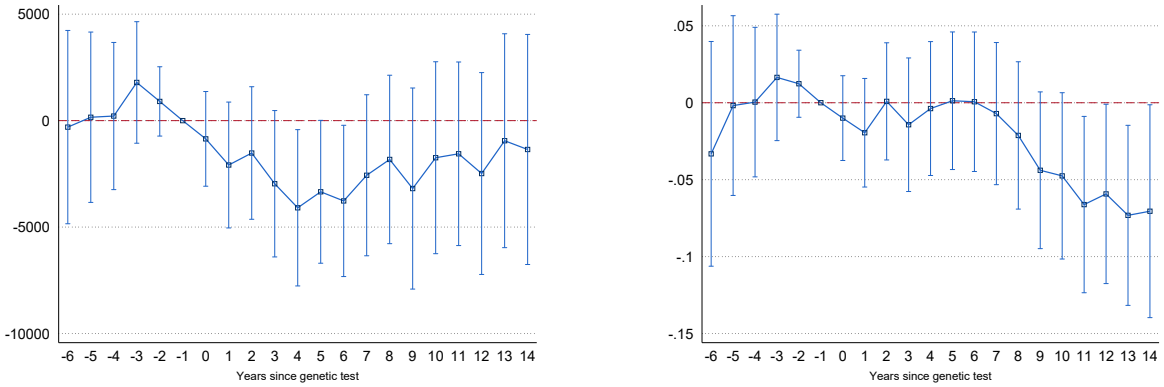
7.1 Own labor income and entrepreneurship

Economic theory predicts that decreasing life expectancy may lead to a lower labor supply (early retirement) as the need for retirement savings is lower (Bloom et al., 2003). Figure 6 indeed reveals a negative effect of testing positive on labor income (panel a), and the probability of being an entrepreneur (panel b). While labor income exhibits a rapid drop following genetic testing (and some recovery starting from four years after testing), the probability of working as an entrepreneur is only affected in the long run.

Table 6 presents difference-in-differences estimates based on Model 2a. As column 1 reveals,

positive-tested individuals earn on average EUR 2,900 lower labor income during the first 15 years following testing. This is equivalent to about 12% of the median labor income in my sample. The negative effect is only present for males (columns 2-3). Men earn on average 6,600 EUR less during the first 15 years, equivalent to about 17% of the median male labor income in my sample. The different effects might be explained by the stronger labor market attachment of males in the Netherlands during the sample period.[11]

Turning to entrepreneurship, while I do not find a statistically significant effect on the average probability of working as an entrepreneur during the first 15 years after testing, column (4) of Table 6 shows that 10 to 14 years after testing, positive-tested individuals are about 6 pp less likely to be entrepreneurs. Although men's entrepreneurial participation is also slightly negatively affected (not statistically significant), it is women who are most impacted.



(a) Labor income (EUR)

(b) Entrepreneruship (binary)

The figure shows the dynamic effects of testing positive on labor income in EUR (panel a) and the probability of working as an entrepreneur (panel b). Coefficient estimates from Model 1a are presented. *Labor income* equals to the pre-tax salary if an individual is working and to zero otherwise. *Entrepreneurship* (binary) is determined by the annual socio-economic classification of Statistics Netherlands, primarily based on whether the individual had any profit/loss from entrepreneurial activities in the given year. 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 effects of testing positive on labor income and entrepreneurship

Table 7 studies the components of male labor income. The drop in male labor income can be explained both by a somewhat lower probability of working (column 2, not statistically significant) and by reduced labor earnings (salary) conditional on working (column 3). Columns (4) to (7) further decompose salary (column 3) into measures of the intensity of working and wages. Column (4) studies treatment effects on full-time equivalent days, a measure which reflects both the number of annual working days and the intensity (FTE) of employment. In

[11] Males work in 73% of all person-year observations that meet the sample restriction criteria in Table 6, compared to 65% for females. In the Netherlands part-time work is prevalent, mostly among female employees. In my sample, conditional on working, men's full-time equivalent equals on average 92% compared to 66% for women. Mean labor income (including working and non-working individuals) reaches EUR 15,000 for females, and EUR 33,000 for males.

Table 6: Effects of testing positive on labor income and entrepreneurship

	Labor income (EUR)			Entrepreneurship		
	(1) All	(2) Male	(3) Female	(4) All	(5) Male	(6) Female
DiD	-2892*	-6580**	-296	-.022	-.0019	-.036
	(1617)	(3167)	(1134)	(.023)	(.037)	(.024)
t=0-4	-2833*	-6529**	-270	-.011	.007	-.026
	(1477)	(3018)	(1031)	(.022)	(.037)	(.024)
t=5-9	-3420*	-7694**	-348	-.016	.0079	-.031
	(1941)	(3709)	(1391)	(.026)	(.042)	(.029)
t=10-14	-2115	-4751	-270	-.063**	-.045	-.075**
	(2324)	(4238)	(1938)	(.029)	(.046)	(.035)
Cons	29062***	42611***	17250***	.1***	.12***	.082***
	(481)	(984)	(324)	(.0071)	(.012)	(.0072)
N	12341	5817	6524	10694	4972	5722
Ind	832	390	441	811	378	431

The table presents the effects of testing positive for the suspected Lynch syndrome gene mutation on labor income (columns 1 to 3) and on being an entrepreneur (columns 4 to 6). *Labor income* equals to the pre-tax salary if an individual is working and to zero otherwise. *Entrepreneurship* (binary) is determined by the annual socio-economic classification of Statistics Netherlands, primarily based on whether the individual had any profit/loss from entrepreneurial activities in the given year. The row *DiD* reports the coefficient δ 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 δ_s, δ_m, δ_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

the first 15 years after testing, positive-tested working males work on average 20 fewer full-time equivalent days per year than negative-tested ones. This effect is equal to about 5.5% of the median male FTE days in my sample. Columns (5) and (6) further split FTE days into the calendar days worked during the year (column 5) and the full-time equivalent of the employee (column 6).[12] The results reveal that the negative effects on FTE days arise as a composite effect of working few days in a given year, and working fewer hours conditional on the number of days worked. Finally, column (7) studies the effects on wages, which are defined as Salary divided by FTE days, and reports no statistically significant effects.

While Table 6 studies the effects of a Lynch diagnosis on labor income in terms of the years passed since diagnosis, Table 8 presents treatment effects along the life-cycle, based on Model 2a where the difference-in-differences indicator is interacted with age group indicators. The table shows that it is in the pre-retirement period when working men reduce their labor supply

[12]The number of calendar days worked (more precisely being contractually employed) may be lower than the calendar days in the year (365/366) for various reasons. These include working non-consecutive spells (e.g. switching employers throughout the year, working on-call), becoming unemployed, or retiring during the year. FTE can be lowered by working fewer hours.

Table 7: Effects of testing positive on male labor market outcomes

The table presents the effects of testing positive for the suspected Lynch syndrome gene mutation on male labor income and its components. *Labor income* (column 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. *FTE days* (column 4) are the number of full-time equivalent days, while *Days* (column 5) are the number of calendar days and individual worked in the given year. *FTE* (column 6) is the full-time equivalent, e.g. an FTE of 0.5 signals half of a full work load (part-time). Daily wage is *Salary* divided by *FTE days*. The row *DiD* reports the coefficient δ 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 δ_s, δ_m, δ_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. 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, * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

(on the intensive margin) the most. Between the ages 60 and 64, positive-tested men work on average 49 fewer FTE days (column 3), with approximately half of this reduction originating from working fewer days (column 4) while the other half from working at a lower FTE (column 5).

7.2 Partner's labor income

Having studied tested individuals' labor response, I turn to estimating the effects on their partners' labor outcomes. This is important because most individuals in my sample (77%) have a partner when they undergo genetic testing, and partners may make labor market choices collectively (Blundell and MaCurdy, 1999).

Table 9 reports that it is only the labor income of positive-tested women's partners (98% of whom are male) that is affected (column 3). They have on average EUR 9,200 lower labor income during the first 15 years after testing compared to partners of negative-tested women. Columns (4) to (7) decompose this negative effects to the probability of working at all (column 4), salary conditional on working (column 5), the FTE days worked (column 6), and the daily wage (column 7). While the extensive margin of labor participation is unaffected, both FTE days (intensive margin) and daily wages are negatively impacted (albeit the former not statistically significantly).

7.3 Household income

Lower probability of having a partner, lower own labor income (for men) and lower labor income of the partner (for women) suggest that positive-tested individuals would exhibit a lower household income. Indeed, as Figure 7 presents, the household income of positive-tested individuals experience a substantial drop following genetic testing compared to the income of negative-tested people. Most of this drop occurs during the first four years after genetic testing, from which point on the difference stabilizes. These dynamics are approximately in line with the treatment effect dynamics observed for labor income (Figure 6). During the first 15 years following genetic testing, positive-tested households have on average about 15% or EUR 12,000

Table 8: Effects of testing positive on male labor income over the life-cycle

	(1) Working	(2) Salary	(3) FTE days	(4) Days	(5) Part-time
25-34	-.055	1818	-.64	8.9	-.0089
	(.05)	(3435)	(12)	(8.4)	(.023)
35-49	-.029	-5901*	-19**	-8.8*	-.017
	(.037)	(3466)	(9.1)	(4.9)	(.017)
50-59	-.014	-3485	-18**	-8.6	-.019
	(.048)	(3103)	(9.1)	(5.7)	(.018)
60-64	.023	-8608*	-49***	-25*	-.079**
	(.079)	(4629)	(17)	(14)	(.034)
Cons	.83***	50746***	342***	354***	.96***
	(.011)	(831)	(2.7)	(1.4)	(.0051)
N	5809	4747	4063	4550	4063
Ind	390	358	341	356	341

The table presents the effects of testing positive for the suspected Lynch mutation on male labor earnings and its constituents over the life-cycle, based on Model 2a where the difference-in-differences indicator is interacted with age groups. *Working* (column 1) is an indicator whether the individual had non-zero pre-tax salary in the given year. *Salary* (column 2) stands for pre-tax salary. *FTE days* (column 3) are the number of full-time equivalent days, while *Days* (column 4) are the number of calendar days and individual worked in the given year. *FTE* (column 5) is the full-time equivalent, e.g. an FTE of 0.5 signals half of a full work load (part-time). *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

lower disposable household income. Table A2 of Appendix A presents evidence that household composition can only account for a small share of these negative effects, which continue to hold once controlling for having a partner and children, and in the sub-sample of individuals whose household composition is little affected.

Table 10 summarizes the treatment effects on disposable household income and its two main components, the tested person's and their partner's labor income. 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. This exclusion mostly concern instances of unearned 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). Restricting our attention to columns 4 to 6, the table 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. The table also reiterates the finding that testing positive for Lynch syndrome predominantly affects own labor income for positive-tested males and the partners' (who are mostly male) labor income for positive-tested females.

Table 9: Effects of testing positive on partners' labor market outcomes

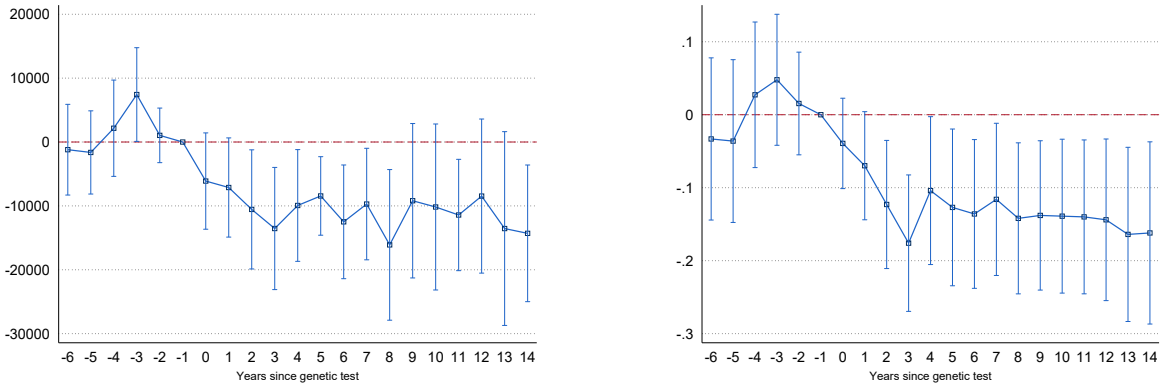
	Labor income (EUR)			Female's partner			
	(1) All	(2) Male	(3) Female	(4) Working	(5) Salary	(6) FTE days	(7) Daily wage
DiD	-5357*	-280	-9198*	.0031	-13526	-3.2	-11*
	(3188)	(2024)	(5245)	(.024)	(9068)	(7)	(6)
t=0-4	-8046*	-452	-13801*	-.0047	-15935	-2.5	-10*
	(4867)	(1931)	(8205)	(.023)	(11231)	(6.9)	(5.7)
t=5-9	-1538	36	-2015	.019	-9440	-1.6	-9.8
	(2234)	(2319)	(3591)	(.031)	(7023)	(8.8)	(7.5)
t=10-14	-4907*	-370	-9312*	-.0036	-13872*	-10	-17*
	(2940)	(2828)	(5120)	(.04)	(7582)	(9.4)	(8.7)
Cons	34952***	16934***	50727***	.84***	61452***	314***	161***
	(928)	(619)	(1461)	(.0067)	(2528)	(2.1)	(1.8)
N	9764	4604	5160	5160	4314	3614	3347
Ind	742	342	398	398	359	335	332

The table presents the effects of testing positive for the suspected Lynch syndrome gene mutation on the labor outcomes of the tested person's partner. Columns (1) to (3) presents estimates on labor income, which equals to the pre-tax salary of the partner if they are working and to zero otherwise. Column (1) include the partners of all individuals who were tested at the age of 60 or younger, while columns (2) and (3) only include tested males (and their mostly female partners) and females (and their mostly male partners), respectively. Columns (4) to (7) study the labor income components of the partners of tested females. *Working* (column 4) is an indicator whether the partner had non-zero pre-tax salary in the given year. *Salary* (column 5) stands for pre-tax salary. *FTE days* (column 6) are the number of full-time equivalent days. Daily wage (column 7) is *Salary* divided by *FTE days*. The row *DiD* reports the coefficient δ 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 δ_s, δ_m, δ_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 partner-year observations when the partner was between 25 and 64 years old. Years when an individual had no partner are treated as missing. Standard errors clustered at the level of the individual are reported in parentheses, * p < 0.1, ** p < 0.05, *** p < 0.01

8 Savings and investment decisions

Besides changing household composition, and the drop in household income, lower wealth accumulation may also be explained by a lower savings rate and by a more conservative portfolio allocation. Results presented in panel (a) of Figure 8 and column (1) of Table 11 show that the households of positive-tested individuals indeed save a substantially lower (-13% on average during the first 15 years after testing) share of their disposable household income. This is in line with theoretical models where a lower lifespan implies a lower savings rate (e.g., Bloom et al., 2003). Accounting for changes in household composition (Table A2 of Appendix A) has almost no effects on these estimates.

Regarding their financial portfolio, panel (b) of Figure 8 and column (2) of Table 11 present evidence that positive-tested households invest an about 8 pp lower share of their financial assets into financial securities (which mostly consist of investments in stocks and investment funds).

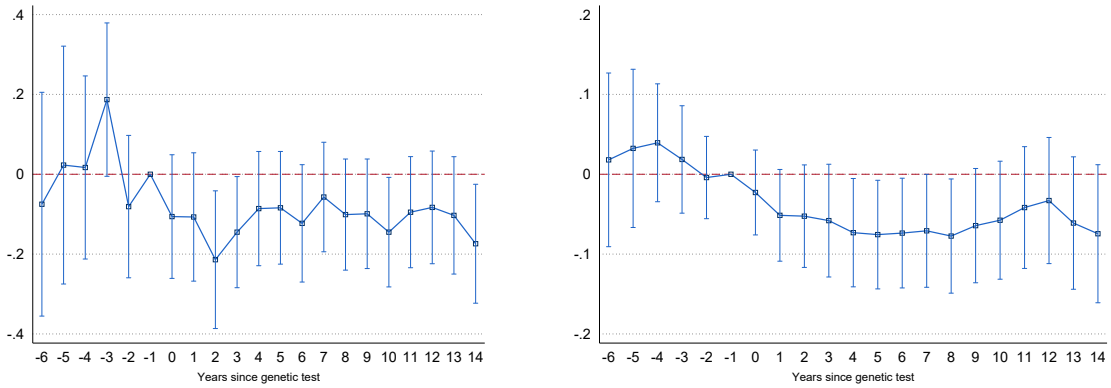


(a) Disposable household income (EUR)

(b) Log disposable household income

The figure shows the dynamic effects of testing positive on disposable household income in euros (panel a) and in logs (panel b). 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 7: Dynamic effects of testing positive on household income



(a) Savings rate out of disposable income

(b) Risky share of financial assets

The figure shows the dynamic effects of testing positive on the savings rate out of disposable income (panel a) and the risky share of 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 8: Effects of testing positive on savings out of disposable income and the risky share of financial assets

This is an economically (and statistically) significant effect given the unconditional risky share of 12 pp in my sample. The observed drop in the risky share is in line with models where due to return predictability investors should allocate substantially more to stocks if their horizon is longer (Barberis, 2000). Adjusting for potential changes in household composition has again little effects on the estimates.

Columns (3) and (4) of Table 11 splits the risky share of financial assets into an extensive margin component (stock market participation), and the risky share conditional on participation. The results show that all negative effects on the risky share are driven by a lower

Table 10: Effects of testing positive on household income and its components

	Full sample			$< 99^{pctl}$ household income		
	(1) All	(2) Male	(3) Female	(4) All	(5) Male	(6) Female
Household income (EUR)	-11701***	-14329**	-9989	-5534***	-7441***	-4262**
	(4127)	(6148)	(6110)	(1570)	(2573)	(1981)
Own labor income (EUR)	-2742*	-6086*	-512	-3010	-6137	-1046
	(1618)	(3284)	(1148)	(1846)	(3860)	(1284)
Partner's labor income (EUR)	-5314*	-1537	-7766*	-2890*	-1887	-3013
	(2733)	(1424)	(4577)	(1523)	(1755)	(2278)
N	10271	4763	5508	10163	4725	5438
Ind	804	374	429	804	374	429

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 δ, 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. 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

probability of investing in risky securities at all.

Table 11: Effects of testing positive on savings out of disposable income and the risky share of financial assets

	(1) Savings rate	(2) Risky share	(3) Has stocks	(4) Risky share
DiD	-.13***	-.081**	-.15**	-.016
	(.041)	(.035)	(.071)	(.058)
t=0-4	-.15***	-.075**	-.12*	-.033
	(.045)	(.032)	(.067)	(.059)
t=5-9	-.1**	-.093**	-.16**	-.04
	(.043)	(.036)	(.074)	(.062)
t=10-14	-.13***	-.073*	-.17**	.031
	(.044)	(.039)	(.08)	(.066)
Cons	.037***	.15***	.38***	.37***
	(.013)	(.013)	(.026)	(.023)
N	6521	8112	8112	2673
Ind	808	846	846	386

The table presents the effects of testing positive for the suspected Lynch syndrome gene mutation on savings out of disposable household income and risky financial investments. *Savings rate* (1) is defined as savings divided by disposable household income. Savings is imputed from year-on-year changes in hosuehold 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. *Risky share* (2, 4) 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. *Has stocks* (3) is an indicator of having any risky financial securities. The row *DiD* reports the coefficient δ 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 δ_s, δ_m, δ_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 (2) to (4) include only observations when the individual had at least EUR 2,500 in bank deposits or savings. Column (4) include only observations where *Has stocks* is equal to 1. Standard errors clustered at the level of the individual are reported in parentheses, * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

9 The effects of 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 home ownership. 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 12 reveal that while some effects that I have previously documented are mainly the outcome of a negative effect on positive-tested individuals (e.g., probability of having children, column 2) or a positive effect on negative-tested ones (e.g., labor supply, column 5), most effects arise as the difference of opposite sign impacts. For example, compared to the general Dutch population, positive-tested individuals decrease their allocation to risky financial assets following genetic testing, while negative-tested ones increase it (column 7).

Table 12: Effects of testing positive or negative compared to the general population

	(1) Partner	(2) Child	(3) HHI	(4) Labor	(5) FTE days	(6) Assets	(7) Risky	(8) Savings
Positive	-.021	-.12***	-2126*	-2541	-.67	-10662	-.032	-.079***
	(.019)	(.045)	(1245)	(2401)	(5.5)	(8918)	(.028)	(.03)
Negative	.017	-.0016	7763**	1911	14**	15819	.035*	.053*
	(.017)	(.045)	(3616)	(2038)	(6.2)	(10996)	(.018)	(.029)
Cons	.77***	.16***	49705***	38586***	333***	61934***	.1***	.006***
	(.00045)	(.0011)	(92)	(59)	(.19)	(905)	(.0017)	(.0015)
N	211949	38817	211953	122232	82537	186981	156425	132506
Ind	11509	1986	16139	7874	6788	17733	16969	16590

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). *Partner* (1) is a binary indicator if the individual had a (married or non-married) partner. *Child* (2) is a binary indicator if the individual had any children. *HHI* (3) stands for disposable household income, which is the sum of all labor and non-labor income (including transfers and capital income) of the household minus taxes paid. *Labor* (4) represents labor income, which equals the pre-tax salary if the individual was working and zero otherwise. *FTE days* (5) are the number of full-time equivalent days (missing if an individual was not working). *Assets* (6) 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* (7) stands for the share of risky financial securities among total financial assets. *Savings* (8) 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, * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

These findings suggest that genetic testing can be beneficial for the socioeconomic outcomes of negative-tested individuals, possibly because it eliminates the fears of facing a high risk of cancer.[13]

[13]In a sample of individuals at risk of Lynch syndrome, Hadley et al. (2008) report that 86% estimated their colon cancer risk to be higher than the average for a person their age.

10 Conclusions

This paper studies the causal effects of life expectancy on financial, economic, and demographic decisions over the life-cycle, exploiting a natural experiment presented by predictive genetic testing. Individuals in my sample face a genetic disease that can substantially increase their lifetime cancer risk, and reduce their life expectancy. Merging genetic data with rich administrative data, I study how individuals' behavior changes once they learn about their genetic test outcome and future cancer risks.

I find that in the decades following testing, individuals who turn out to carry the gene mutation accumulate lower financial wealth. I document fours channels that explain lower wealth accumulation under reduced life expectancy, lower savings rates, safer portfolios, decreased household and labor income, and different preferences for household composition.

I argue that this paper makes two main contributions. While calibrations of life-cycle models to more accurate mortality expectations can help to explain important household finance puzzles (Heimer, Myrseth, and Schoenle, 2019), empirical evidence on the causal effects of shocks to life expectancy on economic behavior is limited. The first and foremost contribution of this paper is that it provides a sharp test for life-cycle models that predict that lower life expectancy leads to lower labor supply, reduced savings and dampened wealth accumulation over the life-cycle.

Second, this paper also provides evidence on the beneficial consequences of genetic testing for negative-tested individuals. I document a positive effect of learning about the non-carrier status, among others outcomes, on partnership formation, household income, the savings rate and investment in financial securities. This underlines that genetic testing may offer not only medical but also socio-economic benefits.

A Additional figures and tables

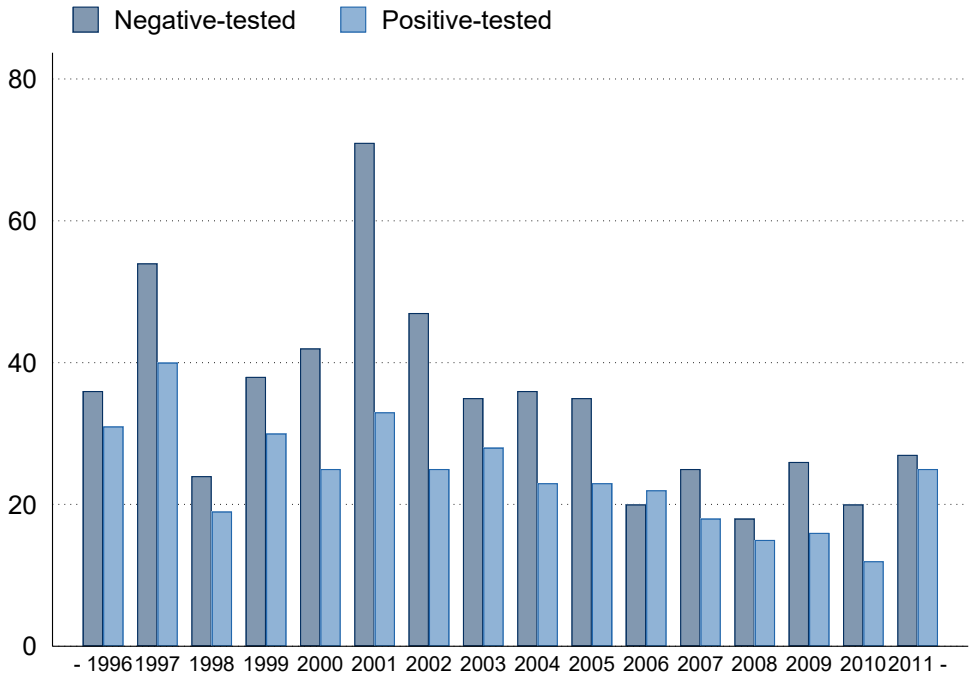


Figure A1: Distribution of positive and negative tested individuals in the sample by year of testing

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*

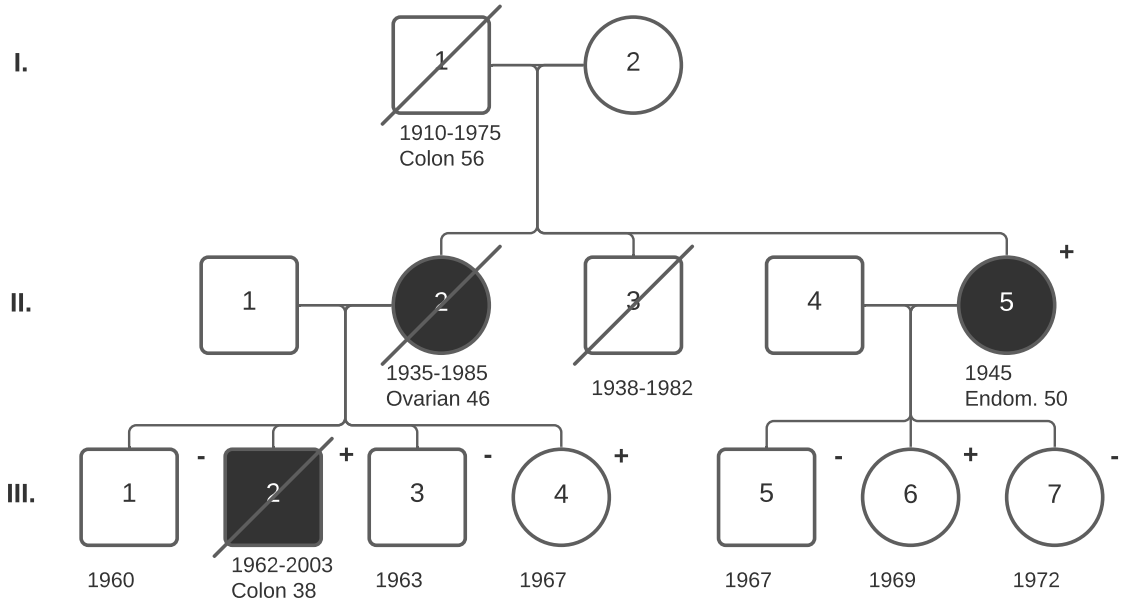
You were referred for genetic advice because several of your family members had colon cancer that DNA testing had shown is of a hereditary form. The DNA test shows that you also have this predisposition for HNPCC (also referred to as Lynch syndrome). People who have the predisposition (= the gene) for 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

Table A1: Effects of testing positive on financial assets, robustness

	(1) Baseline	(2) F.e.	(3) (1,99.5)	(4) (1,95)	(5) Log
DiD	-57058***	-60347**	-97925*	-19266**	-.43**
	(20839)	(28574)	(54745)	(9040)	(.21)
t=0-4	-27402	-49203*	-40939	-9514	-.23
	(17061)	(25447)	(46202)	(7970)	(.19)
t=5-9	-69587***	-75344**	-124895**	-20664**	-.47**
	(24038)	(33471)	(62792)	(9469)	(.22)
t=10-14	-63079***	-70670**	-106282*	-24265**	-.53**
	(23431)	(32549)	(60495)	(10478)	(.25)
Cons	96244***	97267***	135748***	60515***	10***
	(10785)	(8943)	(26740)	(3725)	(.086)
N	9093	9093	9093	9093	9042
Ind	858	850	858	858	866

The table presents the effects of testing positive for the suspected Lynch syndrome gene mutation on financial assets. Financial assets include bank deposits and risky financial securities (stocks, bonds, and investments in mutual funds). The row *DiD* reports the coefficient δ 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 δ_s, δ_m, δ_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. Column (1) presents the baseline specification where financial assets are winsorized at the 1^{st} (at 0) and 99^{th} percentiles. Column (2) controls for individual fixed effects estimating Models 2a-3a. column (3) and (4) presents results based on alternative winsorizations at the top of the distribution at the 99.5^{th} and the 95^{th} percentiles, respectively. Column (5) presents a specification where the dependent variable is log financial assets. Standard errors clustered at the level of the individual are reported in parentheses, * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A2: Controlling for the effects of having a partner and having children

Panel A

	(1) Assets	(2) Assets	(3) Assets	(4) HHI	(5) HHI	(6) HHI
DiD	-57058***	-55777***	-43591**	-11701***	-10780***	-10817**
	(20839)	(20850)	(19755)	(4127)	(4073)	(4234)
t=0-4	-27402	-26920	-16741	-10918**	-10757**	-11350**
	(17061)	(17009)	(14827)	(4603)	(4594)	(5082)
t=5-9	-69587***	-67793***	-49987**	-12378***	-10857***	-11201***
	(24038)	(23940)	(22756)	(4158)	(4069)	(4275)
t=10-14	-63079***	-61709***	-55931**	-12495**	-10616**	-8702
	(23431)	(23573)	(23823)	(5298)	(5210)	(5695)
Cons	96244***	95900***	93285***	56186***	55894***	57254***
	(10785)	(10748)	(10258)	(1283)	(1267)	(1172)
N	9093	9064	7009	10271	10246	7790
Ind	858	858	651	804	804	594

Panel B

	(1) Savings	(2) Savings	(3) Savings	(4) Risky	(5) Risky	(6) Risky
DiD	-.13***	-.12***	-.13***	-.081**	-.067**	-.08**
	(.041)	(.041)	(.047)	(.035)	(.033)	(.038)
t=0-4	-.15***	-.14***	-.17***	-.075**	-.062**	-.065*
	(.045)	(.045)	(.051)	(.032)	(.031)	(.036)
t=5-9	-.1**	-.1**	-.11**	-.093**	-.079**	-.089**
	(.043)	(.043)	(.049)	(.036)	(.035)	(.041)
t=10-14	-132***	-.13***	-.14***	-.073*	-.059	-.08*
	(.044)	(.044)	(.051)	(.039)	(.038)	(.044)
Cons	.037***	.035***	.025*	.15***	.14***	.14***
	(.013)	(.013)	(.014)	(.013)	(.012)	(.013)
N	6521	6521	5121	8112	8041	6357
Ind	808	808	617	846	846	649

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 model 2a (*HHI*) or 2b (*Assets*, *Savings*, and *Risky*) are presented. For each dependent variable three columns are presented, the first providing the baseline estimates, the second additionally controls for having a partner and having childre, while the third restricts the sample to individuals whose household composition is empirically little affected by the LS diagnosis (those who tested at the age of 46 or older and those who already had children before testing). *Assets* 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).*HHI* stands for disposable household income, which is the sum of all labor and non-labor income (including transfers and capital income) of the household minus taxes paid. *Savings* is the savings rate, defined as savings divided by disposable household income. *Risky* stands for the share of risky financial securities among total financial assets. *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

B Summary statistics, variable and dataset descriptions

Variable	Age range	Age at test	Other sample criteria	N	Mean	S.d.	10^{th} pctl.	Median	90^{th} pctl.
Having a partner (0/1)	20≤	≤45		10227	0.78	0.41	0.00	1.00	1.00
Has any children (0/1)	20≤	≤45		10859	0.68	0.47	0.00	1.00	1.00
Number of children	20≤	≤45		10767	1.47	1.22	0.00	2.00	3.00
Working (0/1)	25-64	≤60		12341	0.75	0.43	0.00	1.00	1.00
Entrepreneur (0/1)	25-64	≤60		10694	0.09	0.29	0.00	0.00	0.00
Labor income (2015 EUR)	25-64	≤60		12341	28202	29981	0	24615	59776
Salary (2015 EUR)	25-64	≤60	working	9295	37444	29109	9344	33663	65245
Fulltime equivalent days worked	25-64	≤60	working	7998	284	102	122	341	366
Days worked	25-64	≤60	working	8879	347	62	335	365	366
Part-time factor	25-64	≤60	working	7998	0.81	0.25	0.40	0.98	1.00
Wage (2015 EUR)	25-64	≤60	working	7998	127	70	67	109	202
Partner working (0/1)*	25-64	≤60	working	9764	0.78	0.41	0.00	1.00	1.00
Patner's salary (2015 EUR)*	25-64	≤60	working	7657	42515	68674	9320	34500	73512
Partner's FTE days worked*	25-64	≤60	working	6526	265	123	47	325	366
Partner's wage (2015 EUR)*	25-64	≤60	working	6060	142	208	67	111	222
Disposable household income (EUR)	25-64	≤60	HH head/partner	10271	52564	51475	23694	45252	78974
Financial assets	25≤	all	HH head/partner	9093	78428	209908	2020	24345	167038
Bank deposits/savings	25≤	all	HH head/partner	9093	48039	84580	1763	20327	112924
Financial securities	25≤	all	HH head/partner	9093	25752	120571	0	0	34046
Share of risky financial assets	25≤	all	HH head/partner; ≥EUR 2500 in deposits	7947	0.12	0.24	0.00	0.00	0.51
Has risky financial assets (0/1)	25≤	all	HH head/partner; ≥EUR 2500 in deposits	7947	0.33	0.47	0.00	0.00	1.00
Savings rate	25≤	all	HH head or partner**	6533	0.00	0.37	-0.42	0.02	0.39

Table A3: Summary statistics of the main dependent variables

* The age criterion and the working (non-zero labor income) criterion applies to the partner

** Additional sample selection criteria apply, see Table A5

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 A4: 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.	DHCR
Genetic test outcome	Mutation carrier (positive-tested) or non-carrier (negative-tested)	DHCR
Prophylactic (preventive) surgeries	All prophylactic surgeries listed with the date of operation and type of the operation (e.g., colectomy, hysterectomy)	DHCR
Cancer diagnoses	All diagnoses listed with the diagnosis date and the International Classification of Diseases (ICD) code	DHCR
Labor market and income		
Pre-tax labor income	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
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 annum.	2001-2016: BAANS./DEELTIJDFACT. 2017-2019: SPOLIS./VOLTIJDDAGEN
Wage	= Pre-tax labor income / Full-time equivalent days worked	derived
Working (indicator)	= Pre-tax labor income > 0	derived

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
Entrepreneur (indicator)	Defined based on the socio-economic category on an annual basis. The annual socio-economic category of a person is determined in principle by the largest source of income, with some exceptions (e.g., students).	1992-2002: IPOREV/INPSECJ 1995-2002: RIO/SEC 2003-2004: IPI/SECCOAL1 2005-2010: IPI/PSECJ 2011-2019: INPATAB/INPSECJ
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, 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.	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.	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
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

Variable name	Description	Data sources / variables
Substantial interests	Substantial share (>5%) in equity in incorporated businesses.	1992-2005: IPOREV/VEHW1140ABEH 2006-2020: VEHTAB/VEHW1140ABEH
Other 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 principal residence loans, education loans, and other loans.	1992-2005: IPOREV/VEHW1200STOH 2006-2020: VEHTAB/VEHW1200STOH
Principal residence loans	Loans for the purpose of constructing, purchasing, or improving the owner-occupied 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
Share of risky financial assets	= Financial securities / Financial assets	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 stocks and bonds due to financial transactions and due to changing prices, following Ji, Teulings, and Wouterse (2019). For the *Primary residence* I assume that all value changes are due to capital gains unless the household changes address (or moves from being a renter to an owner or vice-versa) in which case I assume zero capital gains. 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 more 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 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 break in the series) 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
Homeowner	Binary indicator of homeownership.	1992-2002: IPOREV/INPPERSBRUT 1999-2002: OBJECTWONINGTAB/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. See description of PARTNERBUS dataset for the definition of partner.	1995 - 2020: 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	People registered in the Municipal Personal Records Database, exclude e.g., people who died before 1995.	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 A5: Variable definitions

C Hereditary disorders and access to insurance in the Netherlands

Since 2006, the whole Dutch population is insured under the mandatory basic health insurance scheme. Individuals pay a flat premium (about 1200 EUR per annum) and face an annual deductible (EUR 150 in 2008, EUR 385 since 2016). Prior to 2006, employees, self-employed individuals and retirees with a gross income below a specific limit had to join a sickness fund (about 2/3 of the population), where they paid an income-dependent insurance premia, and were offered a no-claim bonus (EUR 255 in 2006). Others could enter an individual or group private health insurance plan (about 30%). Such private plans offered a variety of benefits packages, deductibles and mostly charged flat premia but were permitted to reject applicants and exclude pre-existing conditions. Individuals insured in private plans paid on average EUR 209 in deductible in 2006. People who were not eligible for the sickness fund scheme and were not able to join a private plan due to financial or medical reasons could join a special plan (WTZ, about 5% of the population), whose regulated flat-rate premium was not substantially higher than the average premium of private plans (+200 EUR per annum in 2006) (Lever, 2005)

A further consideration with genetic disorders is access to financial and insurance products. In the Netherlands, term life insurance is often demanded by mortgage lenders for taking out a mortgage. The insurance policy must in general cover the amount of mortgage that exceeds 60-100% of the house's market value. Medical underwriting for non-health insurance products (life insurance, which is often required for mortgage, and private disability insurance) is strictly regulated under the Dutch Medical Examinations Act of 1998. Under a relatively high limit of insurance coverage, insurers may not ask for any information about exposure to hereditary conditions (family history or DNA test results), although prior disease manifestation (e.g., cancer) must be reported when taking out the insurance. For life insurance this limit was initiated at EUR 136,000 in 1998 and by continuous indexation it increased to EUR 294,000 by 2022. Some insurers set a more advantageous (higher) question limit voluntarily. In general, the limit sufficiently covers the insurance demanded for a mortgage. Mortgage lenders usually demand that life insurance covers for the difference of the mortgage value and 60% to 100% of the value of the house. For example, in 1998 the average home was sold for EUR 125,000. Assuming the purchase was entirely financed by mortgage, and that the lender demands coverage for the mortgage amount above 60% of the house's value, the required coverage is 125,000*(1-60%) = 50,000 EUR. This is well under the 136,000 EUR question limit. For disability insurance, by 2022 the limit reached EUR 42,000 for the first year of payment and EUR 28,000 for subsequent years.

D 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.

D.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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