What Can Shocks to Life Expectancy Reveal About Bequest Motives?

(Preliminary)

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Abstract

This paper infer bequest motives by using a unique data set containing individual cancer diagnoses, wealth, and family linkages of all citizens in Norway. Cancer diagnoses are useful instruments for identifying bequest motives because they provide new information about life expectancy, which affects a person's consumption plan differently depending on the relative strength of bequest and classical life-cycle-savings motives. My empirical estimates show strong evidence for bequest motives. A spouse creates a direct bequest motive; couples tend to respond to a cancer diagnosis by saving more. This result holds both across the wealth distribution and over the life-cycle. In contrast to couples, singles respond to a cancer diagnosis by spending more. However, a large part of the decrease in financial wealth among singles with children reflects transfers of wealth during a person's lifetime, so-called *inter vivos* transfers.

JEL Classifications: D1, D14, D31, E21, I12

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1 Introduction

Identifying bequest motives has posed major empirical difficulties. Until now, intentional bequest savings have been considered indistinguishable from other life-cycle savings (Dynan et al., 2002). Since death is unpredictable, even those who plan to exhaust all financial resources before death tend to leave bequests (Abel, 1985; David, 1981). As a result, observed bequests carry limited information about bequest motives.

By using a unique dataset collected of Norwegian register data on cancer diagnoses, financial wealth, and family linkages, this paper is the first to analyze how shocks to life expectancy impact consumption and saving decisions, and to infer family-related bequest motives from these decisions.[1] The random nature of cancer at the individual level ensures causal interpretation of the impact of a cancer diagnosis on financial wealth.[2]

A cancer diagnosis is a useful instrument for identifying bequest motives because it reduces the individual's expected remaining life span, which changes the person's consumption and savings plan, depending on the relative strengths of life-cycle and bequest savings motives. If individuals save solely for life-cycle purposes they will increase present consumption when expected years in retirement decrease (Yaari, 1965). In contrast, if individuals derive utility from passing on wealth to either spouse or children, and therefore also save for bequest reasons, new information about life expectancy may affect their consumption and savings differently. Consequently, consumption and saving decisions after a cancer diagnosis are informative about bequest motives.

For the average cancer patient in this dataset, which includes individual diagnoses, wealth, and family linkages, the direct effect of a cancer diagnosis on financial situation must be considered small. In general, there are two important private financial costs associated with illness: direct costs associated with treatment, and indirect costs through lower productivity and reduced labor income supply. For two reasons, both these costs must be considered relatively small for the average cancer patient in Norway. First, specialized and standardized cancer treatment is promptly available, almost free of charge, and private treatment is almost nonexistent (Molven

[1] Family proxies for bequests have been used by among others Hurd (1987, 1989); Douglas B. Bernheim and Kotlikoff (2003); Inkmann and Michaelides (2010); Ameriks et al. (2011).

[2] Conditional on individual characteristics, cancer can be considered unpredictable at the individual level (Syse et al., 2008). Making predictions from statistical models about whether a specific individual will be diagnosed with a particular disease is indeed difficult for most medical risks. Some persons in the low risk or unexposed category will develop disease, while the majority of those who are exposed will remain healthy (Rockhill et al., 2000). The lack of predictive power from well-known risk factors is often referred to as the prevention paradox first described by Geoffrey Rose in the early 1980s. (Syse et al., 2008).

and Ferkis, 2011; Fiva et al., 2014).[3] This alleviate concerns of direct negative wealth effect resulting from cancer treatment. Second, the observed high average age of cancer patients (65 years in my sample) combined with generous disability benefits, minimizes the average human capital loss associated with cancer.[4] The potential adverse effect of cancer on permanent income would anyway have a small impact on financial wealth only about 1-2 years after the diagnosis as people will tend to smooth out possible income losses over the life-cycle.

By empirically identifying family-related bequest motives and estimates of intergenerational transfers, this paper contributes to the large body of research that followed from the dispute between Kotlikoff and Summers (1981) and Modigliani (1988) about the importance of bequest motive in accounting for the aggregate capital stock. Bequests in these papers are inferred from departure from the life-cycle model. However, since death is unpredictable, even those who plan to exhaust all financial resources before death tend to leave bequests (Abel, 1985; David, 1981). As a result, intentional bequest savings have been considered indistinguishable from other life-cycle savings (Dynan et al., 2002). Early attempts to solve the problem of separating classical life-cycle saving motives from bequest motives include; Gale and Scholz (1994) and Laitner and Juster (1996) which both use micro-data from surveys, and Hurd (1987, 1989), which estimate the difference between the change in wealth for couples with and without children. Hurd (1987, 1989) central finding is that people with children decummulate wealth faster than people without children.

Several recent papers papers have used structural estimation and additional aspects of household data to separate bequest motives from other life-cycle saving motives. The overall finding in these papers are in favor of a bequest motive, at least for richer families. DeNardi et al. (2010) show that economic models with and without bequest motives fit the data almost equally well, and Inkmann and Michaelides (2010) conclude that the life insurance holdings of UK couples are consistent with bequest motives. Ameriks et al. (2011) use responses to survey questions about individuals' state-contingent plans. They find significant evidence for heterogeneity in bequest motives: these motives are minimal for at least a substantial minority of the population, and are higher on average for those with children than for those without children. DeNardi et al. (2013) match Medicaid recipiency rates and transfer amounts. To match observed assets

[3]The Norwegian State has the full responsibility for necessary specialist health services. Cancer treatment is regulated by the Ministry of Health. In 2011, the Ministry of Health decided that the time from referral to treatment received in the form of surgery, chemotherapy and / or radiation should be under 20 working days for most patients.

[4]Syse et al. (2008) study the impact of cancer on labor income in Norway. They find that cancer is associated with a 12% overall reduction in labor income compared to that of employed persons without cancer, but of similar age and education. However, these estimates are likely to have smaller impact on people's purchasing power as the Norwegian welfare state will to a large extent compensate for the loss in earnings.

holdings in this environment, the model attributes part of savings to bequest motives. Finally, Lockwood (2014) shows that models without bequests cannot simultaneously match the low rates of long-term care insurance coverage among middle-class retirees and the slow drawdown of wealth among middle-class retirees.

The identification strategy in this paper complements the existing literature by inferring family-related bequest motives through revealed preferences, using register data on financial wealth and family linkages, before and after, individuals are hit by an unanticipated shock to life expectancy. The baseline empirical strategy is to regress financial wealth after a cancer diagnosis onto a cancer dummy variable and a vector of covariates, and to compare the changed in financial wealth caused by a cancer diagnosis of individuals with and without presumed bequest motives. Estimates of intergenerational transfer are based on projecting the financial wealth of children onto a zero-one cancer dummy variable that takes the value of one if either parents are diagnosed with cancer.

The results obtained from this dataset of how a cancer diagnosis affects financial wealth have several interesting findings: (i) Direct evidence of inter vivos transfers. Children of either single mothers or single fathers that have been diagnosed with cancer have statistically significant more wealth than otherwise comparable children. The transfer amount depends in particular on the gender of the parent diagnosed with cancer; children with a mother diagnosed with cancer receive about twice the amount of children with a father diagnosed with the same cancer. Inconsistently with the common perception that bequest enters the utility functions directly as a luxury good, I find that the transfer amount is statistically unrelated to the level of wealth of the parent. Instead, at least for men diagnosed with cancer, my results indicate that the desire to leave bequests is influenced by the marginal valuation of the transfer received among the recipients of the inheritance. (ii) A spouse creates a direct bequest motive. At the end of the calender year after the diagnosis, couples with a poor cancer diagnosis (low expected survival), have about 5% more financial wealth than non-diagnosed comparables. In contrast, singles diagnosed with a poor cancer diagnosis (low expected survival) have about 30% less financial wealth than their peers at the end of the calendar year after the diagnosis. As already pointed out, a large part of the decrease in the financial wealth among singles with children reflects inter vivos transfers. (iii) The life-cycle has large impact on how households respond to a shock to life expectancy. In particular, at the beginning of the life-cycle both couples and singles respond to cancer by saving more whilst at the end of the life-cycle couples stop saving and singles reduce their financial wealth substantially. (iv) Couples in working age with little financial wealth and material leverage save the most after a cancer diagnosis. For singles, the largest decrease in financial wealth is for rich retirees with little leverage. The results for singles

are consistent with DeNardi et al. (2010, 2009), namely that wealthier households may save more because they expect to live longer. Therefore, the same mortality shock have a relatively much larger impact on their savings as the relative reduction in remaining expected years to live, is largest for the them. (v) Finally, using the variation in survival within cancer stage, I show that the average causal effect of a cancer diagnosis with low expected survival is about the same for good and poor prognosis. This result is a clear indication that the estimates reflect thoughtful decision making and not mechanical effects of the treatment.

The microfindings reported in this paper also relate to the literature that emphasizes the role of the family composition in determining the outcome of financial decisions.[5] Standard work in macroeconomics uses some form of equivalence scales to construct stand-in couples with direct preferences rather than modeling its individual members (Hong and Ríos-Rull, 2012). The empirical results presented in this paper show that the household composition has economic and statistical significant impact on whether people respond to a cancer diagnosis by consuming or saving.

The rest of the paper is organized as follows. Section 2 contains summary statistics, and a detailed description of the key aspects of a cancer diagnosis with a particular focus on the implications for life expectancy. Section 3 describes the empirical strategy, and Section 4 contains the results. Lastly, I conclude in Section 5.

2 Data Description and Definitions

2.1 The Dataset

The unique dataset is a result of merging register data from the Cancer Registry of Norway (CRN), the Norwegian Tax Administration, and Statistics Norway through unique personal numbers assigned to each individual in Norway (equivalent to social security number in the United States). All data are collected for administrative purposes and therefore the likelihood of large measurement–errors are small. In any case, any measurement error ought to be uncorrelated with the likelihood of developing cancer, and will therefore not impact the coefficient estimates. With the exception of data on cancer, the dataset is similar in content to that used by Fagereng et al. (2015), and covers the entire population of Norway over the period 2008-2012.

The Cancer Registry of Norway (CRN) has registered all cancer cases in Norway since 1953.

[5] Family structure may affect various financial decisions, including wealth accumulation (Cubeddu and Ríos-Rull, 2005), the demand for life insurance (Hong and Ríos-Rull, 2012), and asset allocation Love (2010).

Mandatory reporting from clinicians, pathologists, and death certificates ensures high quality data on the date of diagnosis, the patient's age at diagnosis, gender, tumor location (International Classification of Diseases, 10th revision (ICD-10)), and stage at diagnosis (local, regional, distant or unknown). Records held in the CRN are supplemented with relevant information on vital status from the National Population Registry. Records are regularly linked with the Cause of Death Registry run by Statistics Norway. Further information about the cancer data is available from CRN's website.[6]

The Norwegian Tax Administration and Statistics Norway have provided the financial and demographic data. The Norwegian Tax Administration is responsible for collecting income and wealth tax in Norway. Employers, banks, and public agencies are obliged by law to submit personal information (for the previous year) on income, total assets, and transfers to the Tax Administration before the end of April each year, which is the date individuals are required to submit their tax returns. The tax return includes all sources of income as well as detailed information on wealth and debt. Individuals are themselves accountable for the information in the tax return, and submission of inaccurate information, is punishable by Norwegian law. Educational level is based on the Norwegian Standard Classification of Education,[7] and includes the highest level of education every year. It is reported by the educational establishment directly to Statistics Norway. For a more detailed description of the financial and demographic data, see Fagereng et al. (2015).

2.2 Cancer and Survival Probabilities

Cancer has become the leading cause of death worldwide. More than half of current adults under the age of 65 years will be diagnosed with cancer at some point in their lifetime (Sasieni et al., 2015). About half of those receiving treatment will die from cancer or its treatment and about half will survive for more than 10 years (Jemaland et al., 2011). The probability of surviving cancer depends on the cancer type, the cancer stage, age, and individual comorbidities. Across all cancer types, the cancer stage at the time of the diagnosis is the most important determinant of survival, in addition to the type of cell the cancer originates from.[8]

The main analysis in this paper is based on cancer stages. Staging is a way of describing where the cancer is located, if or where it has spread, and whether it is affecting other parts of the body. The staging system used to describe cancer staging in this paper is the international

[6] http://www.kreftregisteret.no/Global/Cancer%20in%20Norway/2013/CIN_2013.pdf

[7] http://www.ssb.no/utdanning/_attachment/159352?_ts=143b5108f70

[8] http://www.cancerresearchuk.org/about-cancer/what-is-cancer/how-cancer-starts/types-of-cancer

Classification of Malignant Tumours (TNM). TNM has four stages, with increasing degree of severity. Stage 1 refers to a small cancer or tumor that has not grown deeply into nearby tissues, and has not spread to the lymph nodes or other parts of the body. Stage 2 and stage 3 indicate larger cancers or tumors that have grown more deeply into nearby tissue. They may have also spread to lymph nodes but not to other parts of the body. Stage 4 means that the cancer has spread to other organs or parts of the body.

Based on historical relative survival rates, the Cancer Registry of Norway (CRN) provides estimate of 5-year survival for each cancer type, stage and gender. Relative survival is defined as the observed survival in a patient group divided by the survival of a comparable group in the general population with respect to key factors affecting survival such as age, sex and calendar year of investigation (Hakulinen, 1982). Relative survival is thus a measure of excess mortality of cancer patients regardless of the reason for death.

For the analysis that follows, two facts about cancer stage are of particular importance. First, stage 1 has limited impact on life expectancy; 55% of all incidents have a 5-years relative survival close to 100%. Second, relative survival rates decrease monotonically by stage until stage 4. The nonmonotonic relation between stage 3 and 4 is due to the fact that stage 4 primarily represents the three cancer types Hodgins and non-Hodgings lymphoma and leukemia, originating in the bone marrow or lymph nodes. Most cancers are staged based on the size and spread of tumors. Because the latter three already occurs in the developing blood cells or within the bone marrow, staging for these cancer types is a little bit different.

To make sure that all analyses are carried out on samples obtained from a range of cancer treatment options, I perform the empirical analysis on the full sample (stages 1-4), only stage 1, and the sum of stage 2 and stage 3.[9] The relative good prognosis of stage 1 makes it an interesting comparison to cancer discovered in stage 2 or stage 3 (high versus low probability of survival). The reason I add stage 2 to stage 3, is to make the number of observations in this group close to the number of stage 1 cases. To eliminate any concern about sample selection, all analyses also include the results obtained from the full sample (stage 1-4). For the sake of simple notation, I will refer to stage 1 as "Good" prognosis and stage 2 and stage 3 has "Poor" prognosis. "All" stands for the full sample including all stages.Figure 1 shows the realized absolute survival rates for singles diagnosed with cancer during 2010 conditioning on being alive at the end of 2010.

[Insert Figure 1 here]

[9]In the robustness analysis, I verify that the empirical results are invariant to the choice of "mortality index", that is, using relative survival probabilities instead of stages

The figure illustrates the large difference in expected survival between good and poor prognosis. For example, close to 85% of those diagnosed with a good prognosis during 2010 were alive at the end of 2012, while only about 55% of those diagnosed with a poor prognosis were alive at the end of the same year.

2.3 Definitions and Summary Statistics

The main outcome variable in this paper is financial wealth constructed from Tax Returns following the setup in Calvet and Sodini (2014). In short, financial wealth is the sum of safe and risky financial assets. Safe assets are the sum of bank account balances and money market funds. Directly held stocks and risky mutual funds constitute risky financial assets. Financial wealth is thus a gross measure of financial wealth, as it is not net of mortgage nor includes real estate. The benefits of using financial wealth instead of net worth ($\approx$ financial wealth plus real estate minus debt) as the dependent variable is that it consists solely of liquid asset reported at market value.

Inclusion of real estate, which is both relatively illiquid and reported at tax value[10], might bias the estimate on how shocks to life expectancy impact savings for at least two reasons.[11] First, in the case of a pure life-cycle saver who wants to increase present consumption after an unanticipated shock to the planning horizon might not do so because the expected utility gain from increasing present consumption is lower than the utility loss caused by the transaction costs. Resulting from the difference in the life-cycle saver's information set and the information set of the econometrician, the empirical estimate of the change in financial wealth caused by a negative shock to life expectancy would be misleading in favor of a bequest motive. Second, if the individual who is experiencing a negative shock to life expectancy would sell the real estate to increase consumption, since tax value is likely lower than market value, it could appear in the tax returns as a positive change in wealth, which would lead to incorrect estimate of mechanism between life expectancy and savings. Also, a technical benefit of using financial wealth instead of net worth is that it permits log transformation without changing the sample, and thereby render a meaningful comparison between estimates in log and levels.

Figure 2 plots the financial wealth relative to the sum of financial wealth and real estate against age. The figure shows that financial assets make up 40-70% (55-75%) of couples (singles) total

[10]For most people, the tax value of real estate is lower than market value. However, the conversion factor between tax value and market value is likely to differ both over time and across states. In order to avoid introducing systematic biases due to the potential large heterogeneity in conversion factor, I use tax values throughout the paper.

[11]Omitting real estate might also introduce a bias, if for instance, bequest motives are associated with saving in real estate while precautionary motives favor saving in financial wealth.

assets (financial wealth plus real estate), making it an important part of total savings.

[Insert Figure 2 here]

Other explanatory financial variables include: income, defined as the sum of pension and salary before taxes, as reported in the Tax Returns, the leverage ratio, computed as debt divided by the sum of financial wealth and real estate. All financial quantities are converted into U.S. Dollars at $ 0,1731, which was the exchange rate of US Dollars to Norwegian Krone as of 31 December 2009. This fixed conversion factor is used throughout the paper. Demographic control variables include: gender, educational level, and the number of children.

One key issue in this paper is how to econometrically treat different family structures. With the purpose of exploring potential differences in bequest motives between family structures, I follow Rosen and Wu (2004) and estimate separate equations for single and married individuals. Specifically, I use the following definitions: singles are widows or widowers, or those who never married. Couples are defined as in Fagereng et al. (2015); individuals registered as married and with valid ID for spouse, or registered with valid cohabitant ID and common children. Individual assets are aggregated to the household level, while household demographics (age, education, sex, etc.) are based on the household head, defined as the oldest individual. If one of the individuals in the couple gets cancer, I refer to this as if the couple were diagnosed with cancer. The definitions of single and couple are mutually exclusive, meaning that no individual in the full sample is defined as both. Figure 3 shows the relative fraction of singles and couples as well as their ownership of financial wealth, real estate, and debt as of 31 December 2009.

[Insert Figure 3 here]

The figure shows that singles constitute 30% of the sample, and that these 30% hold about 24%/28%/35% of total real estate/debt/financial wealth. The similar aggregate portfolio between singles and couples indicates similar distribution of preference parameters. The similarity means that the preference for either family structures are likely unrelated to preference parameters for saving plans. In this case, potential bequest motives for spouse is a consequence of the relationship, whilst the relationship is not a result of the bequest motive. Therefore, comparing the savings of singles and couples experiencing the same life expectancy shock, is likely to provide a good estimate of the strength of the "operative" bequest motive for spouse.

The final sample is obtained by imposing the following restrictions. First, all individuals must be between 30-85 years in 2011. Second, financial wealth in 2009 (i.e., before anyone has been diagnosed with cancer) must be between the 1% and 99% percentile, which corresponds to

approx. $ 866 to $ 862,000. To eliminate the possibility that some of the estimated difference in savings between those diagnosed with cancer and their reference groups is due to death, I discard individuals with zero financial wealth . Last, individuals must be diagnosed with cancer for the first time, or not diagnosed at all. In 2009, the final sample contains 1,284,039 observations, covering 13,216 cancer cases and 1,270,823 non-diagnosed controls. Table 1 reports the corresponding summary statistics as well as presenting the results from testing for difference in means between cases and controls at the age of 50, 60, 70, and 80. Out of the 72 t-tests for difference in means, six are larger than two in absolute value, of which five are demographic and only one financial. The lack of significant differences between cases and controls prior to the cancer diagnosis is supportive of the postulate that cancer is random conditional on age.

[Insert Table 1 here]

Figure 6 shows the life cycle savings pattern in Norway by plotting net assets per capita for singles and couples against age. People start out at age 30 with substantial human capital and a house financed with debt. Over the life-cycle, the human capital is converted into housing equity through down payment of the mortgage, and investments in financial assets. More importantly, Figure 6 highlights the difficulty in separating intentional bequest savings from other life-cycle savings. In particular, on the one hand, the lack of dissaving in retirement (usually around 67 years in Norway) could reflect a general reluctance among retirees to rely on social insurance at old age (see e.g., DeNardi et al. (2010); Ameriks et al. (2011)). On the other hand, it would also be consistent with retirees deriving utility from leaving wealth to their heirs.

[Insert Figure 4 here]

3 Baseline Econometric Specification

Due to the cross-sectional nature of the research question, the baseline empirical strategy is to regress the outcome variable (O_{it+1}), financial wealth, onto a cancer dummy variable (D_{it}) and a vector of covariates (X_{it-1}). The interpretation of the dummy coefficient (β) is the causal effect of a cancer diagnosis ($C_{it\in(t-1,t]}$) on financial wealth (O_{it+1}).

Before we can arrive at an estimable equation, we must control for variables outside the model that may be correlated with both cancer and financial wealth. Age is an example of one such variable as both financial wealth and cancer incidence rates increase over the life-cycle. To capture life-cycle effects, all regressions (if not otherwise stated) are run conditional on a vector

of life-cycle controls (X_{it-1}). The vector of controls (X_{it-1}) includes variables that are either constant or known before the cancer diagnosis (t). Finally, I add a vector of ones (α) and a mean-zero residual (u_{it+1}), which captures latent forces of financial wealth that are outside the model and that are orthogonal to the right-hand side of the regression equation. The regression is equation is (for notational clarity, I drop family structure subscript)

$$O_{it+1} = \alpha + \beta C_{it\in(t-1,t]} + \gamma X_{it-1} + u_{it+1} \tag{1}$$

The outcome variable financial wealth (O_{it+1}) can be in either levels ($O_{it+1} = FW_{it+1}$) or in logs ($O_{it+1} = ln(FW_{it+1})$. Both the level and the logarithmic transformation are estimated with ordinary least squares (OLS). In levels, β is the ordinary least squares estimator of the average causal effect of cancer on financial wealth measured in U.S. dollars. In logs, $\exp(\beta) - 1$ is the average percentage change in financial wealth caused by the cancer diagnosis. The benefits with logarithmic transformation of the dependent variable is that it converts the skewed financial wealth variable into one that is approximately normal, and eases comparison of the estimated effect of a cancer diagnosis on the outcome variable between both family structures, and across the wealth distribution. For simplicity, when discussing the empirical estimates, I refer to the coefficient estimates (β) rather than $\exp(\beta) - 1$ as the percentage difference in means between the two groups.

Timing. All cross-sectional regressions are carried out using the financial wealth reported at the end of December the year after the diagnosis year. At this point, the average cancer patient in the sample has lived 18 months after the diagnosis. The choice of the period length is anchored on two premises, related to mental and somatic aspects of cancer treatment and follow-up. First, during an interval of 18 months most patients will have come to an acceptance of the implications of a cancer diagnosis, as the reported peak interval of grief is six months post-trauma (Maciejewski et al., 2007).[12] Second, most patients will approximately have experienced one year treatment-free interval 18 months past diagnosis, since average duration of cancer treatment is estimated to six months.[13] Taken together, the relatively long period between the diagnosis and the time the outcome (financial wealth) variable is measured alleviates potential concerns that the results reflect temporary changes in financial wealth caused by treatment rather than active saving choices.

[12]The estimate is based on the The Kübler-Ross model of grief, referred to by the The National Cancer Institute. The model described five stages of grief: denial, anger, bargaining, depression and acceptance. These stages represent the normal range of feelings people experience when dealing with a personal crisis.

[13]https://helsedirektoratet.no/kreft/nasjonale-handlingsprogrammer-for-kreft

Life-cycle controls. The conditioning variables in X_{it-1} include a broad range of variables related to the life-cycle, socioeconomic status, and the financial situation of the individual at $t-1$. I adopt the notion of Brunnermeier and Nagel (2008) and refer to this vector of covariates simply as life-cycle controls. In the base line regressions, I include age, age2, and age3; indicator for completed college education, and its interaction with age and age2, and dummy variables for gender and their interaction with age and age2, income, which is the sum of pension and labor income, financial wealth, leverage, and a dummy variable that takes the value of one if the family structure has children, and the actual number of children. The control variable lagged financial wealth is measured in the same units as the outcome variable, whilst income and leverage are included in levels. The rationale for the latter is to avoid sample selection due to, for instance, that many individuals have no debt.

Placebo regressions and alternative specification. One potential concern with the econometric specification in Equation (1) is that the least square estimator (β) may pick up preferences over savings plans (η_i) that are uncorrelated with (X_{it-1}), but correlated with the probability of developing cancer. In this case, if the true model is $FW_{it+1} = a + bC_{it} + f(X_{it-1}) + \eta_i$, the least square estimator would be biased by a magnitude of $Cov(C, \eta) \times Var(C)^{-1}$. To eliminate this possibility, I include the following two tests. First, I conduct an identical analysis but with people diagnosed with cancer in year $t+2$ (instead of in year t). Because those diagnosed with cancer in year $t+2$ will have the same characteristics as those who get the diagnosis in year t, the estimates from these regression provide a powerful test of whether the least square estimator is consistent. If a cancer diagnosis has a causal impact on the outcome variable, then the regression coefficients on the zero-one dummy variable that takes the value of one if a person is diagnosed with cancer should all be insignificant before the person know about the diagnosis. Second, I estimate a simplified version of Equation (1) after first taking the first difference of both sides of the regression. To make sure that non of the control variables are influenced by the cancer diagnosis, I only include a constant and the first difference of age2 and age3 as independent variables. The regression is estimated with OLS.

4 Results

This section contains the empirical results. I first compare how the financial wealth of couples and singles respond to a cancer diagnosis, and document that the coefficient estimates are insensitive to alternative specifications, and that a cancer diagnosis is unforseen. Thereafter, I explore whether singles with children make different savings choices than singles without children after being diagnosed with cancer. Inter vivos transfers play a central part in this

regard. The final sections investigate whether the stage in the life-cycle, and financial situation prior to the diagnosis, affect how households adjust their saving and consumptions plans after experiencing a shock to life expectancy.

4.1 Do People Hold Direct Bequest Motive Towards their Spouse?

I begin by investigating whether a spouse impacts how individuals make saving decisions after cancer diagnosis. Table 2 presents the result of the cross-sectional regressions for couples and singles (couples and singles may or may not have children). Two results stand out. First, the absolute value of the coefficient estimates for large changes in survival probability ("poor prognosis") are economically larger and more statistically significant than the coefficient estimates for relative small changes in life expectancy ("good prognosis"). This is true for both couples and singles, albeit more pronounced for singles. Larger coefficient estimates in absolute value for diagnoses with lower survival probability indicates that people respond to information about survival, and not to treatment, or the diagnosis per se. Second, the family structure has a significant effect on how people respond to mortality shocks. Couples tend to increase their savings after being diagnosed with cancer, indicating direct evidence of bequest motive toward spouse.

This finding lends support to "unitary" (same discount factor) models of household intertemporal allocation, which assume that the couple has a single utility function. In contrast to couples, singles tend to reduce their financial wealth after being diagnosed with cancer. Panel B in Table 2 shows that the results are invariant to whether the dependent variable is measured in logs or levels.

[Insert Table 2 here]

The average causal effect of a cancer diagnosis (using all diagnoses) on financial wealth is about 3% for couples while it is about -20% for singles (point estimates are based on the regression model M1). In other words, on average, couples (singles) diagnosed with cancer changed their financial wealth by 3% (-20%) over the 18 months after the diagnosis relative to their non-diagnosed reference group. Measured in US dollars, the corresponding coefficients are $ 3,617 (couples) and $ -3,055 (singles).

Moreover, couples diagnosed with a poor prognosis have about 6% more financial wealth than non-diagnosed comparables, while singles diagnosed with a poor prognosis have -40% less financial wealth than their peers. The corresponding numbers in US Dollars are $ 6,833 (couples)

and $ -8,101 (singles). Importantly, for both couples and singles, the estimated coefficients with the parsimonious first difference specification (M2) are not significantly different from the full level-model. Similarly, adding dummy-variables that takes the value of one if the composition of the household changes (preference shifters), have no material impact on the estimated coefficient (M3). The coherent estimates across the three models imply that a cancer diagnosis does not proxy for time-invariant preference parameters. That is, the regression residual from the first model (M1) is close to uncorrelated with individual fixed effects and is not a result of changes in family characteristics (M3).

Table 3 contains the placebo regressions. As mentioned, the estimated coefficients of the cancer dummy variables provides a test of causality between a cancer diagnosis and financial decisions. It shows the estimates from identical regressions but using the the cancer population in 2012, instead of 2010. Because the cancer diagnoses are not yet in the information set of the individuals, causality between cancer diagnosis and financial decision making implies that the estimate of the cancer coefficient (β) should be indistinguishable from zero. The results provide strong support of causal interpretation. All cancer coefficient estimates are either small in absolute value or as the opposite sign as those presented in Table 2. Out of the 36 regressions, only two have a t-statistics above 2, which is close to what we would expect given the high number of regressions. The two coefficient estimates with a t-statistics above two are for singles using all diagnoses and log-transformation with only individual fixed effects as controls.

In sum, the empirical evidence presented in Table 2 indicates that the degree of revision of current savings and consumption plan after receiving new information about life expectancy depends on the family structure prior to the diagnosis. The coherent estimates across different regression models (M1-M3) combine with the insignificance of the placebo regressions imply that a cancer diagnosis does not proxy for time-invariant preference parameters, or can be foreseen. In order to reduce the number of estimated coefficients, results obtained from M2 (individual fixed effects), M3 (preference shifters), and placebo regressions are dropped in the rest of the paper.

4.2 Are Children a Motive for Leaving Bequests Among Single Parents?

Are children a motive for leaving bequests among single parents? To answer this question, I investigate the saving behavior of singles with and without children who are faced with the same mortality shock. This empirical strategy for studying bequest motive goes back to Hurd (1987). He shows that, given initial wealth, and the same resources, the wealth of someone with a bequest motive will decline more slowly than the wealth of someone without a bequest

motive, as long as the distributions of the future random variables are the same for the two types. Conditional on pre-cancer characteristics and the cancer diagnosis, there are no a priori reasons to expect the distributions of future random variables to differ between the two types. Therefore, for a given mortality risk conditional on resources, comparing the saving behaviour of singles with and without children, ought to provide empirical evidence as to whether children give a bequest motive. However, a fly remains in the ointment. The test implicitly assumes that the decision to have children is random, which of course is unlikely. For example, the preference parameter that affects the aspiration to have children may also impact the care for others. If so, the wealth of this hypothetical group of singles would decline more slowly than those who choose not to have children regardless of whether they actually happen to have children. Having that in mind, at the minimum, the results provide a description of how children impact the financial decisions of singles following a downward revision in life expectancy.

The main result reported in Table 4 is in consonance with the surprising result in Hurd (1987, 1989); singles with children do in fact decummulate their wealth faster than singles without children. To focus on the main findings, I center the attention on poor prognoses. For this group, the average causal effect of a cancer diagnosis on financial wealth 18 months after the diagnosis is about -32% for singles without children, and -44% for singles with children. The substantial estimate of -44% corresponds to the sum of the cancer dummy coefficient (-32%) and the interaction term between the cancer dummy variable and a variable that takes the value of one if a single person has children prior to the diagnosis (-12%). The difference is statistically significant with a t-statistics of 2.3. The corresponding results obtained with the dependent variable measured in US Dollars, tell the same story albeit with larger standard errors. The large standard errors of the coefficient estimates is in part due to multicollinearity between the cancer dummy variable and the interaction term.

However, in isolation, the fact that singles with children decummulate their wealth faster than singles without children is inconclusive about the existence of bequest motives. The reason is that the large negative change in financial wealth among singles with children may reflect intergenerational transfers of wealth, so-called inter vivos transfers. The next section explore the role of inter vivos transfers in explaining the reduction in financial wealth among singles with children after receiving a cancer diagnosis.

[Insert Table 4 here]

4.3 Single Parents with Cancer Transfer Wealth to their Children

I begin with a key question left hanging from the previous section: how do singles with children spend their money after a cancer diagnosis? And are there differences between genders? To empirically determine the role of inter-vivo transfers, I use the intergenerational link available in the dataset to estimate the impact a single parent's cancer diagnosis has on a child's financial wealth. Specially, I project the financial wealth of children onto a zero-one cancer dummy variable for whether parents are diagnosed with cancer, an interaction between cancer and a "particular variable" (described below), and a vector of controls,

$$FW_{Chit+1} = \alpha + \beta_C C_{Pait\in(t-1,t]} + \beta_I (C_{Pait}\mathbf{I}_{t-1}) + \rho X_{t-1} + u_{it+1} \tag{2}$$

where $\mathbf{I}_{t-1}$ refers to a 1 x 2 vector with either the financial wealth and squared financial wealth of parent or child, or a family characteristic as the first element and zero as the second element. In this setup, the estimated coefficient on the cancer dummy variable (β_C) that takes the value of one if one of the parents are diagnosed with cancer, provides a direct test of accidental bequest against the intentional alternative. The idea with the interaction terms (β_I) is to investigate whether the decision to transfer wealth depends on financials or demographic characteristics prior to the diagnosis. For example, the inclusion of the quadratic function of the financial wealth prior to the diagnosis might be informative about to what extent inter vivos transfers depend on the marginal value of the wealth of either parent or child. To examine potential gender effects in intergenerational transfers, I report the results for children of both single fathers and single mothers diagnosed with cancer.

Table 5 shows direct evidence of inter vivos transfers. Children with either single mothers or fathers that have been diagnosed with cancer have statistically significant more wealth than otherwise comparable children. Children with a father diagnosed with a poor cancer diagnosis has about $ 2,960 more financial wealth than their peers, while children with a mother having the same diagnosed has about $ 7,630 more. To put the number in the context of a typical household balance sheet, the transfer from father to child correspond to about 7% (30%) of the median financial wealth of single individual with 65 (30) years of age. The equivalent ratios for transfers from mothers receiving a poor diagnosis are 19% (76%) respectively. Importantly, when interpreting the transfers in terms of single parent's average financial wealth, to account for the fact that the average individual has 2,16 children, the transfer amounts should probably be multiplied by about 2. Therefore, the direct evidence of inter-vivo transfer documented in Table 5 are highly economically significant. The coherent estimates between the model with (M2) and without controls (M1) imply that the transfer is close to uncorrelated with typical

life-cycle variables.

[Insert Table 5 here]

The results from the other empirical specifications in Table 5 (M3-M6) reveal a complex relation between the transfer amount and financial and demographic characteristics prior to the diagnosis. The table has three main findings. First, above a minimum transfer, the transfer amount from either parent with a cancer diagnosis is statistically unrelated to the their level of wealth. This is surprising, and is in disagreement with the common perception that bequest enters the utility functions directly as a luxury good.[14] Second, for fathers, the interaction term between financial wealth and squared financial wealth of the children and the cancer dummy variable (M4), indicate that the transfer amount is decreasing in the level of financial wealth of the children. In particular, the expected transfer from father to child goes to zero as the children move towards the upper end of the financial wealth distribution. For mothers, the estimated coefficients are economically insignificant (the product of coefficient on the squared financial wealth of the child and the level of financial wealth at the 99 percentile is less than $ 1). Lastly, the interaction term between whether the child is defined as single or belongs to a household (M5) and the interaction term between whether the child has children (M6), are both insignificant for fathers. In stark contrast, for the mothers, both interaction terms are large in absolute value and statistically significant; mothers tend to transfer more financial wealth when the child is part of a couple than when it is single (M5), and if the their children have offsprings (M6).

In conclusion, the direct evidence of inter vivos transfers documented in this section comes from a preference for bequest. Collectively, the results are inconsistent with the recent notion of bequest motive as a luxury good. Instead, the results indicate that the desire to leave bequest, at least for men, is influenced by the marginal valuation of the transfer received among the recipient of the inheritance.

4.4 Mortality Shocks Over the Life Cycle

As shown in the beginning, net household assets follow a clear life-cycle pattern.[15] Figure 6 shows a disaggregated version; it shows the life-cycle pattern in financial wealth and leverage

[14] For example DeNardi (2004) concludes; "The presence of a bequest motive also generates lifetime saving profiles more consistent with the data: saving for precautionary purposes and saving for retirement are the primary factors for wealth accumulation at the lower tail of the distribution, while saving to leave bequests significantly affects the shape of the upper tail."

[15] Net household assets are defined as financial wealth plus the book value of real estate less gross debt.

for couples and singles using data for 2009.

[Insert Figure 6 here]

The figure shows that the median couple (single) starts out at age 30 with around $ 20,000 ($ 10,000) in financial assets and leverage[16] of about 4,0 (3,5), and enters retirement at around 67 years with $ 65,000 ($ 40,000) in financial wealth and leverage of about 0,2 (0). These plots point to potential important life-cycle effects in how people respond to mortality shock. The focus of this section is thus to examine how couples and singles respond to mortality shocks over the life-cycle. Table 6 presents the results for singles and couples at four stages in the life-cycle: 30-44, 45-59, 60-74, 75-85. Coefficient estimates in both logs and levels are included to facilitate comparison across the life-cycle.

[Insert Table 6 here]

Table 6 shows that a cancer diagnosis has different impact on the saving behavior over the life-cycle for couples and singles. However, the trend in the coefficient estimates over the life-cycle is similar. For couples, the propensity to increase savings after a cancer diagnosis decreases monotonically over the life-cycle. At the first stage of the life-cycle (30-44 years), couples increase savings by about 35% ($ 15,367) while couples at the last stage of the life-cycle (75-85 years) do nothing. Bequest motive for spouse combined with a hump shaped labor income profile over the life-cycle could generate generate this result. The intuition is that young people facing high labor income growth and long life expectancy view bequestable wealth as a long-term liability to be paid for with human capital. When life expectancy declines, the present value of the human capital also declines whilst the utility of bequest increases, since it is less discounted. As a result, it is optimal to save more.

Somewhat surprising, the same pattern emerges for singles. At the first stage of the life-cycle (30-44 years), singles increase savings by about 26% ($ 18,738)[17] while singles at the last stage of the life-cycle (75-85 years) decrease their financial wealth by about 62% ($ 17,020). The increase in savings among singles at the first stage of the life-cycle is puzzling because at this stage most singles do not have children. Therefore, the utility gain from spending out of retirement wealth should be large in this group.

[16] Leverage is defined as debt divided by the sum of financial assets and real estate.

[17] The point estimate of 26% is not statistically different from 0 with a t-value of 1,5 whilst the coefficient estimate of $ 18,738 has a t-value of 3,3.

In sum, the similar trend among the coefficient estimates of couples and singles, in particular at the early stage of the life-cycle in which most singles do not have children is difficult to reconcile with standard life-cycle models.

4.5 Mortality Shocks Across the Wealth and Leverage Distribution

In the United States between 2001 and 2014, higher income was associated with greater longevity, and differences in life expectancy across income groups increased over time (Chetty et al., 2016). In particular, the difference in life expectancy between the richest 1% and poorest 1% of individuals was 14,6 years for men and 10,1 years for women (Chetty et al., 2016). These differences in life expectancy might be important for understanding saving motives across the wealth distribution (DeNardi et al., 2009). For example, wealthier households may save more because they expect to live longer –and are therefore more likely to need expensive private services at very old age. Following this train of thought, the same mortality shock will have a much larger impact on the saving decisions of people in the top of the wealth distribution than in the lower end. This is intuitive –as the planning horizon after a given cancer diagnosis is now the same for the rich and the poor, the relative reduction in remaining expected years to live, is largest for the those who expected to live the longest.

Table 7 presents the result of the cross-sectional regressions of financial wealth on cancer diagnoses for four different wealth and leverage percentiles, as well as their interaction with retirement. Coefficient estimates in both logs and levels are included to facilitate comparison across deciles.

[Insert Table 7 here]

Panel A in Table 7 shows that financial wealth prior to the cancer diagnosis has large impact on the saving behavior after the diagnosis for both couples and singles. For couples, the tendency to increase savings after a cancer diagnosis is centered in below-median wealth families. For singles, the pattern of the estimated dummy coefficients is striking. The tendency to decrease financial wealth increases monotonically in financial wealth. Singles belonging to the 25-50 wealth percentile decrease savings by about -38% ($\approx$ \$ 0), while singles belonging to the 90-99 wealth percentile decrease savings by about 52% (\$ 34,661).

Panel B in Table 7 reports the corresponding results for leverage percentiles.[18] Leverage prior to the diagnosis also impact how people make saving decisions after a cancer diagnosis, but

[18]Leverage is defined as: debt / (financial wealth + real estate).

there are no clear pattern in the coefficient estimates. For couples, the tendency to save after a cancer diagnosis is increasing in leverage until the highest leverage percentile (75-99 percentile). For singles, people without leverage decrease their financial wealth most.

Because young individuals are less wealthy and have more leverage than older individuals, the results are indistinguishable from the life-cycle effects previously documented. In an attempt to disentangle the former from the latter, Panel C reports the coefficient estimates from four subgroups sorted on the interaction between financial wealth and leverage for working age adults (Age < 70) and retirees (Age ≥ 70).[19]

Starting with the results for couples. First, the bequest motive for spouse is mostly evident among couples below 70 years. Among this age group, the largest coefficient estimates are for couples with high leverage and little financial wealth (G1), with an estimated change in financial wealth of about $ 13,023 (17%) 18 months after a poor cancer diagnosis. Also, these coefficient estimates are the most precise ones with a t-statistics of 5,7 (logs) and 6,7 (levels). Second, rich couples above 70 years with leverage (G2) tend to decrease their financial wealth after a cancer diagnosis. Since otherwise comparable couples without leverage (G4) do nothing, the reduction in financial wealth among rich couples above 70 years with leverage (G2) might reflect capital structure adjustments. For couples that belong to the lower 25 leverage percentile (G3 and G4), there is little difference in saving behaviour between the working age adults and retirees.

Regarding singles, retirees are more likely to reduce their financial wealth than financially comparable working age adults. The largest decrease in financial wealth among singles is for singles above 70 years that belong to the upper 75 financial wealth percentile and the lower 25 leverage percentile. This result indicates that the reduction in financial wealth among singles reflect consumption and not capital structure adjustments. In sum, the results for singles are consistent with that reported in DeNardi et al. (2010, 2009); wealthier households may save more because they expect to live longer –therefore the same mortality shock have a relatively much larger impact on their savings as the relative reduction in remaining expected years to live, is largest for the them.

4.6 Stage Controls

The external validity of reported results rests on the premise that the coefficient estimates reflect active choices (thereby revealed preferences) and not treatment bias.[20] The purpose

[19] The high age is necessary to ensure a reasonable number of cancer cases in each group.

[20] A brief description of the relation (and the lack thereof) between survival and cancer treatment, including general principles for treatment exemplified by two cancers of different prognosis is available in the Appendix.

with this section is to empirically examine the possibility that treatment has material impact on the estimation results. To ascertain the potential treatment mechanisms, I redo the main analysis using the within cancer stage variation in survival. This control strategy hinges on the fact that treatment protocols is closer related to the cancer stage than to the 5-year survival rate.

In general, cancer treatment has been moving away from the use of one-size-fits-all therapy and toward the use of targeted treatment designed for specific patients and tumors –that is, personalized medicine. Accordingly, a potential treatment bias is likely to be independent of cancer type, stage, affected organ, and thus allows for comparison across strata and cancer staging. Above all, prognosis is based on 5-year survival rates, a type of survival rate for estimating the prognosis of a particular disease, normally calculated from the point of diagnosis. Yet, to empirically examine the possibility that treatment has material impact on the estimation results, I redo the main analysis using the within cancer stage variation in survival using the following model,

$$FW_{it+1} = \alpha + \sum_{k=1}^{3} \beta_k RS_{kit\in(t-1,t]} + \gamma X_{it-1} + u_{it+1} \tag{3}$$

The only difference between the specification in Eq. (3) and the baseline model is the swap between stages ("Good" and "Poor" prognosis) and relative survival (RS). Here, RS is the 5-year relative survival, β_1 is a zero-one dummy variable that takes the value of one if $RS \in (0, 0.3]$, β_2 is a zero-one dummy variable that takes the value of one if $RS \in (0.3, 0.7]$, and β_3 is a zero-one dummy variable that takes the value of one if $RS \in (0.7, 1]$. The cutoffs are equally spaced and ensure dispersion among cancer stages and types. The results from the estimation is presented in Table 8.

[Insert Table 8]

For both couples and singles, the estimated cancer dummy coefficients $(\beta_1, \beta_2, \beta_3)$ exhibit similar pattern using all stages, only good prognoses (stage 1), and only poor prognoses (stage 2 and stage 3). In all three samples, the largest effects, both economically and statistically, are for those receiving the largest shock to life expectancy ($RS \in (0, 0.3]$); a clear indication that individuals respond to the information about expected survival rather than treatment. The last Panel in Table 8 shows the result from testing the null hypothesis that all the cancer dummy coefficients $(\beta_1, \beta_2, \beta_3)$ are identical. For both couples and singles, the null hypothesis that the cancer dummies coefficients $(\beta_1, \beta_2, \beta_3)$ are identical is rejected for each sample specifications. In summary, the results are in favor of that the estimates reflect thoughtful decision making

and not mechanical effects of the treatment.

5 Conclusion

By using a unique dataset collected of Norwegian register data on cancer diagnoses, financial wealth, and family linkages, this paper has estimated the causal effect of a cancer diagnosis on financial wealth, and inferred family-related bequest motives from these estimates.

The results obtained from the dataset provided direct evidence of inter vivos transfers. Children of either single mothers or single fathers that have been diagnosed with cancer had statistically significant more wealth than otherwise comparable children. The transfer amount depended in particular on the gender of the parent diagnosed with cancer; children with a mother diagnosed with cancer received about twice the amount of children with a father diagnosed with the same cancer. Inconsistently with the common perception that bequest enters the utility functions directly as a luxury good, the estimated transfer amount was statistically unrelated to the level of wealth of the parent. Instead, at least for men diagnosed with cancer, the results indicated that the desire to leave bequests was influenced by the marginal valuation of the transfer received among the recipients of the inheritance.

We have learned that spouse provides a direct bequest motive; at the end of the calendar year after the a poor cancer diagnosis (low expected survival), couples have about 5% more financial wealth than their non-diagnosed comparables. Apart from rich retired couples, the tendency to respond to a cancer diagnosis by saving more hold across both the wealth distribution and over the life-cycle. In contrast to couples, singles with a poor cancer diagnosis (low expected survival), have about 30% less financial wealth than their reference group at the end of the calendar year after the diagnosis.

I showed that the stage of the life-cycle had large impact on how households responded to a shock to life expectancy. In particular, at the beginning of the life-cycle both couples and singles responded to a cancer diagnosis by saving more. At the end of the life-cycle couples stopped saving, and singles reduced their financial wealth substantially. These saving tendencies were particularly pronounced for couples in their working age with little financial wealth and material leverage, and for rich single retirees with little leverage.

Using the variation in survival within cancer stage, I showed that the average causal effect of a cancer diagnosis with low expected survival is about the same for good and poor prognosis, a clear indication of that the estimates reflect thoughtful decision making –and not mechanical

effects of treatment. In ongoing work, I extend the current study by using structural estimation to identify preference parameters in a full life-cycle model of single retirees.

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A Appendix

A.1 Brief Descriprtion of Cancer Treatment

Growing knowledge about the genetic makeup of tumors is leading to a revolution in cancer treatment. For many years, doctors knew that certain groups of individuals benefited from certain types of treatments. For example, older women with breast cancer tended to benefit more from hormone treatments than younger women with breast cancer. In recent years, scientists discovered that not all cancers are alike. There are variations of each type of tumor. This was discovered when researchers focused on the genetics of tumors. Our genes are the blueprint for control of every cell in the body. In summary, considerable improvements in the surgical and medical spheres have introduced more gentle treatments that reduce the impact of patients' quality of life. A combination of improved and more personalized therapies for various cancers, prevention, and earlier diagnosis have increased the numbers of cancer patients who recover completely.

The main target in the cancer treatment, independent of cancer type, affected organ or characteristics of the cell type the cancer originates from, is the removal of the tumor. The general principles are complete surgical removal of the tumor, followed by radiotherapy, chemotherapy and other adjuvant treatment when indicated. If a complete surgical removal cannot be performed, the introduction of personalized medicine has made possible effective tailored cancer treatments.

Breast cancer is the most common cancer among women, accounting for 22% of all cancers incidents. The cumulative risk of developing breast cancer at 75 years is about 8%. The prognosis of breast cancer is highly dependent on the stage. For example in Norway, the 5-year relative survival for breast cancer discovered in stage 1 is about 99%, whilst it is about 26% for breast cancer discovered in stage 4. Primary treatment of breast cancer is surgery (breast-conserving surgery or removal of the chest lymph glands in the armpit) combined with chemotherapy before surgery. Nine out of ten diagnosed with breast cancer will also get radiotherapy or hormone therapy. The radiotherapy starts about 4-5 weeks after the chemotherapy.

Lung cancer is the most common cancer worldwide, and the second most common cancer in Norway. In Norway, it accounts for approximately 10% of all new cancer cases, and is the cancer type that causes most deaths. The stage of the lung cancer at the time of the diagnosis impacts the probability of surviving. For example in Norway, the 5-year relative survival for lung cancer discovered in stage 1 is about 41% in men and 51% in women, whilst it is about 16%/1% and 21%/2% for lung cancer discovered in stage 2/3. Primary treatment of lung

cancer is surgery. For stage 2 and stage 3, surgery is often supplemented with radiotherapy and possibly chemotherapy in four cycles at three weeks intervals. Chemotherapy starts about eight weeks after surgery. If the tumor cannot be removed with surgery, radiotherapy is given in stage 1.

B Figures

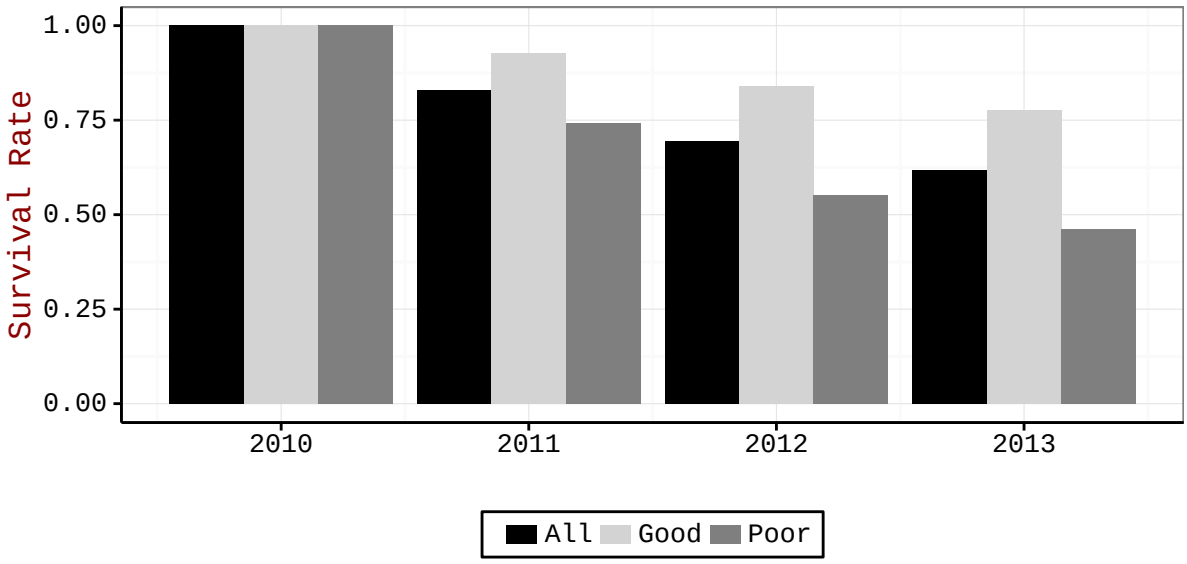


Figure 1: Absolute Survival Rate

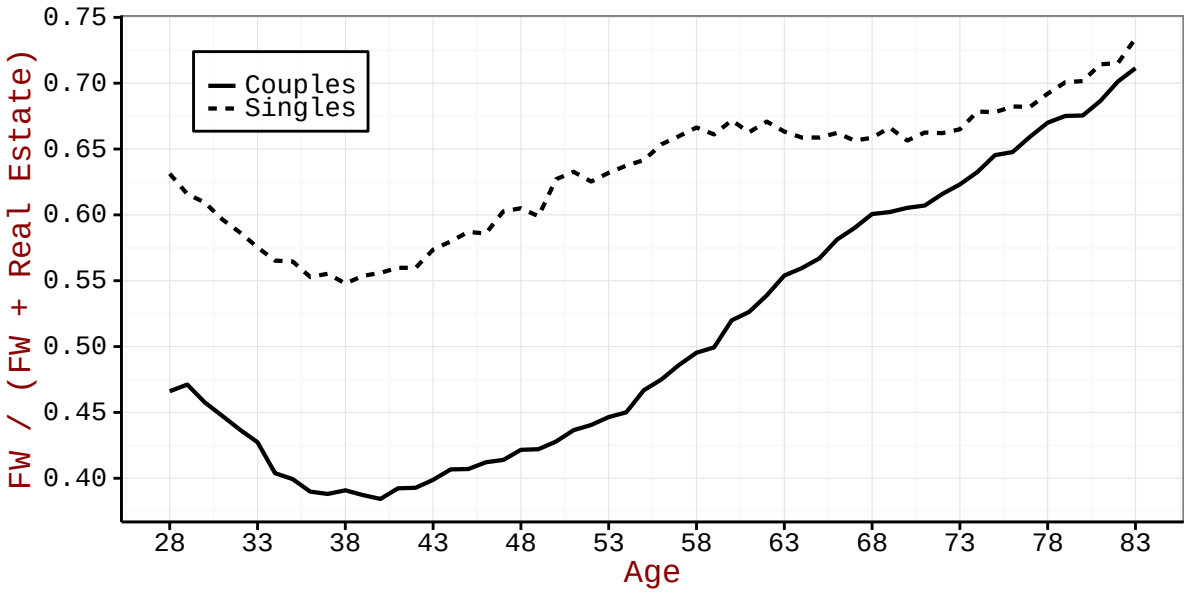


Figure 2: Financial Wealth to Total Assets in 2009

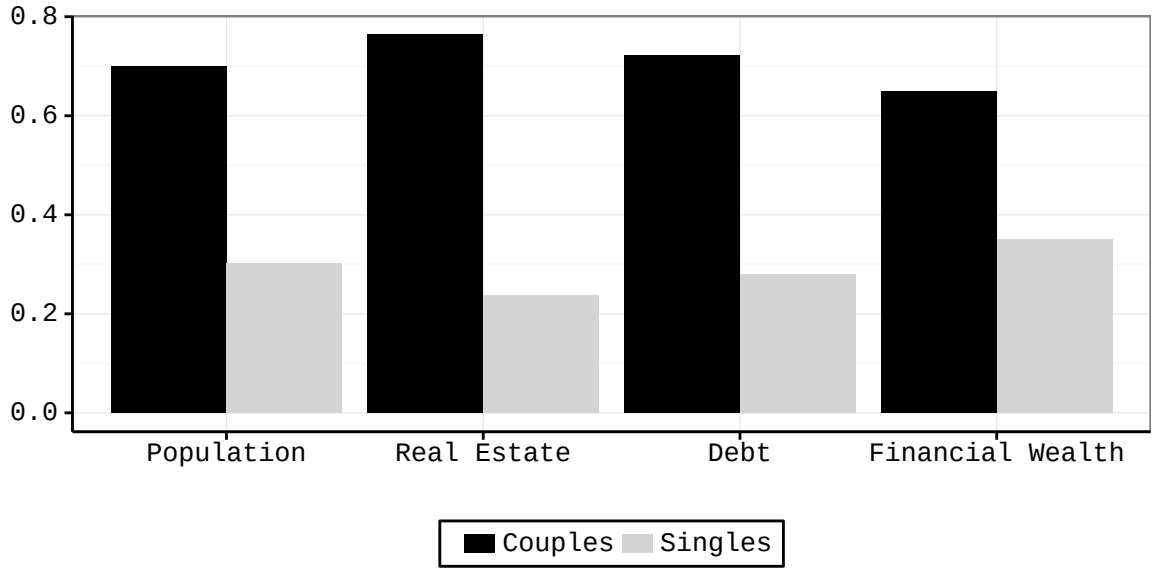


Figure 3: Relative Distributions of Family Structures and Assets

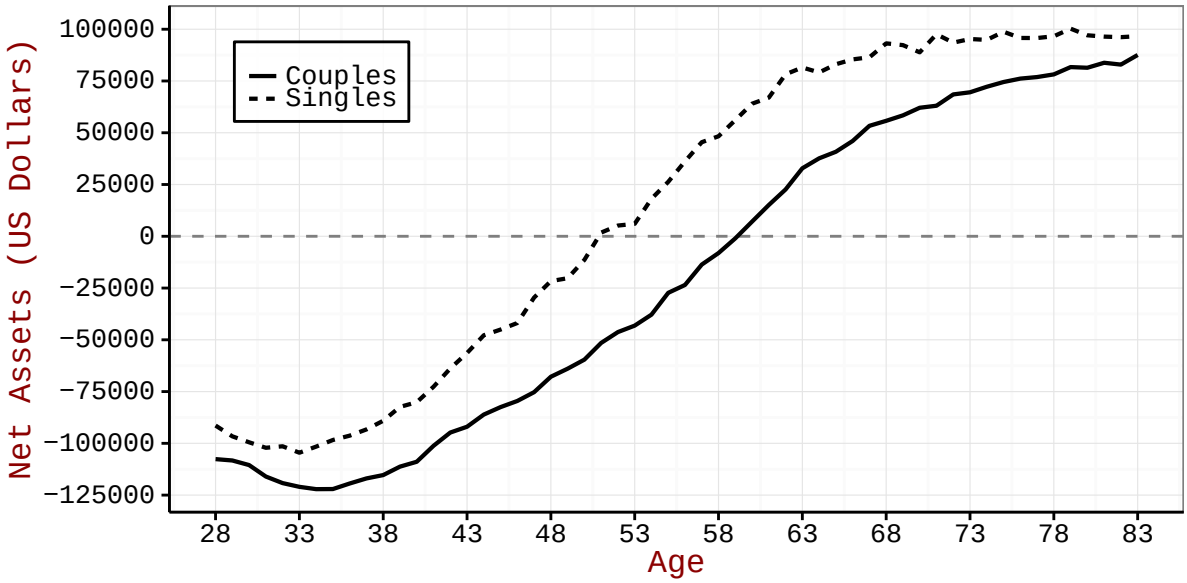
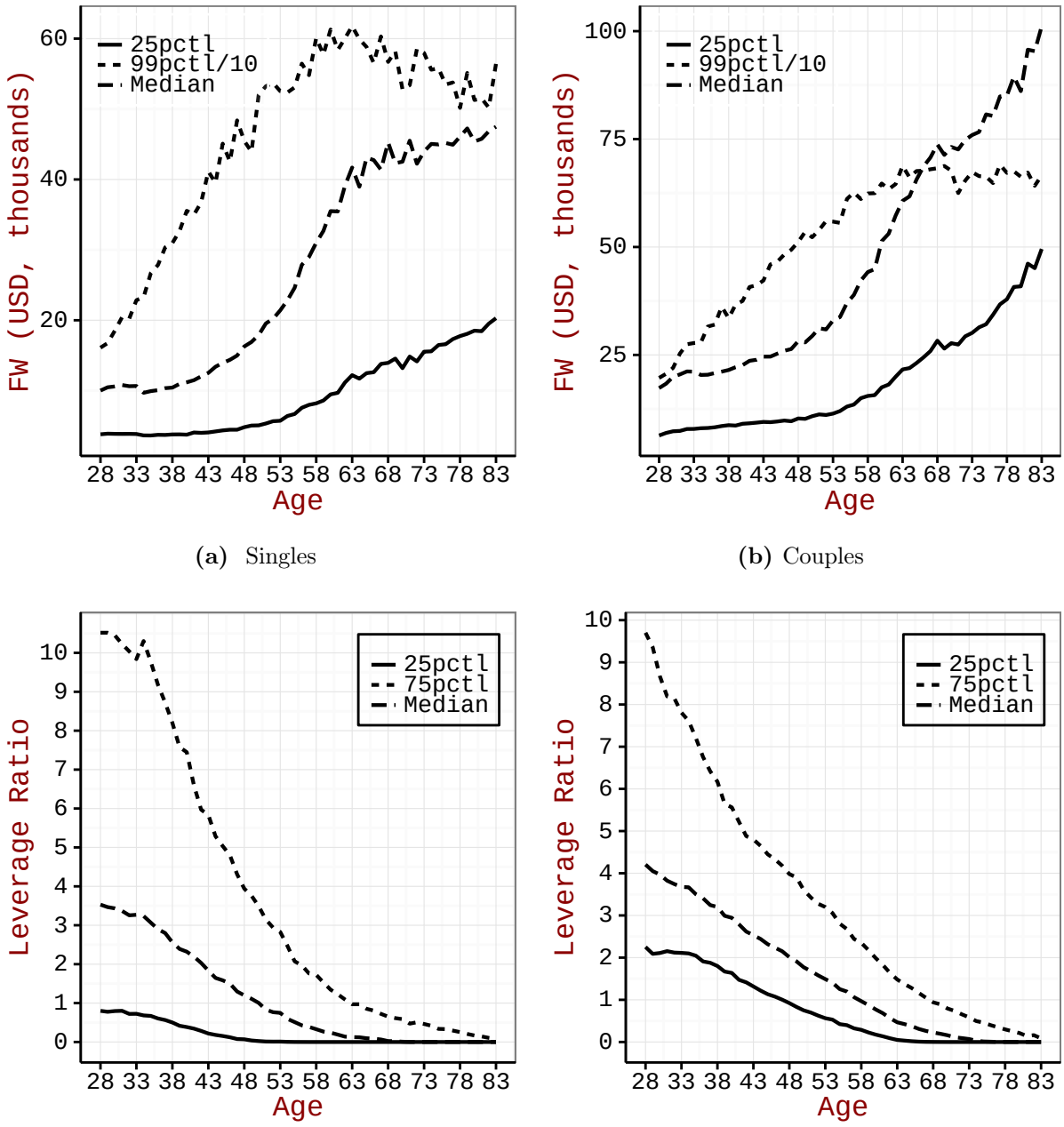


Figure 4: Net Assets in US Dollars (per individual)



(a) Singles

(b) Couples

(c) Singles

(d) Couples

Figure 6: Financial Wealth and Leverage over the Life-Cycle

C Tables

Table 1: Summary Statistics in 2009

Panel A: Summary Statistics

	Control Population (N=1270.8k)					**Cancer Population (N=13.216k)**				
	Median	Mean	StdDev	Min	Max	Median	Mean	StdDev	Min	Max
Share Married	1.00	0.54	0.50	0.0	1.0	1.00	0.73	0.45	0.0	1.0
Share Men	1.00	0.67	0.47	0.0	1.0	1.00	0.72	0.45	0.0	1.0
Number Children	2.00	1.67	1.38	0.0	17.0	2.00	2.16	1.35	0.0	12.0
Age	48	50.28	15.01	28.0	83.0	66	65	12.02	28.0	83.0
Share Low Education	0.0	0.24	0.43	0.0	1.0	0.0	0.29	0.46	0.0	1.0
Share High School	0.0	0.44	0.50	0.0	1.0	0.0	0.48	0.50	0.0	1.0
Share College	0.0	0.32	0.47	0.0	1.0	0.0	0.23	0.42	0.0	1.0
Financial Wealth	26	64	101	1	865	51	96	125	1	862
Real Estate	95	170	254	0.0	30 874	37	112	196	0.0	3 406
Debt	43	55	64	0.0	4 037	62	68	68	0.0	1 666
Leverage	1.36	7.32	27.64	0.0	4 524	0.34	3.44	19.62	0.0	1 018
Income	81	96	67	0.0	2 437	81	94	60	0.0	1 561

Panel B: T-test for difference in means between Cases and Controls

	Households				Singles			
Age	50	60	70	80	50	60	70	80
Share Women	-0.64	-1.11	-0.32	-1.14	3.19	-1.64	-0.32	-4.13
Number Children	1.17	-0.48	-1.15	2.10	-0.66	-0.85	-1.15	-0.12
Share Low Education	0.14	-0.93	0.13	-1.30	-0.75	-1.53	0.13	-0.36
Share High School	-1.65	1.47	-1.75	1.35	1.71	1.11	-1.75	0.58
Share College	1.75	-0.77	2.08	-0.06	-0.80	0.47	2.08	-0.35
Financial Wealth	-0.14	0.30	0.44	-0.35	0.75	0.70	0.44	0.46
Real Estate	0.52	-0.61	1.74	-2.18	-0.06	0.02	1.74	-1.04
Debt	0.34	0.52	0.66	0.15	1.23	-0.39	0.66	0.28
Income	1.06	0.17	0.88	-1.40	-0.15	-1.08	0.88	-1.75

Panel A reports summary statistics of demographic and financial variables for the control population (not diagnosed with cancer during 2010) and the cancer population (diagnosed with cancer during 2010) as of 31.12.2009 (i.e., before anyone has been diagnosed with cancer). Variables starting with "Share" show the fraction of the sample that meets a particular criteria. For example, Share Married shows the fraction of the sample that is married. Financial variables are measured in thousands (k) USD using the exchange rate as of 31.12.2009. Panel B reports the t-statistics of the difference between the mean of the cancer and the control population for a given age. The t-statistics of the difference between the mean is computed using $(\mu_{cans} - \mu_{cont}) \times \sqrt{(se(\mu_{case})^2/n_{case}) + se(\mu_{cont})^2/n_{cont})}$.

Table 2: Do People Hold Direct Bequest Motive Toward their Spouse?

$$O_{it+1} = \alpha + \beta C_{it\in(t-1,t]} + \rho X_{it-k} + u_{it+1}$$

Panel A: Outcome Variable: Log Financial Wealth: $O_{it+1} = \ln(FW_{it+1})$									
	All Cancer Types			**Good Prognosis**			**Poor Prognosis**		
Model	M1	M2	M3	M1	M2	M3	M1	M2	M3
Couples (β)	0.03	0.03	0.03	0.02	0.02	0.02	0.05	0.06	0.05
t(β)	(3.2)	(3.4)	(3.3)	(1.8)	(1.7)	(1.8)	(3.0)	(3.4)	(3.1)
Singles (β)	-0.20	-0.19	-0.21	-0.04	-0.02	-0.04	-0.43	-0.41	-0.43
t(β)	-(8.6)	-(7.9)	-(8.7)	-(1.1)	-(0.7)	-(1.2)	-(11.2)	-(10.6)	-(11.2)
Panel B: Outcome Variable: Financial Wealth in US Dollars: $O_{it+1} = FW_{it+1}$									
Couples (β)	3 617	3 697	3 570	2 574	2 757	2 560	6 833	6 901	6 757
t(β)	(3.5)	(3.6)	(3.4)	(1.8)	(1.9)	(1.8)	(4.1)	(4.1)	(4.0)
Singles (β)	-3 055	-2 268	-3 121	-270	511	-324	-8 101	-7 311	-8 100
t(β)	-(2.8)	-(2.1)	-(2.9)	-(0.2)	(0.3)	-(0.2)	-(4.6)	-(4.1)	-(4.6)
Regression Summary									
Life-cycle controls	Y	N	Y	Y	N	Y	Y	N	Y
Individual Fixed Effect	N	Y	N	N	Y	N	N	Y	N
Preference Shifters	N	N	Y	N	N	Y	N	N	Y
R2: Couples (A)	0.65	0.00	0.65	0.65	0.00	0.65	0.65	0.00	0.65
R2: Singles (A)	0.56	0.00	0.57	0.56	0.00	0.57	0.56	0.00	0.57
R2: Couples (B)	0.55	0.00	0.55	0.55	0.00	0.55	0.55	0.00	0.55
R2: Singles (B)	0.67	0.00	0.67	0.67	0.00	0.67	0.67	0.00	0.67
NOBS(k): Couples	665.0	665.0	665.0	660.6	660.6	660.6	659.2	659.3	659.2
NOBS(k): Singles	568.7	568.7	568.7	567.4	567.4	567.4	567.0	567.0	567.0

The table presents the estimate on the causal impact of a cancer diagnosis during 2010 on financial wealth as of December 2011. Panel A has the natural logarithm of financial wealth as the dependent variable while Panel B has financial wealth measured in US Dollars as the dependent variable. In addition to the coefficient estimates, both panels include t-statistics in parenthesis, information about which independent variables that are included in the regressions, R2, and the sample size (under Regressions Summary). The table contains three main columns with different prognosis for the cancer cases. The first main column uses all cancer cases (stage 1-4), the second column uses only good prognoses (stage 1), and the last column uses only poor prognosis (stage 2 and stage 3). Each of the three main column is dividend into three minor columns, each of which represents a particular regression model. The differences between the regressions models are the control variables included in the regression. Life-cycle controls include age, age2, and age3; indicator for completed college education, and its interaction with age and age2, and dummy variables for gender and their interaction with age and age2, income, which is the sum of pension and labor income, financial wealth, leverage, and a dummy variable that takes the value of one if the family structure has children, and the actual number of children. The control variable lagged financial wealth is measured in the same units as the outcome variable. Preference shifters include three zero-one dummy variables that take the value of one if there is a change in the household composition (couples versus single and the number of children) during the period of the analysis.

Table 3: Do People Hold Direct Bequest Motive Toward their Spouse?

$$O_{it+1} = \alpha + \beta C_{it\in(t-1,t]} + \rho X_{it-k} + u_{it+1}$$

Panel A: Outcome Variable: Log Financial Wealth: $O_{it+1} = \ln(FW_{it+1})$									
	All Cancer Types			**Good Prognosis**			**Poor Prognosis**		
Model	M1	M2	M3	M1	M2	M3	M1	M2	M3
Couples	0.00	0.01	-0.00	0.02	0.02	0.02	-0.02	-0.00	-0.02
t(b)	(0.0)	(0.8)	(0.0)	(1.7)	(1.6)	(1.7)	-(1.4)	-(0.1)	-(1.4)
Singles	0.04	0.06	0.04	0.06	0.07	0.05	0.03	0.05	0.02
t(b)	(2.0)	(2.8)	(1.9)	(1.8)	(2.2)	(1.6)	(0.9)	(1.5)	(0.8)
Panel B: Outcome Variable: Financial Wealth in US Dollars: $O_{it+1} = FW_{it+1}$									
Couples	-656	-348	-659	771	1 236	768	-1 771	-1 401	-1 773
t(b)	-(0.7)	-(0.4)	-(0.7)	(0.6)	(0.9)	(0.6)	-(1.2)	-(0.9)	-(1.2)
Singles	230	910	131	130	760	32	526	1 355	432
t(b)	(0.2)	(0.9)	(0.1)	(0.1)	(0.5)	(0.0)	(0.4)	(0.9)	(0.3)
Regression Summary									
Life-cycle controls I	Y	N	Y	Y	N	Y	Y	N	Y
Individual Fixed Effect	N	Y	N	N	Y	N	N	Y	N
Preference Shifters	N	N	Y	N	N	Y	N	N	Y
R2: Couples (A)	0.65	0.00	0.65	0.65	0.00	0.65	0.65	0.00	0.65
R2: Singles (A)	0.57	0.00	0.57	0.57	0.00	0.57	0.57	0.00	0.57
R2: Couples (B)	0.55	0.00	0.55	0.55	0.00	0.55	0.55	0.00	0.55
R2: Singles (B)	0.67	0.00	0.67	0.67	0.00	0.67	0.67	0.00	0.67
NOBS(k): Couples	667.2	667.2	667.2	661.4	661.4	661.4	660.2	660.2	660.2
NOBS(k): Singles	569.6	569.6	569.6	567.5	567.5	567.5	567.5	567.5	567.5

The table presents the estimate on the causal impact of a cancer diagnosis during 2012 on financial wealth as of December 2011. Panel A has the natural logarithm of financial wealth as the dependent variable while Panel B has financial wealth measured in US Dollars as the dependent variable. In addition to the coefficient estimates, both panels include t-statistics in parenthesis, information about which independent variables that are included in the regressions, R2, and the sample size (under Regressions Summary). The table contains three main columns with different prognosis for the cancer cases. The first main column uses all cancer cases (stage 1-4), the second column uses only good prognoses (stage 1), and the last column uses only poor prognosis (stage 2 and stage 3). Each of the three main column is dividend into three minor columns, each of which represents a particular regression model. The differences between the regressions models are the control variables included in the regression. Life-cycle controls include age, age2, and age3; indicator for completed college education, and its interaction with age and age2, and dummy variables for gender and their interaction with age and age2, income, which is the sum of pension and labor income, financial wealth, leverage, and a dummy variable that takes the value of one if the family structure has children, and the actual number of children. The control variable lagged financial wealth is measured in the same units as the outcome variable. Preference shifters include three zero-one dummy variables that take the value of one if there is a change in the household composition (couples versus single and the number of children) during the period of the analysis.

Table 4: Are Children a Motive for Leaving Bequest Among Singles?

$$O_{it+1} = \alpha + \beta_C C_{it\in(t-1,t]} + \beta_I (C_{it} \times D_{Ch_{it-1}}) + \rho X_{it-1} + u_{it+1}$$

Outcome Variable:	$O_{it+1} = \ln(FW_{it+1})$			$O_{it+1} = FW_{it+1}$		
Prognosis:	All	Good	Poor	All	Good	Poor
β_C	-0.13	-0.01	-0.32	-274	516	-7 178
t(β_C))	-(3.4)	-(0.1)	-(5.1)	-(0.2)	(0.2)	-(2.5)
β_I	-0.12	-0.12	-0.12	-5 086	-5 071	-5 071
t(β_I)	-(2.7)	-(0.8)	-(2.3)	-(2.1)	-(0.4)	-(0.4)
Child Dummy ($D_{Ch_{it-1}}$)	-0.12	-0.12	-0.12	-5 086	-5 071	-5 071
t($D_{Ch_{it-1}}$)	-(20.3)	-(20.3)	-(20.4)	-(19.2)	-(19.2)	-(19.2)
# Children	-0.01	-0.01	-0.01	-845	-854	-850
t(# Children)	-(4.9)	-(4.9)	-(4.8)	-(7.8)	-(7.9)	-(7.8)
Regression Summary						
Life-cycle controls	Y	Y	Y	Y	Y	Y
R2	0.56	0.56	0.56	0.67	0.67	0.67
NOBS(k)	568.7	567.4	567.0	568.7	567.4	567.0

The rows in the table provides estimate on the causal impact of a cancer diagnosis on financial wealth, corresponding t-statistics in parenthesis, information about independent variables that are included in the regressions (under Regression summary), R2, and the sample size. All coefficients report the least square estimate of the cancer dummy variable (β_C) for singles diagnosed with cancer during 2010, and the interaction term (β_I) which is equal to one if the single individual who is diagnosed with cancer has one or more children prior to the diagnosis. Child dummy ($D_{Ch_{it-1}}$) refer to a dummy variable which is equal to one if the single individual has children in 2009, and # Children represents the number of children the individual has in 2009. The table contains two main columns. The first main column has the natural logarithm of financial wealth as the outcome variable, while the second column has the financial wealth measured in US Dollars as the dependent variable. Each of the two main column is dividend into three minor columns. The first minor column includes all prognoses, the second column includes only good prognoses, while the last column includes only poor prognosis. Life-cycle controls include age, age2, and age3; indicator for completed college education, and its interaction with age and age2, and dummy variables for gender and their interaction with age and age2, income, which is the sum of pension and labor income, financial wealth, and leverage. The control variable lagged financial wealth is measured in the same units as the outcome variable.

Table 5: Single Parents with Cancer Transfer Wealth to their Children

$$FW_{Chit+1} = \alpha + \beta_C C_{Pait\in(t-1,t]} + \beta_I (C_{Pait} \times I_{t-1}) + \rho X_{t-1} + u_{it+1}$$

	Single DAD						**Single MOM**					
Model	**M1**	**M2**	**M3**	**M4**	**M5**	**M6**	**M1**	**M2**	**M3**	**M4**	**M5**	**M6**
β_C	3 429	2 960	2 025	-22	-1 641	5 162	6 781	7 630	3 758	4 521	20 074	-1 185
	(2.9)	(2.6)	(1.1)	(0.0)	-(0.7)	(0.5)	(2.1)	(2.4)	(0.8)	(1.3)	(0.6)	-(0.2)
$C \times FW_{Pait-1}$			0.02						0.08			
			(0.9)						(1.0)			
$C \times FW^2_{Pait-1}$			-0.00						-0.00			
			-(0.8)						-(1.1)			
$C \times FW_{Chit-1}$				0.15						-0.01		
				(5.6)						-(0.3)		
$C \times FW^2_{Chit-1}$				-0.00						0.00		
				-(10.4)						(7.7)		
$C \times Single_{Chit-1}$					6 183						-17 713	
					(0.4)						-(2.6)	
$C \times Child_{Chit-1}$						-3 740						14 048
						-(0.3)						(2.1)
Regression Summary												
Life Cycle controls	N	Y	Y	Y	Y	Y	N	Y	Y	Y	Y	Y
Financial Wealth Child	N	Y	Y	N	Y	N	N	Y	Y	N	Y	N
Financial Wealth Parent	N	Y	N	Y	Y	N	N	Y	N	Y	Y	N
R2	0.00	0.03	0.00	0.02	0.03	0.03	0.00	0.00	0.00	0.01	0.00	0.00
NOBS(k):	225.86	225.86	225.86	225.86	225.86	225.86	110.96	110.96	110.96	110.96	110.96	110.96

The rows in the table provides estimate of the causal impact a single parent's cancer diagnosis has on a child's financial wealth measured in US Dollars, corresponding t-statistics in parenthesis, information about which independent variables that are included in the regressions, R2, (under Regression Summary). All estimates are based on a poor cancer diagnosis (stage 2 or stage 3). The table contains two main columns which each includes the results from six different regression models. The first main column includes the cancer diagnosis of single fathers, and the second column includes the cancer diagnosis of single mothers. β_C refers to the cancer dummy variable which takes the value of one if an individual is diagnosed with cancer during 2010. All other independent variables are interactions with cancer (C) and a particular variable. Life-cycle controls include child's age, child's age2, parent's age, and age2. Financial Wealth below Life Cycle controls refer to whether lagged financial wealth of either child or parent is included in the regression.

Table 6: Mortality Shocks Over the Life-Cycle

$$O_{it+1} = \alpha + \beta C_{it\in(t-1,t]} + \rho X_{it-1} + u_{it+1}$$

Outcome Variable:	$O_{it+1} = \ln(FW_{it+1})$				$O_{it+1} = FW_{it+1}$			
Age	[30-44]	[45-59]	[60-74]	[75-85]	[30-44]	[45-59]	[60-74]	[75-85]
Couples (β)	0.35	0.13	0.04	-0.02	15 367	9 270	10 055	-1 527
t(β)	(3.5)	(3.3)	(2.0)	-(1.0)	(2.0)	(2.1)	(4.2)	-(0.7)
Singles (β)	0.26	-0.28	-0.39	-0.62	18 738	-4 196	-3 343	-17 020
t(β)	(1.5)	-(2.8)	-(7.8)	-(15.5)	(3.3)	-(0.8)	-(0.9)	-(6.4)
Regression Summary								
Life-cycle controls	Y	Y	Y	Y	Y	Y	Y	Y
R2: Couples	0.52	0.60	0.70	0.79	0.33	0.43	0.64	0.79
R2: Singles	0.44	0.57	0.68	0.65	0.51	0.63	0.71	0.76
NOBS(k): Couples	169.4	247.8	189.8	52.2	169.4	247.8	189.8	52.2
NOBS(k): Singles	274.7	136.8	79.7	75.6	274.7	136.8	79.7	75.6

The rows in the table provides estimate on the causal impact of cancer on financial wealth, corresponding t-statistics in parenthesis, information about which independent variables that are included in the regressions, R2, and the sample size (under Regression statistics). All results in this table are based on a poor cancer diagnosis (cancer in stage 2 or stage 3). The table contains two main columns. The first main column has the natural logarithm of financial wealth as the outcome variable, while the second column has the financial wealth measured in US Dollars as the dependent variable. Each of the two main column is dividend into four minor columns, which each represent a particular stage in the life-cycle. Life-cycle controls include age, age2, and age3; indicator for completed college education, and its interaction with age and age2, and dummy variables for gender and their interaction with age and age2, income, which is the sum of pension and labor income, financial wealth, leverage, and a dummy variable that takes the value of one if the family structure has children, and the actual number of children. The control variable lagged financial wealth is measured in the same units as the outcome variable.

Table 7: Mortality Shock Across the Wealth and Leverage Distribution over the Life-Cycle

$$O_{it+1} = \alpha + \beta C_{it\in(t-1,t]} + \rho X_{it-1} + u_{it+1}$$

Outcome Variable	$O_{it+1} = \ln(FW_{it+1})$				$O_{it+1} = FW_{it+1}$			
Panel A: Financial Wealth Pctl	[25,50]	[50,75]	[75,90]	[90, 99]	[25,50]	[50,75]	[75,90]	[90, 99]
Couples (β)	0.13	0.05	0.01	-0.04	7 741	8 913	8 181	2 780
t(β)	(4.3)	(1.7)	(0.5)	-(1.3)	(5.9)	(3.1)	(2.4)	(0.4)
Singles (β)	-0.38	-0.40	-0.53	-0.52	427	-3 448	-15 323	-34 661
t(β)	-(5.4)	-(6.7)	-(8.8)	-(6.5)	(0.3)	-(1.2)	-(3.1)	-(2.8)
Panel B: Leverage Pctl	[0,25]	[25,50]	[50,75]	[75, 99]	[0,25]	[25,50]	[50,75]	[75, 99]
Couples (β)	0.02	0.03	0.09	0.03	5 935	6 417	10 059	-303
t(β)	(1.0)	(1.5)	(2.3)	(0.3)	(1.7)	(2.2)	(2.6)	-(0.1)
Singles (β)	-0.42	-0.50	-0.24	-0.08	-15 485	-2 577	2 486	2 327
t(β)	-(8.4)	-(7.1)	-(2.2)	-(0.4)	-(5.1)	-(0.6)	(0.5)	(0.6)
Panel C: Financial Wealth and Leverage Groups (G1-G4)								
Couples	G1	G2	G3	G4	G1	G2	G3	G4
Age < 70 (β)	0.17	0.03	0.10	0.03	13 023	14 211	7 172	5 725
t(β)	(5.7)	(0.8)	(0.8)	(0.8)	(6.7)	(0.3)	(1.3)	(0.1)
Age ≥ 70 (β)	-0.07	-0.16	0.05	-0.01	-1 397	-16 244	5 617	1 772
t(β)	-(1.8)	-(4.0)	(1.3)	-(0.5)	-(0.1)	-(2.0)	(2.0)	(0.1)
Singles	G1	G2	G3	G4	G1	G2	G3	G4
Age < 70 (β)	-0.25	-0.29	-0.32	-0.21	3 254	13 329	-4 512	-11 219
t(β)	-(2.8)	-(2.1)	-(2.3)	-(2.1)	(1.4)	(0.8)	-(1.2)	-(0.8)
Age ≥ 70 (β)	-0.65	-0.87	-0.35	-0.64	-3 962	-28 059	-2 447	-37 978
t(β)	-(8.1)	-(7.3)	-(5.0)	-(12.8)	-(1.3)	-(1.8)	-(1.1)	-(6.1)
Regression Summary								
Life-cycle controls	Y	Y	Y	Y	Y	Y	Y	Y
R2: Couples (A)	0.29	0.12	0.13	0.21	0.73	0.62	0.49	0.31
R2: Singles (A)	0.27	0.09	0.10	0.18	0.65	0.55	0.41	0.23
R2: Couples (B)	0.05	0.03	0.08	0.20	0.66	0.51	0.19	0.11
R2: Singles (B)	0.07	0.07	0.13	0.31	0.74	0.54	0.29	0.10
NOBS(k): Couples (A)	271.9	182.9	119.9	75.6	123.7	188.9	200.6	42.5
NOBS(k): Singles (A)	338.3	123.1	66.3	35.7	179.0	116.2	108.6	67.6

The rows in the table provides estimate on the causal impact of cancer on financial wealth in both logs and levels, corresponding t-statistics in parenthesis, information about which independent variables that are included in the regressions, R2, and the sample size (for Panel A) and Panel B. (under Regression Summary). Further regression summary have no important implications for the interpretation of the results and is therefore omitted to save space. All results in this table are based on a poor cancer diagnosis. Panel A presents the result from subgroup analysis based on financial wealth percentiles prior to the diagnosis, Panel B presents the corresponding results from subgroup analysis based on leverage percentiles. Each of the two main column (log and level) is dividend into four minor columns, which each represent a particular percentile. Panel C includes the results for double sorting on financial wealth and leverage prior to the diagnosis. G1 and G2 refer to leverage above the 25th pctl and financial wealth below (above) the 75th financial wealth pctl. G3-G4 refer to the same wealth percentiles, but include people with leverage below the 25th percentile (for a majority this means zero leverage). Life-cycle controls include age, age2, and age3; indicator for completed college education, and its interaction with age and age2, and dummy variables for gender and their interaction with age and age2, income, which is the sum of pension and labor income, financial wealth, leverage, and a dummy variable that takes the value of one if the family structure has children, and the actual number of children. The control variable lagged financial wealth is measured in the same units as the outcome variable.

Table 8: Treatment or Life Expectancy?

$$FW_{it+1} = \alpha + \sum_{j=1}^{3} \beta_j RS^j_{it\in(t-1,t]} + \rho X_{it-1} + u_{it+1}$$

	All			**Stage 1**			**Stage 2 and 3**		
5-Year Survival (RS)	$(0, 0.3]$	$(0.3, 0.7]$	$(0.7, 1]$	$(0, 0.3]$	$(0.3, 0.7]$	$(0.7, 1]$	$(0, 0.3]$	$(0.3, 0.7]$	$(0.7, 1]$
Couples (β)	95 882	10 182	9 304	145 093	16 828	8 896	95 994	23 264	19 292
t(β)	(5.4)	(0.6)	(1.2)	(2.0)	(0.5)	(0.9)	(5.2)	(0.8)	(1.3)
Singles (β)	-114 792	-31 632	-7 674	-201 861	8 646	-8 066	-106 069	-85 270	-9 999
t(β)	-(6.0)	-(1.9)	-(0.9)	-(3.2)	(0.2)	-(0.7)	-(5.3)	-(3.2)	-(0.6)
F-Test H0: $\beta_1 = \beta_2 = \beta_3$ conditional on cancer stage									
		F-test	P-value		F-test	P-value		F-test	P-value
Couples		10.39	<.0001		1.75	0.1736		5.76	0.0032
Singles		13.18	<.0001		4.74	0.0087		7.9	0.0004
Regression Summary									
Life-cycle controls	Y	Y	Y	Y	Y	Y	Y	Y	Y
R2: Couples	0.6707	0.6707	0.6707	0.6708	0.6708	0.6708	0.6706	0.6706	0.6706
NOBS(k): Couples	568.75	568.75	568.75	567.37	567.37	567.37	566.97	566.97	566.97
R2: Singles	0.553	0.553	0.553	0.5525	0.5525	0.5525	0.5522	0.5522	0.5522
NOBS(k): Singles	665.00	665.00	665.00	660.58	660.58	660.58	659.25	659.25	659.25

The rows in the table provides estimate on the causal impact of cancer as a function of relative survival (RS), given cancer stage, on financial wealth, corresponding t-statistics in parenthesis, information about which independent variables that are included in the regressions , R2, and sample size (under Regression summary). Relative survival (RS) is defined as the observed survival in a patient group divided by the survival of a comparable group in the general population with respect to key factors affecting survival such as age, sex and calendar year of investigation. The outcome variable is financial wealth in US Dollars. The first main column includes all cancer stages, the second column includes only cancer diagnoses discovered in stage 1, and the last column shows the results from cancer diagnoses discovered in stage 2 or 3. The table reports the least square estimate of the cancer dummy variable for households and singles that are diagnosed with cancer during 2010. They show the causal impact of cancer on financial wealth in 2011. Life-cycle controls include age, age2, and age3; indicator for completed college education, and its interaction with age and age2, and dummy variables for gender and their interaction with age and age2, income, which is the sum of pension and labor income, financial wealth, leverage, and a dummy variable that takes the value of one if the family structure has children, and the actual number of children. The control variable lagged financial wealth is measured in the same units as the outcome variable.