What Can Shocks to Life Expectancy Reveal About Bequest Motives?

(Preliminary)

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Abstract

This paper infers 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 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

Little is known regarding why individuals desire to leave bequests if they do at all (Kopczuk and Lupton, 2007). Understanding individuals' bequest motives are important to understand capital accumulation and the time horizon for economic decisions, including investment decisions and portfolio choice. Theoretical motivations for leaving bequests include altruistic motives suggested by Becker (1974); Barro (1974) and strategic reasons proposed by Bernheim et al. (1985). However, intentional bequest savings have been considered empirically indistinguishable from other life-cycle savings (Dynan et al., 2002). The reasons is that since death is unpredictable, even those who plan to exhaust all financial resources before death tend to leave bequests (David, 1981; Abel, 1985).

Similar to intentional bequests, transfers of wealth during a person's lifetime, so-called *inter vivos* transfers, will arise in dynastic models in which family members derive utility from the well-being of other family members (Gale and Scholz, 1994). Previous studies from survey data have found that between 15% to 31% of total household wealth comes from inherited wealth (Modigliani, 1988; Gale and Scholz, 1994; Laitner and Juster, 1996; Menchik and David, 1983; Hurd and Mundaca, 1989). However, as pointed out by Kopczuk and Lupton (2007), it is unclear whether individuals can accurately distinguish inherited wealth from that stemming from their own labor income. Even with perfect memory, the estimates of inherited wealth include both intentional and accidental bequests, and may therefore say little about saving behavior.

Using a unique dataset collected from Norwegian register data on cancer diagnoses, which are unpredictable at the individual level, income, wealth, and family linkages of all citizens in Norway from 2009-2011, I identify intentional bequest motives for spouse and children from shocks to mortality risk (cancer diagnoses). Using the family linkages in the data, I provide estimates of actual *inter-vivos* transfers after an increase in mortality risk of a parent, and investigate potential effect modifiers in the relation between mortality and *inter-vivos* transfers. The random nature of cancer at the individual level ensures causal interpretation.[1]

A cancer diagnosis is useful for identifying intentional bequest motives because it reduces the individual's expected remaining life span, which changes the person's consumption and savings

[1]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.

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 life expectancy falls. Given initial wealth and resources, the wealth of individuals who also save for bequest reasons, and therefore derive utility from passing on wealth to either spouse or children, will decline more slowly than someone without a bequest motive after experiencing the same shock to morality. Consequently, the sensitivity of savings with respect to changes in mortality risk (cancer) is informative about the strength of intentional bequest motives.

There are two reasons for why a cancer diagnosis is primarily perceived as a shock to life expectancy in Norway. First, specialized and standardized cancer treatment is promptly available, almost free of charge, and private treatment is almost nonexistent (Molven and Ferkis, 2011; Fiva et al., 2014).[2] This alleviates 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.[3] 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.

This paper contributes to the large body of research that followed from the dispute between Kotlikoff and Summers (1981) and Modigliani (1988) about whether intentional bequest motives are necessary in addition to life-cycle saving motives to explain the aggregate stock of capital. There are at least five important findings: (i) A spouse creates a direct bequest motive. At the end of the calendar year after the diagnosis, couples with a poor cancer diagnosis (low expected survival), have about 7% (t-value of 4.1) more financial wealth than non-diagnosed comparables. The strong bequest motive for spouse is consistent with the finding in Inkmann and Michaelides (2010) that married households hold significantly larger amounts of all life insurance forms.

(ii) In contrast to couples, there is a horizon effect in the saving choices of singles. As the expected life span goes down, the representative single household reduces its wealth. The estimated coefficient of the interaction term between being single prior to the diagnosis and being diagnosed with cancer (β_{cs}) is -18% (t-value of 5.2) using net worth and -15% (t-value of

[2]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.

[3]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.

4.5) using financial wealth. In consonance with Hurd (1987), singles with children reduce their wealth more than singles without children after a cancer diagnosis.

(iii) Direct evidence of inter vivos transfers with a gender bias; having a father diagnosed with a poor diagnosis leads to an increase in financial wealth of about 7% (t-value of 3.0) whereas having a mother diagnosed with poor diagnosis causes an increase of about 12% (t-value of 2.9). These estimates of *inter-vivos* transfers which reflects only intentional transfers differ from previous estimates which are based on either the total amount of wealth that has been inherited or the amount of savings planned for bequest (Modigliani, 1988; Gale and Scholz, 1994; Laitner and Juster, 1996; Menchik and David, 1983; Hurd and Mundaca, 1989). Moreover, I investigate four potential effect modifiers of the relation between mortality risk and *inter-vivos* transfers. First, being single prior to the diagnosis does not affect the transfer amount. Consequently, the large reduction in wealth among singles cannot be fully explained by *inter vivos* transfers. Second, I find weak evidence in favor of women transferring more when their children are relatively poor. Third, the transfer amount appears independent of the wealth of the parent, which is inconsistent with the common perception that bequest enters the utility functions directly as a luxury good (DeNardi, 2004; Kopczuk and Lupton, 2007; Ameriks et al., 2011; Lockwood, 2014). Fourth, only children of fathers receive more than four times that of an otherwise comparable children with siblings, which may indicate a pronounced strategic bequest motive among men.

(iv) A life-cycle component in how savings of households respond to mortality risk. For couples, the propensity to save after a cancer diagnosis decreases monotonically over the life-cycle. At the beginning of the life-cycle (30-44 years), couples increase savings by about 47% (t-value of 4.5) while couples between 75-85 years do nothing. A related pattern emerges for singles. At the first stage of the life-cycle (30-44 years), there is statistically no difference between couples and singles, implying that singles too respond to a mortality shock by saving more. In contrast, singles between 75-85 years decrease their financial wealth by about 63% (t-value of 10.1).

(v) Net worth affects the relationship between mortality risk and savings, but less than the stage in the life-cycle. For couples, the tendency to increase savings after a cancer diagnosis remains statistically significant with a point estimate of about 10% for all wealth percentiles except for the richest (net worth between 75-99 percentile), who do nothing. Singles belonging to the 0-25 net worth percentile decrease savings by about 47% relative to couples (t-value of 4.2), while singles belonging to the 75-99 net worth percentile decrease savings by about 54% relative to couples (t-value of 7.3).

(vi) Results from three different empirical tests provides strong support that the results are

driven by news about expected survival, and not treatment, diagnosis per se, or changes in the utility function. First, both the estimated increase in wealth of couples and the reduction in wealth of singles using the full sample are subsumed by poor prognosis (low expected survival). Good prognoses (high expected survival) have no impact on saving choices, which is inconsistent with the notion that cancer has a large impact on marginal utility of consumption. The reason is that savings would adjust if cancer would affect marginal utility of consumption but not the discount factor on future utility. Second, using the variation in expected survival within good and poor prognosis (cancer stage), I show that the average causal effect of a cancer diagnosis with low expected survival is about the same. This result is an 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.[4] 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 mortality shock by consuming or saving. In particular, the strong bequest motive for spouse lends support to "unitary" (same discount factor) models of household intertemporal allocation, in which the caring for spouse fully reverses the impact of the decrease in survival probability on the discount factor.

The rest of the paper is organized as follows. Section 2 describes the data, including a detailed description of the implication of a cancer diagnosis on life expectancy, variable construction, and summary statistics. Section 3 relates a model of savings to empirical predictions about mortality risk and savings under different bequest motives, and discuss various econometric issues. The results are presented in section 4-7, and a discussion on the validity of the study is included in section 8. Lastly, I conclude in Section 9

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

[4] 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).

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. (2016), 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. 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.[5]

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,[6] 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. (2016).

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

[5] http://www.kreftregisteret.no/Global/Cancer%20in%20Norway/2013/CIN_2013.pdf
[6] http://www.ssb.no/utdanning/_attachment/159352?_ts=143b5108f70

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.[7]

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 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 estimates 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.[8] 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). Adding stage 2 to stage 3 ensures a sample of all cancer types. To eliminate any concern about sample selection, all analyses also include the results obtained from the full

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

[8] 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.

sample (stages 1-4). For the sake of simple notation, I refer to stage 1 as "Good" prognosis and stage 2 and stage 3 as "Poor" prognosis. "All" stands for the full sample including all stages. Figure 2 shows the realized absolute survival rates for singles diagnosed with cancer during 2010 conditioned on being alive at the end of 2010.

[Insert Figure 2 here]

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 Defining Household Wealth

Two measures of wealth are the main outcome variables in this paper; net worth, defined as the sum of financial wealth, durable consumption goods, and real estate, after subtracting the gross debt, and financial wealth constructed from Tax Returns following the setup in Calvet and Sodini (2014). 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. Real estate and the stock of durable consumption goods make up the residual category of the household total assets.[9] Because it is a priori difficult to determine which is the "correct" estimate of wealth, the introductory analysis uses both.

Net worth is perhaps the best proxy of the state variable that may influence how shock to life expectancy affects savings. In particular in Norway, where housing wealth is of high relative importance.[10] Yet net worth is theoretically appealing, inclusion of real estate, which is both relatively illiquid and reported at tax value, might bias the estimate on how shocks to life expectancy impact savings for at least three reasons.[11] First, since debt is reported at market value while real estate is not, the inferred saving choice derived from the first difference in net worth is subject to severe measurement-error, which makes it challenging to interpret the magnitudes of the coefficients. In order to reduce the measurement-errors resulting from tax valuation of real estate, I convert tax values to market values by using a factor of five obtained

[9] I do not include other assets such as investment in private firms as these assets are subject to severe measurement error, and most households do not have any material wealth in such assets.

[10] In 2015, 84% of the population above 16 years were living in a house owned by the household: https://www.ssb.no/bygg-bolig-og-eiendom/statistikker/bo/hvert-3-aar/2015-11-25

[11] Housing had a weak relation to actual market values before 2010 (Fagereng and Halvorsen, 2016).

from Statistics Norway.[12] Second, 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. Therefore, 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 wealth caused by a negative shock to life expectancy would be misleading in favor of a bequest motive. Third, if the individual who is experiencing a negative shock to life expectancy would sell the real estate to increase consumption, any systematic differences between estimated market values of real estate based on tax values and actual market value, may lead to incorrect estimate of mechanism between life expectancy and savings. For example, if the estimated values are lower than market values, selling the house to increase present consumption may appear in the tax returns as a positive change in wealth.

Financial wealth is an alternative proxy for wealth that overcomes the potential issues with net worth listed above. It consists solely of liquid assets reported at market values provided by a third-party. Financial wealth is therefore essentially free of measurement-error and can be traded at small transaction costs. However, a potential concern with financial wealth is its relative importance. To empirically investigate the relative importance of financial wealth, Figure 3 plots the average financial wealth relative to total assets against age for couples and singles. The figure shows that financial assets make up 25-70% of total assets depending on the family structure and the stage in the life-cycle, making it an important part of total savings, and a valid proxy for total wealth.

[Insert Figure 3 here]

2.4 Defining Family Structures

With the purpose of identifying both whether family provides a bequest motive and the relative strength of the potential bequest motive for spouse and children, I distinguish between three household types: couples, singles with children, and singles without children. Children is a common proxy of bequest (Hurd, 1987, 1989; Inkmann and Michaelides, 2010; Ameriks et al., 2011) and spouse is included because couples experiencing shocks to mortality may want to leave bequests also to the surviving partner (Bernheim et al., 2003; Inkmann et al., 2011; DeNardi et al., 2015). Moreover, couples are defined as in Fagereng et al. (2016); individuals registered as married and with valid ID for spouse, or registered with valid cohabitant ID and common

[12] https://www.ssb.no/priser-og-prisindekser/artikler-og-publikasjoner/3600-milliarder-i-boligformue

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. Single households are defined as widows or widowers, those who never married, or divorced. The definitions of single and couple are mutually exclusive, meaning that no individual is defined as both.

For a meaningful quantification of the strength of the bequest motives towards spouse and children, preference for any of the three household types must be uncorrelated with the preference parameters that determines the utility from non-bequeathed wealth. For example, if the probability of being a couple would correlate with the marginal valuation of future personal utility for a given level of wealth, the wealth change of couples after a shock to life expectancy would differ from singles regardless of marital status. To examine whether preference for either household types correlates with the preference for saving plans, Figure 4 plots the relative fraction of couples and singles as well as their ownership of financial wealth, real estate, and debt as of 31 December 2009. 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.

[Insert Figure 4 here]

The similar aggregate portfolio of singles and couples indicates similar distribution of preference parameters. The similarity means that the preference for either family structures are unlikely related to preference parameters for saving plans. As a result, the potential difference in sensitivity of wealth to mortality risk between household types is therefor likely a consequence of the family structure, whilst the family structure is not a result of the preference parameters for saving plans.

2.5 Summary Statistics and Conditional Random Assignment

Apart from the key variables defined above, I also include the following financial variables: 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 total assets. Demographic control variables include: gender, educational level, and the number of children. 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.

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. Panel A in Table 1 reports the corresponding summary statistics.

[Insert Table 1 here]

Under the null hypothesis of random assignment of cancer conditional on age, households diagnosed with cancer (cases) should be statistically indistinguishable from non-diagnosed households (controls) of the same age. Panel B in Table 1 reports the results from testing the difference in means of the variables presented in Panel A between cases and controls at the age of 50, 60, 70, and 80. Out of the 80 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, giving causal interpretation of the impact of a cancer diagnosis on wealth.

3 Econometric Issues

3.1 Identification

The Appendix includes a model of saving in the presence of bequest motives which is inspired by Hurd (1987). The model has two key predictions. First, given initial wealth, an increase in mortality risk, which is uncorrelated with the distribution of future random variables affecting future personal utility, decreases next period wealth unless the utility from leaving bequest equates to the marginal utility of all future personal utility. In this case, mortality risk has no impact on savings. Consequently, comparing the change in wealth of a household experiencing a shock to mortality risk with a similar household with no change in mortality risk, is informative of whether spouse or children provide a strong enough bequest motive to neutralize the effect of mortality risk on savings. In a similar vain, comparing the sensitivity of changes in wealth to increases in mortality risk across family structures is informative about the relative strength of bequest motives for different household members.

3.2 Timing

All cross-sectional regressions are carried out using a measure of 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).[13] 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.[14] Taken together, the relatively long period between the diagnosis and the time the outcome variable is measured alleviates potential concerns that the results reflect either temporary changes in wealth caused by treatment or health state dependence.

3.3 Life-cycle controls

In order to get consistent estimates of the causal affect of a cancer diagnosis on savings, 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 wealth and cancer incidence rates increase over the life-cycle. To capture such life-cycle effects, all regressions are run conditional on a vector of life-cycle controls. The conditioning variables in include a broad range of variables related to the life-cycle that are either constant or known before the cancer diagnosis (t). 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, dummy variables for gender and their interaction with age and age2, dummy variables for having children and their interaction with age, age2, and household type (single or couple), number of children, income, which is the sum of pension and labor income, and leverage defined as debt divided by total assets.

[13] 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.

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

3.4 Placebo Regressions

To ensure that developing cancer does not correlate with unobserved determinants of savings, I conduct an identical analysis but with households 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 regressions 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.[15]

4 A Direct Test of Family as Proxy for Bequest

4.1 Empirical Specification

Guided by the theoretical model in the Appendix, the baseline empirical strategy is to regress financial wealth or net worth (W_{it+1}) onto a cancer dummy variable ($C_{it\in(t-1,t]} \triangleq C_{it}$) that takes the value of one if a household is diagnosed with cancer during year t, a vector of covariates (X_{it-1}), and the lagged outcome variable (W_{it-1}). To capture potential differences in the strength of the bequest motives towards spouse and children, I add two interaction terms. The first interaction term is the product of the cancer dummy (C_{it}) and a dummy variable that takes the value of one if the household is single prior to the diagnosis ($S_{it-1} = 1$). The second interaction terms is the product of the first interaction terms and a dummy variable that takes the value of one if the household has offsprings prior to the diagnosis ($O_{it-1} = 1$).[16] The

[15] In an earlier version, I also estimated a simplified version of Equation (1) using individual fixed effects. To arrive at this model, I took the first difference of both sides of the regression equation. To make sure that none of the differenced independent variables were influenced by the cancer diagnosis, I only included a constant and the first difference of age2 and age3. The coefficients were almost identical to those reported here, and are therefore omitted. 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 coefficients. In conclusion, the coherent estimates across the three models (not reported) imply that a cancer diagnosis does not proxy for time-invariant preference parameters.

[16] An alternative specification would be to replace the triple interaction with the product of the cancer dummy variable and the dummy variable that takes the value of one if the household has children prior to the diagnosis. The benefit with this approach is that it would allow me to identify the marginal effect of having children regardless of whether the household is defined as couple or single prior to the diagnosis. However, the drawback with this empirical specification is that it leads to high multicollinearity because being a couples and having children is highly correlated. For that reason, the above procedure is used.

resulting regression equation is

$$w_{it+1} = \alpha + \beta_c C_{it} + \beta_{cs}(C_{it}S_{it-1}) + \beta_{cso}(C_{it}S_{it-1}O_{it-1}) + \gamma X_{it-1} + \chi w_{it-1} + u_{it+1} \qquad (1)$$

Lower case letters denote logs ($w \triangleq \ln W$).[17] Because the definitions of singles and couples are mutually exclusive, the average percentage change in wealth caused by the cancer diagnosis (i.e., $\exp(\beta_c) - 1$), provides an estimate of the strength of the bequest motive towards spouse ($S_{t-1} = 0$). Conditional on being single and having children prior to the diagnosis ($S_{t-1} = 1$ and $O_{t-1} = 1$), the average percentage change in wealth caused by the cancer diagnosis equals $\exp(\beta_c + \beta_{cs} + \beta_{cso}) - 1$. The coefficient on the triple interaction (β_{cso}) provides an estimate of the strength of the bequest motive towards children among singles. Comparing the the two estimates provides information about the relative strength of the bequest motive. For simplicity, when discussing the empirical estimates, I refer to the coefficient estimates (β_c) rather than $\exp(\beta_c) - 1$ as the percentage difference in means between the two groups.

4.2 Results

Results of the cross-sectional regressions of household wealth on the cancer dummy variable, its interaction with family structure, and control variables are reported in Table 2. Model (M1) presents the results using all diagnosis while model (M2) distinguishes between good diagnosis (high expected survival) and poor prognosis (lower expected survival). Results from "NW" have net worth as the outcome variable while results from "FW" are based on financial wealth.[18]

[Insert Table 2 here]

The coefficients on the cancer dummy variable (β_c) show that couples do not reduce their savings after receiving a shock to life expectancy, indicating a direct evidence of bequest motive toward spouse. This finding lends support to "unitary" (same discount factor) models of household intertemporal allocation, in which the utility from leaving bequest to spouse equates to the marginal utility of all future personal utility. Put differently, the caring for spouse fully reverses the impact of the decrease in survival probability on the discount factor. In light of most people

[17] 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.

[18] The estimation error in the assessment of the market value of housing renders net worth negative for several households. As a result, the sample using net worth as the outcome variable contains fewer observations than the sample using financial wealth as the log transformation excludes negative values.

of working age having life insurance, including a lump sum payment to the surviving spouse and a smaller payment to children, the non-negative coefficients might reflect an incompleteness of the life-insurance market. However, I do not dwell more on this point.[19] Furthermore, the coefficient on the cancer dummy variable (β_c) using net worth (NW) as the outcome variable is indistinguishable from zero, while it is about 4% (t-value of 3.4) using financial wealth as the outcome variable. Overall, the strong bequest motive for spouse is consistent with the finding in Inkmann and Michaelides (2010) that married households hold significantly larger amounts of all life insurance forms.

As expected, there is a horizon effect in saving choices of singles. As the expected life span goes down, a representative single reduces its wealth. This finding is invariant to whether wealth is measured in net worth or financial wealth. The estimated coefficient of the interaction term between being single prior to the diagnosis and being diagnosed with cancer (β_{cs}) is -18% (t-value of 5.2) using net worth and -15% (t-value of 4.5) using financial wealth. The coherent estimates between both wealth measures reflect the high elasticity of changes in net worth with respect to changes in financial wealth among singles diagnosed with cancer; a 1% change in financial wealth is associated with a 0.7% (t-value of 40) change in net worth (not reported).[20]

The coefficient on the triple interaction (β_{cso}) provides an estimate of the strength of the bequest motive towards children among singles. Contrary to the common notion that having children provides a bequest motive, the coefficient estimate of the triple interaction terms (β_{cso}) is -17% (t-value of 4.1) using net worth and -13% (t-value of 3.0) using financial wealth. In other words, singles with children reduce their net worth by about 17% more than singles without children, and about 35% (18% + 17%) more than couples. The corresponding numbers using financial wealth as the outcome variable are about 13% (singles without children) and 28% (13% + 15%) relative to couples. The finding is in consonance with Hurd (1987), who finds that people with children decummulate their wealth faster than people without children.

[19] The Appendix [] contains a description of institutional settings in Norway, including the use of life-insurance products.

[20] The estimated elasticity is about twice as high for singles diagnosed with cancer compared with non-diagnosed comparable singles.

5 Do Households Respond to News About Life Expectancy?

5.1 Empirical Specification

Inferring bequest motives from cancer diagnoses hinges on the claim that a cancer diagnosis affects saving choices through its impact on life expectancy. To ascertain the empirical realism of this postulate, I specify a regression equation that includes both a good prognosis (high expected survival) and a poor prognosis (low expected survival). The resulting regression equation is

$$w_{it+1} = \sum_k \beta_c^k C_{it}^k + \sum_k \beta_{cs}^k (C_{it}^k S_{it-1}) + \sum_k \beta_{cso}^k (C_{it}^k S_{it-1} O_{it-1}) + \gamma X_{it-1} + \chi w_{it-1} + u_{it+1} \quad (2)$$

The interpretation of the estimated coefficients is identical to the previous interpretation, except that the cancer dummy variable ($C_{it}^k, k \in [G, P]$), which now has subscript k, depending on whether the household is diagnosed with a good prognosis ($k = G$) or a poor prognosis ($k = P$). Results obtained from this model are reported in Table 2 under the second model (M2).

5.2 Results

The key result from the model (M2) is that households experiencing large changes in life expectancy (poor prognosis) explains the previous results, consistent with postulate that a cancer diagnosis affects saving choices through its impact on life expectancy. The coefficients on the cancer dummy variable that takes the value of one if a household receives a good prognosis, as well as its interaction with family structures are insignificant. In stark contrast, the corresponding coefficients for poor cancer diagnoses are economically large in absolute value and statistically significant. In particular, the estimated coefficient of the interaction term between being single prior to a diagnosis and receiving a poor cancer diagnosis (β_{CS}^P) is -41% (t-value of 7.3) using net worth and -39% (t-value of 6.7) using financial wealth. Moreover, the coefficient of the interaction term between being single, having children prior to the diagnosis, and a poor diagnosis (β_{CSCh}^P) is -21% (t-value of 3.2) using net worth and -17% (t-value of 2.5) using financial wealth. Finally, the coefficient on the cancer dummy variable of a couple diagnosed with a poor cancer (β_C^P) is indistinguishable from zero using net worth (NW) as the outcome variable, while it is about 7% (t-value of 4.1) using financial wealth as the outcome variable. Going forward, unless otherwise stated, the estimation results are based on poor prognoses only.

The four last columns in Table 2 contain the placebo regressions. The coefficient estimates come from the same models using the same data as above except from the cancer population, which is now households diagnosed with cancer in year 2012 (instead of 2010). Because the cancer diagnosis it not yet known to the household, the estimates from the placebo regression should all be insignificant if a cancer diagnosis has a causal impact on wealth. Overall, the results provide strong support of causal interpretation. In model (M1), which is based on all diagnoses, couples that will be diagnosed with cancer during the next year (2012) have about 3% (t-value of 2.9) more net worth than otherwise comparable couples, and singles to be diagnosed with cancer next year have about 7% more financial wealth than their peers. All other coefficients obtained from model (M1) are indistinguishable from zero. When using net worth as the measure of household wealth, the small bias for couples remains after distinguishing between good and poor prognosis (M2). All other coefficients obtained from model (M2) are statistically insignificant. Overall, the placebo regressions provide strong support for causal interpretation. To save on space, results from placebo regressions are omitted in the coming results.

6 Inter-vivos Transfers and Mortality Risk

Similar to intentional bequests, transfers of wealth during a person's lifetime, so-called *inter vivos* transfers, will arise in dynastic models in which family members derive utility from the well-being of other family members (Gale and Scholz, 1994). An interesting question is whether households transfer wealth to their children after being diagnosed with cancer. In particular, whether *inter vivos* transfers can explain why singles with children reduce their wealth more than singles without children. The combination of bequest motives towards children and tax advantages of transferring wealth alive would result in *inter vivos* transfers.[21]

6.1 Empirical Specification

The empirical strategy is to project the change in financial wealth of children onto; a cancer dummy variable that takes the value of one if a parent is diagnosed with cancer, an interaction consisting of the product of the cancer dummy and a dummy variable that takes the value of

[21]The Inheritance Tax Act regulates inheritance tax and rates are determined annually by Parliament. Children can receive up to $ 80,000 tax-free inheritance. The next $ 56,000 is subject to 6% inheritance tax, while the remaining inheritance is taxed at 10%. Each parent can transfer a tax-free amount of about $ 6,300 per child per year. The amount of such transfers does not affect the tax-free part of the inheritance, but unused transfers cannot be transferred to next year. The possibility of reducing taxes by transferring wealth alive creates an incentive for *inter-vivos* transfers after a cancer diagnosis.

one if the parent or child satisfies a certain criteria prior to the diagnosis (I_{it-1}), a vector of controls (X_{it-1}), and a constant term α. The resulting regression equation is

$$\Delta w_{it+1} = \alpha + \sum_k \beta_c^k C_{it}^k + \beta_c^I (C_{it} I_{t-1}) + \beta^I I_{t-1} + \gamma X_{it-1}^* + u_{it+1} \tag{3}$$

The outcome variable is the (continuously compounded) growth in financial wealth from 2009 to 2011 ($\Delta w_{t+1} \triangleq \ln FW_{t+1} - \ln FW_{t-1}$), and age and age2 for both parent and child constitute the controls (X_{it-1}^*). Further, $k = \{A, G, P\}$ where A refers to all diagnoses, G to good prognosis, and P to poor diagnosis. Importantly, the interaction term is only included when the sample is restricted to poor diagnoses. The regression equation has two coefficients of interests. The first is the coefficient on the cancer dummy variable (β_c^k), which provides a direct test of *inter vivos* transfers. The second coefficient of interest is the interaction term (β_c^I), which explores potential effect modifiers of the relation between mortality shocks and inter-vivos transfers. To examine potential gender effects, all models are estimated for both mothers and fathers using separate equations. Table 3 presents the results from the estimation.

6.2 Results

The coefficient of the cancer dummy variable (β_{ck}), which takes the value of one if one of the parents is diagnosed with any type of cancer, is about 4% (t-value of 3.0) for fathers, but insignificant for mothers. However, after distinguishing between good and poor prognoses (second column), the coefficient of the dummy variable that takes the value of one if a parent is diagnosed with a poor diagnosis is positive and significant for both genders, giving direct evidence of *inter vivos* transfers. Interestingly, the coefficients reveal a gender bias; having a father diagnosed with a poor diagnosis leads to an increase in financial wealth of about 7% (t-value of 3.0) whereas having a mother diagnosed with poor diagnosis causes an increase of about 12% (t-value of 2.9). In other words, compared with men, single mothers or couples with a female household head, transfer about 70% more wealth per child after being diagnosed with cancer.

Previous studies using survey data have estimated that between 15% to 31% of total household wealth comes from inherited wealth (Modigliani, 1988; Gale and Scholz, 1994; Laitner and Juster, 1996; Menchik and David, 1983; Hurd and Mundaca, 1989). In order to compare the estimates in this paper with the above, I multiply the coefficient estimates (β_{cP}) with the average financial wealth of a child relative to that of a current retiree. The product provides a rough estimate of how much of the financial wealth at retirement would be due to this particular

transfer.[22] Plugging in the numbers, gives about 2.3% ($0.06 \times 240/600$) for fathers and about 6.0% ($0.12 \times 290/600$) for mothers.[23] At first, these estimate may seem small, but have in mind that they reflect only one of potential several *inter-vivos* transfers. In addition, the estimates constitute only of intentional transfers as opposed to the previous literature in which transfers are based on total inheritance received.

[Insert Table 3 here]

The results from the other empirical specifications in Table 3 (M3-M6) have four main findings. The three first are as follows. First, the coefficient on the interaction between being diagnosed with cancer and being single prior to the diagnosis is insignificant for both genders (M3). As a result, the large reduction in wealth among singles cannot be fully explained by *inter vivos* transfers. Second, the interaction between the cancer dummy variable and a dummy that takes the value of one if the child has net worth above the 25 percentile (not poor) prior to the diagnosis, is small and insignificant for fathers while it is negative and economically large (-14%) for mothers (M4), albeit with relative large standard errors (t-value of 1.6).[24] The negative coefficient on the interaction term reveals some evidence that women transfer less when their children are wealthier. Third, the results from the fifth model (M5) show that the transfer amount is independent of whether the parent belongs to the upper 75 percentile (rich) of the net worth distribution. This finding is inconsistent with the common perception that bequest enters the utility functions directly as a luxury good (among others, DeNardi (2004); Kopczuk and Lupton (2007); Ameriks et al. (2011); Lockwood (2014)).

The final result comes from specification (M6), which interact the cancer dummy variable with a dummy variable that takes the value of one if the child has no siblings (only child). The purpose with the interaction is to examine the likelihood of strategic bequest motives, that is, whether parents hold on to their wealth with the intention of receiving services in exchange for future inheritance. Because an only child is the sole recipient of the heritage (minimum ca. $ 170,000), only wealthy parents with more than one child can credibly use bequests to influence the behavior of their children.[25] Consequently, if strategic motivations are widespread, only

[22] The fraction at retirement financial wealth for the child that is due to this transfer is approx.: $T(1+r)^{(T-t)}/FV(FW_T^C) \approx \beta_{ck} FW_t^C (1+r)^{(T-t)}/FW_t^R (1+g)^{(T-t)} = \beta_{ck}(FW_C/FW_R)_t$ if $r = g$. Here T denotes the transfer, FW_C is the financial wealth of the child today, and FW_R is the financial wealth of a retiree today.

[23] The numbers correspond to the average financial wealth of an individual at the same age as the average child in the sample. The denominator is the approx. average financial wealth of a retiree in 2009).

[24] The unprecise estimate is partly due to relative high correlation between cancer dummy variable and the interaction term.

[25] The general principle from inheritance law is that the surviving spouse is entitled to 25% of the estate

children will be the main recipients of the *inter-vivos* transfers. Interestingly, the coefficient on the interaction term for fathers is 25% (t-value of 1.9), implying that the representative only child received more than four times that of an otherwise comparable child with siblings. This finding may indicate a widespread strategic bequest motive among men. The coefficient for mothers is insignificant.

7 Heterogeneity in Households' Response to Mortality Shocks

Having examined the saving responses to mortality shocks for representative households, this section explores to what extent households respond differently to mortality shocks both over the life-cycle and across the wealth distribution. Figure 5 plots net worth per capita (PC) for couples and singles against age. The representative household starts out at age 30 with substantial human capital and a house financed with debt (negative net worth is likely a consequence of estimation error in the valuation of real estate). Over the life-cycle, the human capital is converted into housing equity through down payment of the mortgage, and investments in financial assets. At retirement at 67 years, both couples and singles stop saving.

[Insert Figure 5 here]

7.1 Empirical Specification

The empirical strategy is to interact the cancer dummy variable with a dummy variable that takes the value of one if a household belongs to a particular stage in the life-cycle, or to a particular net worth percentile. The interaction terms are further interacted with a variable Z_{it-1}, which is either equal to the dummy variable that takes the value of one if the household is single prior to the diagnosis (S_{it-1}), or equal to a dummy variable that takes the value of one if the household is in retirement (R_{it-1}), defined as being above 67 years in 2009. The idea with the retirement indicator is to separate life-cycle effects from wealth effects, which are highly positively correlated. To avoid high multicollinearity among the interaction terms, I estimate separate equations for couples and singles when using the retirement interaction (i.e., $Z_{it-1} = R_{it-1}$). The resulting regression equation is

(minimum ca. $ 50,000) and children are entitled to 75%. Further, 2/3 of the inheritance belongs to heirs, but a family member can never require more than $ 170,000 of the estate. For example, a married individual with two children and a fortune of $ 10 million can choose to leave $ 340,000 ($ 170,000 to each) to the children and $ 50,000 to the spouse, and the remaining as donations to non-family members.

$$w_{it+1} = \alpha + \sum_{k=1}^{4} \beta_{Ik}(C_{it}I_{kit-1}) + \delta_{Ik}(C_{it}Z_{it-1}I_{kit-1}) + \gamma X_{it-1} + \chi w_{it-1} + u_{it+1} \quad (4)$$

In the life-cycle specification, the dummy variable that takes the value of one if a household belongs to a particular place at the distribution ($I_{kt-1} = LC_{kt-1}$) has the following ranges: [30-44], [45-59], [60-74], [75-85]. The corresponding ranges for net worth are ($I_{kt-1} = NW_{kt-1}$): [0-25), [25-50), [50-75), [75-99]. In the separate equations for couples and singles ($Z_{it-1} = R_{it-1}$), β_{NWk} is the causal effect of a cancer diagnosis on wealth for a working age household in the k percentile of net worth distribution, and $\beta_{NWk} + \delta_{NWk}$ is the corresponding estimate for the otherwise similar household in retirement. Results from the estimation are reported in Table 4.

7.2 Results

The first column shows how couples and singles respond to a mortality shock over the life-cycle. Overall, a negative shock to life expectancy has very different impact on the saving behavior over the life-cycle for both couples and singles. For couples, the propensity to save after a cancer diagnosis decreases monotonically over the life-cycle. At the beginning of the life-cycle (30-44 years), couples increase savings by about 47% (t-value of 4.5) while couples between 75-85 years do nothing. Inability to insure the expected labor income growth of the household head combined with a strong bequest motive towards spouse could generate this result. The intuition is that young household heads facing high labor income growth view part of the intended bequest 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. A related pattern emerges for singles. At the first stage of the life-cycle (30-44 years), there is statistically no difference between couples and singles, implying that singles too respond to a mortality shock by saving more. In contrast, singles between 75-85 years decrease their financial wealth by about 63% (t-value of 10.1). Since couples at the same stage of the life-cycle do nothing, the reduction in financial wealth of 63% corresponds to both the relative difference to couples, and the average causal effect of a cancer diagnosis on financial wealth for singles.

[Insert Table 4 here]

The second column in Table 4 shows how couples and singles respond to a mortality shock across the net worth distribution. Similar to age, both for couples and singles, wealth prior to the cancer diagnosis tends to work as an effect modifier of the relationship between mortality

risk and savings. For couples, the tendency to increase savings after a cancer diagnosis remains statistically significant with a point estimate of about 10% for all wealth percentiles except for the richest (net worth between 75-99 percentile), who do nothing. For singles, the tendency to decrease financial wealth increases somewhat monotonically in net worth. In particular, singles belonging to the 0-25 net worth percentile decrease savings by about 47% relative to couples (t-value of 4.2), while singles belonging to the 75-99 net worth percentile decrease savings by about 54% relative to couples (t-value of 7.3). Relative to the poorest singles, the richest singles reduce their financial wealth by about 16% more, which translates into large differences in monetary terms.

The high correlation between age and net worth makes it difficult to tell life-cycle effects apart from wealth effects. The last two columns in Table 4 presents the result for couples (third column) and singles (fourth column) including the retirement interaction ($Z_{it-1} = R_{it-1}$). Due to the presence of modest multicollinearity between the working age and retirement coefficients in this specification, some caution is warranted when interpreting the results. Nevertheless, the coefficient estimates for couples below 68 years in 2009 (the first four coefficients in the third column) are either positive and significant, or indistinguishable from zero. In contrast, couples in retirement (the last four coefficients in the third column), that are equally wealthy, reduce their financial wealth after being diagnosed with cancer. Similarly for singles, the propensity to reduce financial wealth after a cancer diagnosis is about twice as large for retirees compared with working age adults. Overall, the stage in the life-cycle seems to have stronger influence on how households respond to mortality shocks than net worth.

The relatively stable coefficient estimates over the net worth distribution imply that rich households in retirement reduce their wealth many times that of poorer households in monetary terms. Due to the immaterial differences in *inter-vivos* transfers between couples and singles, the large reduction in financial wealth among singles is unlikely transfers. Instead the saving responses are more inline with a combination of differences in life expectancy between the rich and the poor and a general reluctance to rely on social insurance in very old age (DeNardi et al., 2009, 2010).[26] In this context, wealthier households may save more because they expect to live longer –and are therefore more likely to need expensive private services in very old age. Consequently, 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

[26]For example, in the United States, 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).

longest.

8 Robustness

The external validity of reported results rests on the premise that the coefficient estimates reflect active choices (thereby revealed preferences), not preference shocks, or treatment biases. The purpose with this section is to empirically examine the possibility that such biases may have material impact on the estimation results.

8.1 Health State Dependence

If the shape of the utility function varies with health status, this will affect optimal life-cycle savings. Finkelstein et al. (2009) define health state dependence as the effect of health on the marginal utility of a constant amount of nonmedical consumption. A cancer diagnosis may have a negative impact on health, in particular in the initial period after the diagnosis, which may affect the marginal utility of consumption. However, both the sign and magnitude of any health state dependence caused by a cancer diagnosis is ambiguous; the marginal utility of consumption may decline as many consumption goods are complements to good health (tourism, travel sports, etc.). Alternatively, the marginal utility of consumption could increase with deteriorating health, as other consumption goods such as private assistance services may be substitutes for good health. Regardless of the sign and magnitude of any health state dependence caused by a cancer diagnosis, good prognoses (high survival probability) are likely informative about the impact of any health state dependence on the coefficient estimates. The reason is that a good prognosis would affect the marginal utility of current consumption with minimal impact on the discount factor on the utility of future consumption. Figure 1 shows the implications of a good prognosis on changes in wealth for different types of health state dependence.

MU Consumption	$(-)$		$(+)$		(0)	
Bequest	$(+)$	$(-)$	$(+)$	$(-)$	$(+)$	$(-)$
Good Prognosis	$\Delta W\uparrow$	$\Delta W\uparrow$	$\Delta W\downarrow$	$\Delta W\downarrow$	$\Delta W \rightarrow$	$\Delta W \rightarrow$

Figure 1: Optimal Savings under State Dependent Utility

The logic is as follows. Assuming first that cancer has a negative impact on marginal utility of consumption and that the household does not have bequest motives (for example singles without

children). In this case, a good diagnosis is likely to cause an increase in savings through the substitution effect ("postponing traveling"). If on the other hand, a cancer diagnosis has a positive impact on marginal utility of consumption, savings will fall in response to the increase in marginal utility of current consumption. Lastly, if cancer does not impact on marginal utility of consumption, a good diagnosis will have no effect on savings ("nothing has changed"). Strong bequest motives will limit the substitution effect to the point at which the marginal utility of consumption equates to the marginal utility of leaving bequest. The results presented in Table 2 show that a good cancer diagnosis has no impact on savings for any of the three family structures, a result consistent with cancer diagnoses having small impact on the marginal utility of consumption.

8.2 Treatment Bias

A final concern is that cancer treatment might be different across cancer stages (good and poor prognosis), which could have contrasting impact on marginal utility of consumption. 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 ascertain potential treatment biases embedded in the results, I redo the main analysis using the within cancer stage variation in survival. In this setup, if a particular cancer stage has a large impact on the marginal utility of consumption, the coefficient for that particular stage when using only diagnoses with high expected survival will be either positive or negative, depending on cancer affect the marginal utility of consumption. This control strategy hinges on the fact that treatment protocols are closer related to the cancer stage than to the 5-year survival rate.[27]

8.3 Empirical Specification

The empirical strategy is to replace the dummy variables for good and poor prognosis (based on cancer stages) with three dummy variables based on the 5-year relative survival (RS). In this context, RS_1 is a zero-one dummy variable that takes the value of one if $RS \in (0, 0.3]$, RS_2 corresponds to $RS \in (0.3, 0.7]$, and RS_3 completes the survival measure $RS \in (0.7, 1]$. The

[27] 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.

cutoffs are equally spaced and chosen to ensure dispersion among cancer stages and types. The regression equation is

$$w_{it+1} = \alpha + \sum_{k=1}^{3} \beta_k RS_{kit} + \gamma X_{it-1} + \chi w_{it-1} + u_{it+1} \tag{5}$$

The only difference between the specification in Eq. (5) and the baseline model is the swap between stages ("Good" and "Poor") and relative survival (RS). If households respond to news about expected survival, the coefficients of the RS dummy variables should exhibit a monotonic pattern in k, and we should be able to reject the null hypothesis that all the dummy coefficients $(\beta_1, \beta_2, \beta_3)$ are identical. The results from the estimation are presented in Table 5.

8.4 Results

The results provides strong support for the notion that the coefficient estimates reflects revisions in saving plans due to news about life expectancy and not shift in the utility function. Regardless of the stages included in the regression, the estimated cancer dummy coefficients $(\beta_1, \beta_2, \beta_3)$ exhibit a monotonic pattern in k for both couples and singles, which is a clear indication that households respond to information about expected survival. The insignificant impact on savings from a cancer diagnosis with a five year relative survival (RS) between 0.7-1.0 (average ca. 0.9) is difficult to reconcile with state dependent utility given the modest bequest motive for children; if a cancer diagnosis effect the marginal utility of consumption but not the discount factor on future utility, then next period wealth will differ from a comparable household not diagnosed with cancer. The last Panel in Table 5 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.

[Insert Table 5]

9 Conclusion

By using a unique dataset collected for Norwegian register data on cancer diagnoses, financial wealth, and family linkages, this paper estimates the causal effect of mortality risk on wealth, and infers bequest motives from these estimates. The results show that family structure is an

effect modifier in the relation between mortality risk and savings, consistent with the presence of intentional bequest motives towards family members.

Regardless of survival probability, a cancer diagnosis does not cause couples to reduce their wealth. The strong bequest motives towards spouse indicates that the utility from leaving a bequest to spouse equates to the marginal utility of all future personal utility. The fact that the caring for spouse fully reversed any impact of mortality risk on the discount factor of future utility is supportive of "unitary" (same discount factor) models of household intertemporal allocation.

The high negative mortality risk sensitivity of savings among singles is consistent with typical life-cycle saving motives. As the expected life span goes down, singles without children reduce their net worth by about 18% (t-value of 5.2) and their financial wealth by about 15% (t-value of 4.5). Moreover, singles with children reduce their wealth even more than singles without but some of which reflects *inter-vivos* transfers. Inconsistent with the common perception that bequest enters the utility functions as a luxury good, the *inter-vivos* transfers appear independent of the wealth of the parent.

Estimates of *inter-vivos* transfers from both couples and singles show that having a father diagnosed with a poor diagnosis leads to an increase in financial wealth of about 7% (t-value of 3.0) whereas having a mother diagnosed with poor diagnosis causes an increase of about 12% (t-value of 2.9), indicating a gender bias. Opposed to previous estimates of *inter-vivos* transfers, these estimates reflects solely intentional transfers.

Both age and net worth prior to the cancer diagnosis impact how households respond to a shock to life expectancy; at the beginning of the life-cycle both couples and singles respond to a cancer diagnosis by saving more. At the end of the life-cycle couples stop saving, and singles reduce their financial wealth substantially. The propensity to reduce wealth after a cancer diagnosis seems to increase in net worth, implying that the mortality risk sensitivity to savings is the highest for rich singles and poor couples.

Because *inter-vivos* transfers are non-decreasing in the wealth of the parent, the high negative mortality risk sensitivity to savings among singles cannot be explained by a combination of bequest motive towards children and tax incentives to transfer wealth alive. Instead, the high sensitivities may reflect a combination of differences in life expectancy between the rich and the poor and a reluctance among retirees to rely on social insurance in old age.

References

Abel, A. B. (1985). Precautionary saving and accidental bequests. *American Economic Review*, 75(4):777–791.

Ameriks, J., Caplin, A., Laufer, S., and Nieuwerburgh, S. V. (2011). The joy of giving or assisted living? Using strategic surveys to separate public care aversion from bequest motives. *Journal of Finance*, 66(2):519561.

Barro, R. J. (1974). Are government bonds net wealth? *Journal of Political Economy*, 86(6):1095–1117.

Becker, G. (1974). A theory of social interactions. *Journal of Political Economy*, 82(6):1063–93.

Bernheim, B. D., Shleifer, A., and Summers, L. H. (1985). The strategic bequest motive. *Journal of Political Economy*, 93(6):1045–1076.

Bernheim, D. B., Forni, L., Gokhale, J., and Kotlikoff, L. J. (2003). The mismatch between life insurance holdings and financial vulnerabilities: Evidence. *The American Economic Review*, 93(1):354–365.

Brunnermeier, M. K. and Nagel, S. (2008). Do wealth fluctuations generate time-varying risk aversion? Micro-evidence on individuals' asset allocation. *American Economic Review*, 98(3):713–736.

Calvet, L. E. and Sodini, P. (2014). Twin picks: Disentangling the determinants of risk-taking in household portfolios. *Journal of Finance*, 69(2):867906.

Chetty, R., Stepner, M., Abraham, S., Lin, S., Scuderi, B., Turner, N., Bergeron, A., and Cutler, D. (2016). The association between income and life expectancy in the United States, 2001-2014. *Journal of the American Medical Association*, 315(16):1750–1766.

Cubeddu, L. and Ríos-Rull, J.-V. (2005). Families as shocks. *Journal of the European Economic Association*, 1:671–682.

David, J. (1981). Uncertain lifetime, consumption, and dissaving in retirement. *Journal of Political Economy*, 98(3):561–77.

DeNardi, M. (2004). Wealth inequality and intergenerational links. *Review of Economic Studies*, 71(3):743–768.

DeNardi, M., French, E., and Jones, J. B. (2009). Life expectancy and old age savings. *American Economic Review*, 99(2):110–115.

DeNardi, M., French, E., and Jones, J. B. (2010). Why do the elderly save? The role of medical expenses. *Journal of Political Economy*, 118(1):3975.

DeNardi, M., French, E., and Jones, J. B. (2015). Savings after retirement:. *Working Paper*.

Dynan, K. E., Skinner, J., and Zeldes, S. P. (2002). The importance of bequests and life-cycle saving in capital accumulation: A new answer. *American Economic Review*, 92(2):274–278.

Fagereng, A., Gottlieb, C., and Guiso, L. (2016). Asset market participation and portfolio choice over the life-cycle. *Journal of Finance, Forthcoming*.

Fagereng, A. and Halvorsen, E. (2016). Imputing consumption from Norwegian income and wealth registry data. *Working Paper*.

Finkelstein, A., Luttmer, E. F. P., and Notowidigdo, M. J. (2009). Approaches to estimating the health state dependence. *American Economic Review*, 99(2):116–121.

Fiva, J. H., Hægelandb, T., Rønningb, M., and Syse, A. (2014). Access to treatment and educational inequalities in cancer survival. *Journal of Health Economics*, 36:98111.

Gale, W. G. and Scholz, J. K. (1994). Intergenerational transfers and the accumulation of wealth. *Journal of Economic Perspectives*, 8(4):145–160.

Hakulinen, T. (1982). Cancer survival corrected for heterogeneity in patient withdrawal. *Biometrics*, 38:933942.

Hong, J. H. and Ríos-Rull, J.-V. (2012). Life insurance and household consumption. *American Economic Review*, 102(7):3701–30.

Hurd, M. D. (1987). Savings of the elderly and desired bequests. *American Economic Review*, 77(3):298–312.

Hurd, M. D. (1989). Mortality risk and bequests. *Econometrica*, 57(4):779–813.

Hurd, M. D. and Mundaca, B. G. (1989). *The Importance of Gifts and Inheritances among the Affluent*. University of Chicago Press.

Inkmann, J., Lopes, P., and Michaelides, A. (2011). How deep is the annuity market participation puzzle? *Review of Financial Studies*, 24(1):279–319.

Inkmann, J. and Michaelides, A. (2010). Can the life insurance market provide evidence for a bequest motive? *Working Paper*.

Jemaland, A., Bray, F., Center, M. M., Ferlay, J., Ward, E., and Forman, D. (2011). Global cancer statistics. *CA: A Cancer Journal for Clinicians, 61(2):6990.*

Kopczuk, W. and Lupton, J. P. (2007). To leave or not to leave: The distribution of bequest motives. The Review of Economic Studies*, 74(1):207–235.*

Kotlikoff, L. J. and Summers, L. H. (1981). The role of intergenerational transfers in aggregate capital accumulation. Journal of Political Economy*, 89(4):706–732.*

Laitner, J. and Juster, F. T. (1996). New evidence on altruism: A study of TIAA-CREF retirees. American Economic Review,*, 86(4):893–908.*

Lockwood, L. M. (2014). Incidental bequests: Bequest motives and the choice to self-insure late-life risks. Working Paper.

Love, D. A. (2010). The effect of marital status and children on savings and portfolio choice. Review of Financial Studies*, 23(1).*

Maciejewski, P. K., Zhang, B., Block, S. D., and Prigerson, H. G. (2007). An empirical examination of the stage theory of grief. Journal of the American Medical Association*, 297(7):716–723.*

Menchik, P. L. and David, M. (1983). Income distribution, lifetime savings, and bequests. American Economic Review*, 73(4):672–690.*

Modigliani, F. (1988). The role of intergenerational transfers and life cycle saving in the accumulation of wealth. Journal of Economic Perspectives*, 2(2):15–40.*

Molven, O. and Ferkis, J. (2011). Healthcare, welfare and law - health legislation as a mirror of the Norwegian welfare state*. Gyldendal Norsk Forlag.*

Rockhill, B., Kawachi, I., and Colditz, G. A. (2000). Individual risk prediction and population-wide disease prevention. Epidemiol Rev*, 22(1):176–80.*

Sasieni, P., Ahmad, A. S., and Ormiston-Smith, N. (2015). Trends in the lifetime risk of developing cancer in Great Britain: comparison of risk for those born from 1930 to 1960. British Journal of Cancer*, 112:943947.*

Syse, A., Tretli, S., and Øystein Kravdal (2008). Cancer's impact on employment and earnings -a population-based study from Norway. Journal of Cancer Survivorship*, 2(3):149–158.*

A Appendix

A.1 Identifying Bequest Motive from Mortality Risk

The aim of the model is to show that for all possible realizations of mortality risk (cancer diagnosis), given initial wealth, and the same realizations of random variables effecting future utility, someone with a bequest motive will always decrease saving less than someone without a bequest motive. The model is similar to that considered by Hurd (1987).

I assume households live for T periods, and that they may or may not have bequest motives. $B = 1$ refers to households with bequest motives and $B = 0$ means no bequest motive. At time $t < T$, for a given level of wealth (w_t) and mortality risk ($m_t \in (0, 1)$), the household chooses consumption (c_t) and leaves $w_t - c_t$ to spouse or children if they die. If they do not die, they begin next period with $w_{t+1} = (w_t - c_t)(1 + r_t)$. Adding up, the value function of the problem is (to save on notation I skip mortality risk as an argument in the value function)

$$V(w_{t+1}, ; B) = \max\{U(c_{t+1}) + m_{t+1}b(w_{t+1} - c_{t+1}; B) + (1 - m_{t+1})E_{t+1}V(w_{t+2}; B)\} \quad (6)$$

where the maximization is in c_{t+1}. Utility functions have the properties: $U' > 0$, $U'' < 0$, and $U' \to \infty$ $c \to 0$, $b' = 0$ when $B = 0$, $b' > 0$ when $B = 1$, $V' > 0$ and $V'' < 0$. U' is the partial derivative with respect to the first argument, and U'' means the second derivative. Finally, $c_t^*(B)$ expresses the optimal control given bequest motive (B).

The proofs consist of two parts. First, suppressing w_t, I show that $c_t^*(0) > c_t^*(1)$ and consequently $Ew_{t+1}^*(0) < Ew_{t+1}^*(1)$ whenever $E_r r_{t+1}$ is the same for the two types. The second part shows that for all possible realizations of mortality risk (cancer diagnosis), given initial wealth, and the same realizations of random variables (r_{t+1}), someone with a bequest motive will always decrease saving less than someone without a bequest motive.

To keep the problem relatively simple, I assume that the only difference between couples and singles is the interpretation of the bequest function $b(w_t - c_t; B)$. For couples, it correspond to the utility the deceased obtain from passing on $w_t - c_t$ to the surviving spouse, whereas for the singles it represents the standard bequest function.

The method of proof is first to show that a bequest motive increases the marginal utility of wealth V' so that $V'(w_t; 1) > V'(w_t; 0)$ for $t < T$ using induction. The inequality is intuitive as a strong bequest motive will mitigate the utility gain from increases consumption for any mortality risk by offering an alternative to personal consumption. The inequality on the marginal utility

of wealth is demonstrated by induction. Further, I begin by showing that $V'(w_T;1) > V'(w_T;0)$

$$V(w_T;B) = \max\{U(c_T) + b(w_{t+1} - c_{t+1};B)\} \tag{7}$$

subject to $c_T \leqq w_T$. If $B = 0$ then $c_T^* = w_T$ (since $b'(;0) = 0$). If $B = 1$, I concentrate on the solution where $c_T^* < w_T$ and $U'(c_T^*) = b'(w_T - c_T^*;1)$.[28] From the envelope condition we know that the individual's optimal consumption policy equates the marginal utility of current consumption to the marginal utility of wealth. In the case of bequest we have

$$V'(w_T;1) = U'(c_T^*(1)) = b'(w_{t+1} - c_{t+1};1) \tag{8}$$

which gives the following inequality

$$V'(w_T;0) = U'(c_T^*(0)) = U'(w_T) < U'(c_T^*(1)) = V'(w_T;1) \tag{9}$$

in that $c_T^*(1) < w_T = c_T^*(0)$ This shows that the marginal utility of wealth is higher for households with a bequest motive. The next step of the proof is to show that if

$$V'(w_{t+1};1) \geqq V'(w_{t+1};0) \implies V'(w_t;1) > V'(w_t;0) \tag{10}$$

The first-order conditions include

$$U' - m_t b' - (1 - m_t)E_t[V(t_{t+1};B)(1 + r_t)] = 0 \tag{11}$$

For households without bequest motives, the envelope condition gives

$$U'(c_t^*(0)) = (1 - m_t)E_t[V(w_{t+1}^*(0);0)(1 + r_t)] \tag{12}$$

I first want to establish that $c_t^*(s_t;0) > c_t^*(s_t;1)$. Suppose instead that $c_t^*(s_t;0) \leq c_t^*(s_t;1)$. Then

$$\begin{aligned}
&U'(c_t^*(1)) \leqq U'(c_t^*(0)) \\
&< (1 - m_t)E_t[V'(w_{t+1}^*(0);0)(1 + r_t)] + m_t b'(w_{t+1} - c_{t+1};1) \\
&\leqq (1 - m_t)E_t[V'(w_{t+1}^*(0);1)(1 + r_t)] + m_t b'(w_{t+1} - c_{t+1};1) \\
&< (1 - m_t)E_t[V'(w_{t+1}^*(1);1)(1 + r_t)] + m_t b'(w_{t+1} - c_{t+1};1) \\
&= U'(c_t^*(1)) \text{ Contradiction since LHS must equal RHS.}
\end{aligned} \tag{13}$$

[28] Note however that we could have that $c_T^* = w_T$ even in the case of bequest if wealth is sufficiently small.

Therefore, we must have that $c_t^*(0) > c_t^*(1)\ \forall m_t \in (0,1)$. From the first order-conditions

$$\begin{aligned} &V'(w_t;0) = (1-m_t)E_t[V'(w_{t+1}^*(0);0)(1+r_t)] = U'(c_t^*(0)) \\ &< U'(c_t^*(1)) \\ &= E_t[V'(w_{t+1}^*(1);1)(1+r_t)] + m_t b'(w_{t+1}-c_{t+1};1) \\ &= V'(w_t;1) \end{aligned} \tag{14}$$

By induction we have that $V'(w_t;0) < V'(w_t;1)\ \forall t < T$. We therefore have that $V'(w_t;0) < V'(w_t;1) \rightarrow c_t^*(0) > c_t^*(1)$. Substituting the optimal control back into the budget constraint gives

$$w_{t+1}^*(1) = (w_t - c_t^*(1))(1+r_{t+1}) > (w_t - c_t^*(0))(1+r_{t+1}) = w_{t+1}^*(0) \tag{15}$$

for the same realizations of r_{t+1}. Finally we get the cross-sectional prediction

$$E(w_{t+1}^*(1)) > E(w_{t+1}^*(0)) \tag{16}$$

From the first-order conditions

$$\begin{aligned} \frac{\partial U'(c_t^*(0))}{\partial m_t} &= -E_t[V'(w_{t+1}^*(0);0)(1+r_t)] \\ \frac{\partial U'(c_t^*(1))}{\partial m_t} &= -E_t[V'(w_{t+1}^*(1);1)(1+r_t)] + b'(w_{t+1}-c_{t+1};1) \end{aligned} \tag{17}$$

Since $U' < 0$, decreasing marginal utility of current consumption is equivalent to increasing consumption, which reduces next period wealth. For households without bequest motives to always reduce their next period wealth more than households with bequest motive we must have that $|\frac{\partial U'(c_t^*(0))}{\partial m_t}| > |\frac{\partial U'(c_t^*(1))}{\partial m_t}|$. Assume for contradiction that the reverse is true and recall that $V'(w_t;0) < V'(w_t;1)\ \forall t < T$

$$\begin{aligned} &|E_t[V'(w_{t+1}^*(0);0)(1+r_t)]| \leqq |b'(w_{t+1}-c_{t+1};1) - E_t[V'(w_{t+1}^*(1);1)(1+r_t)]| \\ &< |b'(w_{t+1}-c_{t+1};1) - E_t[V'(w_{t+1}^*(0);0)(1+r_t)]| \\ &\text{Contradiction since } b'(w_{t+1}-c_{t+1};1) > 0 \end{aligned} \tag{18}$$

Therefore, on average, $w_{t+1} - w_t$ is decreasing in mortality risk (m_t), but less for households with bequest. Morality risk has no impact on savings if $E_t[V'(w_{t+1}^*(1);1)(1+r_t)] = b'(w_{t+1}-c_{t+1};1)$, which is at the point in which the marginal value of future utility equates to the marginal utility of leaving bequest today.

A.2 Institutional Details

The Norwegian State has the full responsibility for necessary specialist health services.[29] Specialized and standardized cancer treatment is promptly available, almost free of charge, and private treatment is almost nonexistent (Molven and Ferkis, 2011; Fiva et al., 2014).[30] 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. Importantly, standardized treatment available at almost no costs precludes direct relations between cancer treatment and wealth.

The general principle from inheritance law is that the surviving spouse is entitled to 25% of the estate (minimum ca. $ 50,000) and children are entitled to 75%. Further, 2/3 of the inheritance belongs to heirs, but a family member can never require more than $ 170,000 of the estate. For example, a married individual with two children and a fortune of $ 20 million can choose to leave $ 2 million ($ 1 million to each) to the children and $ 50,000 to the spouse, and the remaining as donations to non-family members.

Most often, the spouse will retain an undivided life estate in marital property, which will normally not be distributed until the surviving spouse dies. This also means that the surviving spouse does not inherit from the deceased spouse. If the deceased had any children from another relationship, such children may elect not to allow the surviving spouse to retain that portion of the deceaseds estate they are to inherit. Such children may, thus, claim their share of the inheritance to be paid from the estate.http://www.norjus.no/visartikkel.asp?art=254%20

The Inheritance Tax Act regulates inheritance tax and rates are determined annually by Parliament. Children can receive up to $ 80,000 tax-free inheritance. The next $ 56,000 is subject to 6% inheritance tax, while the remaining inheritance is taxed at 10%. Each parent can transfer a tax-free amount of about $ 6,300 per child per year. The amount of such transfers does not affect the tax-free part of the inheritance, but unused transfers cannot be transferred to next year. The possibility of reducing taxes by transferring wealth alive creates an incentive for *inter-vivos* transfers after a cancer diagnosis.

Most people of working age have group life insurance through their employer. A group life is a death insurance, with potential associated disability coverage. Subscription to life insurance varies, albeit most individuals keep their insurance until retirement age. In case of death,

[29] https://lovdata.no/dokument/NL/lov/1999-07-02-61

[30] 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.

payment is usually paid out as a lump sum that varies with insurance premiums. In 2010, approximately 2.5 million individuals had group life insurance, and approximately $ 340 million was paid out in death insurance. The number of deaths between the ages of 20-69 was about 7,600 the same year, which give a rough estimate of the average paid premium of about $ 45-90,000 (assuming a coverage of 50-100%).

A.3 Brief Description of Cancer Treatment

Growing knowledge about the genetic makeup of tumors is leading to a revolution in cancer treatment. For many years, doctors have known 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

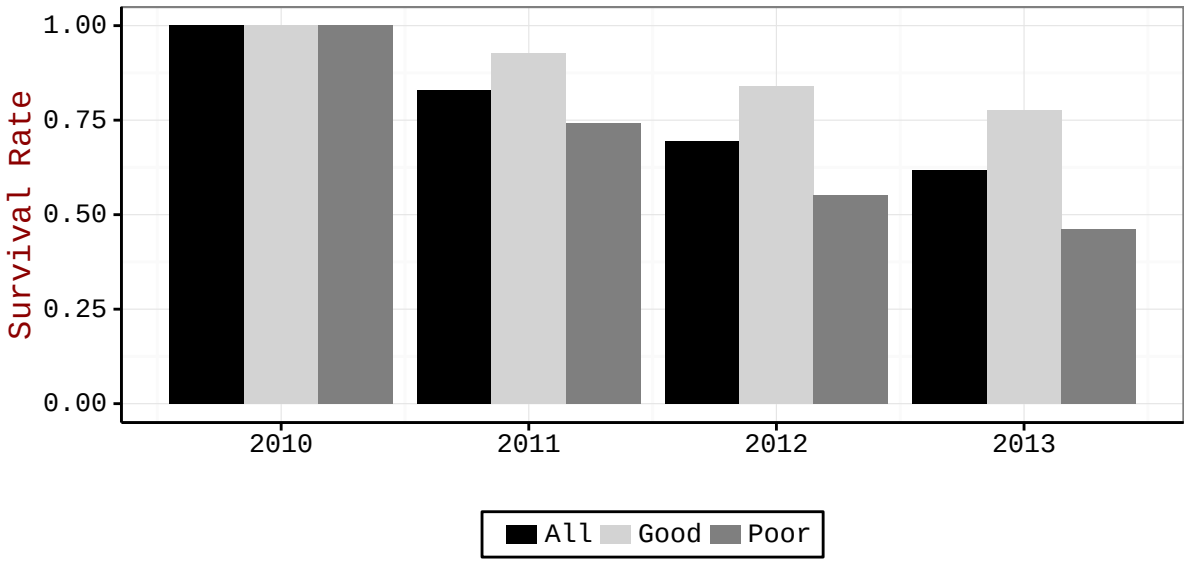

Figure 2: Absolute Survival Rate

Figure 3: Financial Wealth to Total Assets in 2009

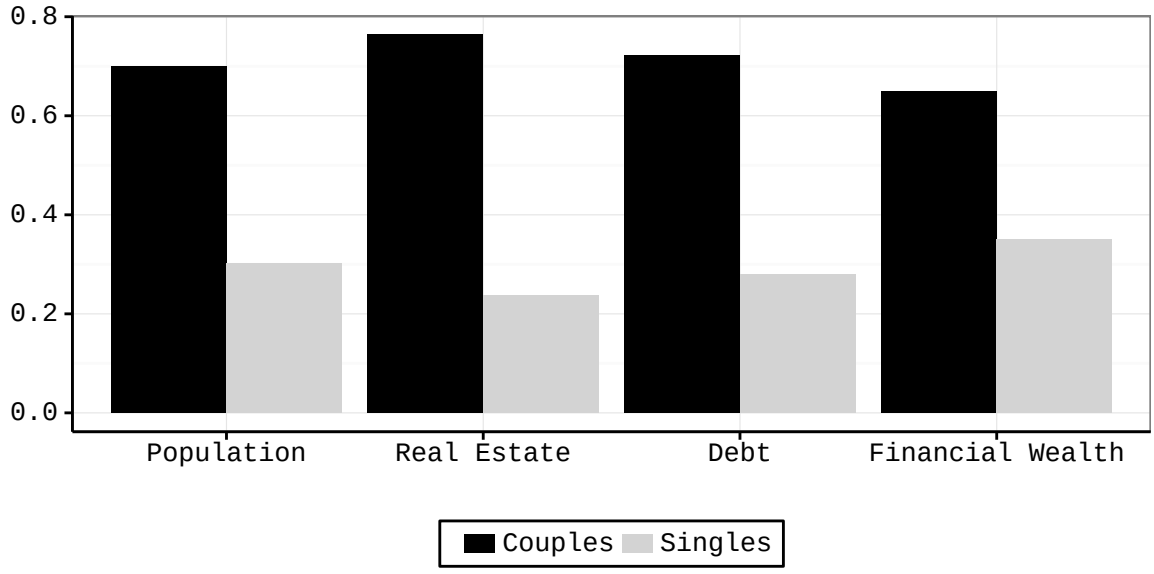

Figure 4: Relative Distributions of Family Structures and Assets

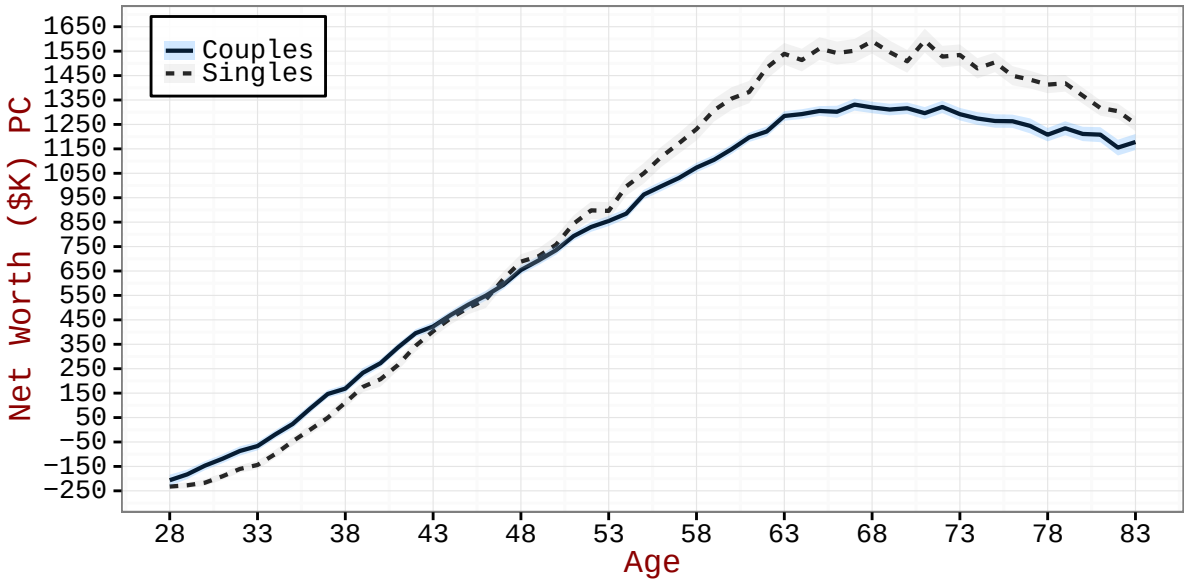

Figure 5: Net Assets in US Dollars (per capita)

C Tables

Table 1: Summary Statistics in 2009

Panel A: Summary Statistics

	Control Population (N=1270.8k)					**Cancer Population (N=13.216k)**				
	Median	Mean	STD	Min	Max	Median	Mean	STD	Min	Max
Share Couple	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	0.87	865	51	96	125	0.87	862
Real Estate	213	277	319	0	20 185	310	341	338	0	8 328
Durable Consumption	4	12	32	0	6 417	5	14	29	0	795
Debt	95	170	254	0	30 874	37	112	196	0	3 406
Leverage	0.42	4.60	21.27	0	4 524	0.12	1.96	0.00	0	539
Income	81	96	67	0	2 437	81	94	60	0	1 561

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

	Couples				Singles			
Age	50	60	70	80	50	60	70	80
Share Men	-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
Debt	0.52	-0.61	1.74	-2.18	-0.06	0.02	1.74	-1.04
Real Estate	0.34	0.52	0.66	0.15	1.23	-0.39	0.66	0.28
Durable Consumption	1.17	0.52	0.37	0.17	0.85	0.28	0.37	-1.42
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 12.31.2009 (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 12.31.2009. 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: A Direct Test of Family as Proxy for Bequest

$$w_{it+1} = \beta_c C_{it} + \beta_{cs}(C_{it}S_{it-1}) + \beta_{cso}(C_{it}S_{it-1}O_{it-1}) + \gamma X_{it-1} + \chi w_{it-1} + u_{it+1}$$

$$w_{it+1} = \sum_k \beta_c^k C_{it}^k + \sum_k \beta_{cs}^k (C_{it}^k S_{it-1}) + \sum_k \beta_{cso}^k (C_{it}^k S_{it-1} O_{it-1}) + \gamma X_{it-1} + \chi w_{it-1} + u_{it+1}$$

	ACTUAL				**PLACEBO**			
	$w_{t+1} = \ln NW_{t+1}$		$w_{t+1} = \ln FW_{t+1}$		$w_{t+1} = \ln NW_{t+1}$		$w_{t+1} = \ln FW_{t+1}$	
Model	**M1**	**M2**	**M1**	**M2**	**M1**	**M2**	**M1**	**M2**
Cancer (β_c)	0.01		0.04		0.03		-0.00	
	(1.1)		(3.4)		(2.9)		-(0.5)	
Single × Cancer (β_{cs})	-0.18		-0.15		0.02		0.07	
	-(5.2)		-(4.5)		(0.7)		(2.4)	
Single × Cancer×Child (β_{cso})	-0.17		-0.13		-0.01		-0.03	
	-(4.1)		-(3.0)		-(0.4)		-(0.7)	
Good Prognosis (β_c^G)		0.03		0.02		0.04		0.01
		(1.8)		(1.0)		(2.9)		(0.9)
Poor Prognosis (β_c^P)		0.01		0.07		0.02		-0.02
		(0.4)		(4.1)		(1.2)		-(1.3)
Single×Good Prognosis (β_{cs}^G)		-0.03		-0.01		0.08		0.06
		-(0.6)		-(0.2)		(1.7)		(1.4)
Single ×Poor (β_{cs}^P)		-0.41		-0.39		-0.03		0.08
		-(7.3)		-(6.7)		-(0.6)		(1.7)
Single×Good Prognosis×Child (β_{cso}^G)		-0.11		-0.05		-0.10		-0.01
		-(1.9)		-(0.8)		-(1.8)		-(0.1)
Single×Poor Prognosis×Child (β_{cso}^P)		-0.21		-0.17		0.07		-0.04
		-(3.2)		-(2.5)		(1.4)		-(0.7)
Regression Summary								
Life-cycle controls	Y	Y	Y	Y	Y	Y	Y	Y
R2	0.62	0.62	0.63	0.63	0.64	0.64	0.62	0.62
NOBS(k)	1254,0	1254,0	828,8	828,8	831,0	831,0	1257,8	1256.79

The table presents the estimate on the causal impact of a cancer diagnosis during 2010 on financial wealth (FW) and net worth (NW) as of December 2011. In addition to the coefficient estimates, the table includes t-statistics in parenthesis, R2, and the sample size. 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. Net Worth is defined as the sum of financial wealth, durable consumption goods, and real estate, after subtracting the gross debt. Moreover, Model M1 (first regression equation) uses all cancer cases' stage whereas the Model M2 uses only good prognoses (stage 1) and poor prognosis (stage 2 and stage 3). Life-cycle controls include a constant term, age, age2, and age3; indicator for completed college education, and its interaction with age and age2, dummy variables for gender and their interaction with age and age2, dummy variables for having children and their interaction with age, age2, and household type (single or couple), number of children, income, which is the sum of pension and labor income, and leverage defined as debt divided by total assets.

Table 3: *Inter-vivos* Transfers

$$\Delta w_{it+1} = \alpha + \sum_k \beta_c^k C_{it}^k + \beta_c^I (C_{it} I_{t-1}) + \beta^I I_{t-1} + \gamma X_{it-1}^* + u_{it+1}$$

	DAD						MOM					
Model	**M1**	**M2**	**M3**	**M4**	**M5**	**M6**	**M1**	**M2**	**M3**	**M4**	**M5**	**M6**
Cancer (β_c^A)	0.04						0.03					
	(3.0)						(1.1)					
Good Prognosis (β_c^G)		0.02						-0.01				
		(0.8)						-(0.4)				
Poor Prognosis (β_c^P)		0.07	0.06	0.07	0.07	0.06		0.12	0.17	0.23	0.10	0.11
		(3.0)	(1.7)	(1.4)	(2.6)	(2.6)		(2.9)	(2.9)	(2.9)	(1.9)	(2.6)
Poor × Single Parent (β_c^{SP})			0.02						-0.10			
			(0.5)						-(1.2)			
Poor × Rich Child (β_c^{RC})				0.01						-0.14		
				(0.0)						-(1.6)		
Poor × Rich Parent (β_c^{RP})					0.00						0.08	
					(0.0)						(0.9)	
Poor × Only child (β_c^{OC})						0.25						0.27
						(1.9)						(1.4)
Regression Summary												
Age controls	N	Y	Y	Y	Y	Y	N	Y	Y	Y	Y	Y
R2	0.01	0.01	0.01	0.01	0.01	0.01	0.00	0.00	0.00	0.00	0.00	0.00
NOBS (k)	346	346	346	346	346	346	184	184	184	184	184	184

The rows in the table provides estimates of the causal impact a parent's cancer diagnosis has on the change of the financial wealth ($\Delta w_{it+1} \triangleq \ln FW_{it+1} - \ln FW_{it-1}$) of the child (on a per child basis). In addition to the coefficient estimates, the table includes t-statistics in parenthesis, R2, and the sample size. 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. Model M1 uses all cancer cases' ($k = A$) stage whereas the Model M2 uses only good prognoses ($k = G$) (stage 1) and poor prognosis ($k = P$) (stage 2 and stage 3). Models M3-M6 use only poor prognoses and their interaction with an indicator variable which equals one if i) parent is single prior to the diagnosis (M3), ii) the child belongs to the upper 25 percentile of the net worth distribution, iii) the parent belongs to the upp 75 percentile of the net worth distribution (iv) the child has no siblings. Age controls (X_{it-1}^*) include a constant age, age2 for both parent and child.

Table 4: Heterogeneity in Households Response to Mortality Shocks

$$w_{it+1} = \sum_{k=1}^{4} \beta_{Ik}(C_{it} I_{kit-1}) + \delta_{Ik}(C_{it} Z_{it-1} I_{kit-1}) + \gamma X^{+}_{it-1} + \chi w_{it-1} + u_{it+1}$$

Interaction Term:	$I = LC$	$I = NW$		$I = NW$	$I = NW$
Households included:	**Both**	**Both**		**Couples**	**Singles**
β_{I1}	0.47	0.12	β_{I1}	0.09	-0.25
	(4.5)	(2.4)		(1.7)	-(2.2)
β_{I2}	0.15	0.12	β_{I2}	0.07	-0.18
	(3.9)	(2.3)		(1.4)	-(2.1)
β_{I3}	0.05	0.10	β_{I3}	0.11	-0.27
	(1.8)	(2.8)		(2.7)	-(2.8)
β_{I4}	0.01	0.03	β_{I4}	0.03	-0.34
	(0.4)	(1.2)		(0.9)	-(3.1)
$\delta_{I1}\|Z = Single$	-0.20	-0.47	$\delta_{I1}\|Z = Retiree$	-0.30	-0.32
	-(1.1)	-(4.2)		-(2.8)	-(1.5)
$\delta_{I2}\|Z = Single$	-0.48	-0.47	$\delta_{I2}\|Z = Retiree$	-0.32	-0.33
	-(5.5)	-(6.1)		-(4.2)	-(2.7)
$\delta_{I3}\|Z = Single$	-0.40	-0.56	$\delta_{I3}\|Z = Retiree$	-0.32	-0.31
	-(6.3)	-(8.3)		-(5.4)	-(2.5)
$\delta_{I4}\|Z = Single$	-0.63	-0.54	$\delta_{I4}\|Z = Retiree$	-0.15	-0.26
	-(10.1)	-(7.3)		-(2.9)	-(1.9)
Regression Summary					
Life-cycle controls	Y	Y		Y	Y
R2	0.62	0.62		0.62	0.62
NOBS(k)	1254,0	1254,0		1254,0	1254,0

The table presents the estimate on the causal impact of a poor cancer diagnosis during 2010 on financial wealth as of December 2011. In addition to the coefficient estimates, the table includes t-statistics in parenthesis, R2, and the sample size. All regressions have the natural logarithm of financial wealth as the dependent variable ($w_{it+1} \triangleq \ln FW_{it+1}$). 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. In the life-cycle specification, the dummy variable that takes the value of one if a household belongs to a particular place at the distribution ($I = LC$) has the following age ranges: [30-44], [45-59], [60-74], [75-85]. The corresponding ranges for net worth ($I = NW$) are: [0-25), [25-50), [50-75), [75-99]. Z_{it-1} corresponds to either the dummy variable that takes the value of one if the household is single prior to the diagnosis (S_{it-1}), or equal to a dummy variable that takes the value of one if the household is in retirement (R_{it-1}), defined as being above 67 years in 2009. In addition to the interaction variables, I_{kit-1} and Z_{it-1}, life-cycle controls (X^{+}_{it-1}) include a constant term, age, age2, and age3; indicator for completed college education, and its interaction with age and age2, dummy variables for gender and their interaction with age and age2, dummy variables for having children and their interaction with age, age2, and household type (single or couple) if both households are included in the regression ("both"), number of children, income, which is the sum of pension and labor income, and leverage defined as debt divided by total assets.

Table 5: Stage Controls

$$w_{it+1} = \alpha + \sum_{k=1}^{3} \beta_k RS_{kit} + \gamma X_{it-1} + \chi w_{it-1} + u_{it+1}$$

	Stage 1			**Stage 2 and Stage 3**			**All Stages**		
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)	0.30	0.14	0.01	0.15	0.06	-0.03	0.15	0.06	-0.00
	(2.6)	(2.3)	(0.8)	(5.0)	(1.2)	-(1.4)	(5.3)	(2.2)	-(0.3)
Singles (β)	-1.36	-0.26	0.01	-1.14	-0.27	-0.11	-1.16	-0.23	-0.03
	-(5.7)	-(1.8)	(0.4)	-(15.2)	-(2.7)	-(1.8)	-(16.2)	-(3.5)	-(0.9)

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	4.86	0.0078	11.49	<.0001	13.44	<.0001
Singles	17.45	<.0001	59.59	<.0001	102.56	<.0001

Regression Summary

Life-cycle controls	Y	Y	Y	Y	Y	Y	Y	Y	Y
Couples: R2	0.65			0.65			0.65		
Couples: NOBS (k)	660,6			659,3			665,0		
Singles R2	0.56			0.56			0.56		
Singles NOBS (k)	567,4			567,0			568,8		

The four first rows in the table provides estimates on the causal impact of cancer as a function of relative survival (RS), given cancer stage, on financial wealth and t-statistics in parenthesis. In addition R2 and the sample size are reported under "regression summary". The second panel reports the results from the F-test, which tests the null hypothesis that all the coefficients on RS are the same within each cancer stage. All regressions have the natural logarithm of financial wealth as the dependent variable ($w_{it+1} \triangleq \ln FW_{it+1}$). 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. Moreover, relative survival (RS) is defined as follow: RS_1 is a zero-one dummy variable that takes the value of one if $RS \in (0, 0.3]$, RS_2 corresponds to $RS \in (0.3, 0.7]$, and RS_3 completes the survival measure $RS \in (0.7, 1]$. Life-cycle controls age, age2, and age3; indicator for completed college education, and its interaction with age and age2, dummy variables for gender and their interaction with age and age2, dummy variables for having children and their interaction with age, age2, number of children, income, which is the sum of pension and labor income, and leverage defined as debt divided by total assets.