BREAKING BAD: HOW HEALTH SHOCKS PROMPT CRIME*

STEFFEN ANDERSEN
GIANPAOLO PARISE
KIM PEIJNENBURG

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We explore the impact of health shocks on criminal behavior by combining nearly four decades of health and criminal records for the entire Danish population. Exploiting plausibly exogenous variations in the timing of cancer diagnoses, we find that health shocks elicit a large and persistent increase in the probability of committing crime. Overall, we estimate that 1,000 diagnoses lead to 14 additional crimes per year perpetrated by either the recovering patients or their healthy spouses. We uncover evidence for two mechanisms explaining our findings. First, an economic motive leads diagnosed individuals to compensate for the loss of earnings on the legal labor market with property crimes. This effect is stronger for people that lack insurance through preexisting wealth, home equity, or marriage. Second, cancer patients face lower expected cost of punishment through a lower survival probability. Experimental evidence does not support a mechanism that operates through changes in preferences. Welfare programs that alleviate the economic repercussions of health shocks are effective at mitigating the ensuing negative externality on society.
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*Corresponding author: Steffen Andersen. san.fi@cbs.dk. Solbjerg Pl. 3, 2000 Frederiksberg, Denmark. For comments, we thank Nicola Gennaioli, Leontine Goldzahl, Tobin Hanspal, Glenn Harrison, Luigi Iovino, John Peijnenburg, Paolo Pinotti, Julien Sauvagnat, Nicolas Serrano-Velarde, Enrique Schroth, Raman Uppal, participants in the reading group "Crime: Law and Economic Analysis" at Bocconi University, and seminar participants at Aarhus University, BI Norwegian Business School, EDHEC Business School, Goethe University Frankfurt, HEC Paris, London Business School, Nova School of Business and Economics, University of St Andrews, Stockholm School of Economics, and the CEPR Household Finance seminar series.

1. INTRODUCTION

ONE OF THE MOST WIDELY ACCEPTED FACTS in criminology is that crime is predominantly a young's man game. Yet, in most developed countries, the demographics of criminals are gradually changing. For example, in the United States the share of adult arrestees 35 years of age and above has increased from 25% in 1985 to 44% in 2019.[1] Hitherto, the main emphasis of the crime economics literature has been on factors that have an early impact on criminal paths, such as education and family background (e.g., Case and Katz 1991, Cullen, Jacob, and Levitt 2006) or adverse labor market outcomes (e.g., Britto, Pinotti, and Sampaio 2021). However, the rising share of crimes committed by older adults, often with a previously clean record, calls for a better understanding of late-in-life determinants. In this paper, we document the effects of some of the most impactful and widespread adverse events over the life cycle: severe health shocks. To that end, we leverage rich administrative data from Denmark that allow us to link health and criminal records at the individual level and empirically explore whether (and why) affected individuals "break bad."

Our investigation of health shocks as trigger events is motivated by the Becker (1968) and Ehrlich (1973) theories of crime. One of the central predictions of these theories is that the decision to commit a crime depends on an array of factors that include the difference between the remuneration of legal and illegal activities, the perceived probability of punishment, and the personal attitude towards risk. Health shocks affect to an extent all these dimensions. First, health shocks impair a person's human capital and her ability to earn legal income, thereby making illegal activities, ceteris paribus, more attractive.[2] Second,

[1] See Figure 1. Similar patterns are common in other countries (see, e.g., "The Rise of the Geriatric Criminal," CBS News 2015, May 29).

[2] For instance, because individuals who have been diagnosed with an illness are less productive, work fewer hours, or are less likely to be promoted (Dobkin, Finkelstein, Kluender, and Notowidigdo 2018, Fadlon and Nielsen 2021).

health shocks decrease survival probabilities, leading to a higher discount rate when evaluating the long-term consequences of breaking the law. Third, health shocks could change a person's overall risk attitude or perception (e.g., Decker and Schmitz 2016). Notably, our empirical setting allows us to suppress potential confounding effects due to financial distress resulting from the cost of the health treatment itself (see, e.g., Dobkin, Finkelstein, Kluender, and Notowidigdo 2018), as Danes benefit from universal health insurance that covers most medical expenses.

Rather than considering all health shocks, we focus on cancer diagnoses for three reasons. First, cancer is widespread in the population—about 40% of people will develop cancer during their lifetime—and affects people of different genders, ages, and social backgrounds. Second, milder or more transitory health shocks are unlikely to alter a person's incentives. Third, cancer often affects a person's physical condition to a lesser extent than other serious diseases (e.g., a stroke) in the medium to long run. Therefore, it is comparatively less likely to impair the ability to commit crime.

A fundamental empirical challenge in establishing causal effects stems from the likely possibility that health shocks and crime are endogenously determined. For instance, lifestyle habits may correlate with the propensity for crime and co-determine an individual's health. We address this problem by exploiting variations in the timing of cancer diagnoses to compare diagnosed individuals with individuals who are born in the same year and will develop cancer at a later point but have not yet been diagnosed. At the same time, we account for the impact of unobservable invariants at the individual level. Essentially, our identification strategy exploits that, conditional on age, invariant traits, and on developing cancer at some point, *the exact timing* of the cancer diagnosis is as good as random.

We find that the probability of committing a crime increases on average by 12% following a cancer diagnosis (from the annual baseline crime rate of 0.69%). This effect is

subdued in the immediate years after diagnosis but intensifies over time and persists for over 10 years. We provide evidence that cancer leads individuals without a criminal record to violate the law for the first time and drives repeat offenders to increase the number of violations. Furthermore, we document the presence of spillover effects on the crime propensity of (healthy) spouses of cancer patients. In terms of economic magnitude the increase in crime is substantial: each thousand cancer diagnoses lead to about 14 additional crimes per year.

A challenge for interpreting these findings stems from the fact that our analysis focuses on convictions rather than criminal offenses, as the latter are not observable in the data (i.e., if the criminal is never caught). Our evidence is therefore observationally equivalent to the case in which diagnosed individuals become less skilled criminals and thus are more likely to be apprehended after cancer. We conduct a number of tests to attenuate concerns about this alternative explanation. Namely, we rely on an exogenous change in Danish welfare programs, we control for proxies of physical ability, we show an effect for the healthy partner, and we document that cancer does not impact how long a criminal avoids apprehension.

In the second part of our analysis, we seek empirical evidence for the mechanisms that link health shocks to crime. In line with the presence of an economic mechanism, we find that most of the crimes that follow a cancer diagnosis are economically motivated. Furthermore, we document that the incentive to break the law is stronger for individuals who experience a decline in income with respect to pre-diagnosis levels and lack insurance through preexisting financial wealth, home-equity (Gupta, Morrison, Fedorenko, and Ramsey 2018), or marriage (Fadlon and Nielsen 2021). Our analysis also confirms the existence of a survival probabilities mechanism: individuals for whom cancer induces an above-median decrease in survival probabilities increase criminal activity to a larger extent

than individuals with better odds of surviving. By contrast, we do not find support for a preference mechanism. Specifically, we rely on risk preference estimates from two experiments that we match to a subset of individuals in our sample. However, we do not find evidence that cancer decreases risk aversion.

In the last part of our analysis, we explore whether welfare policies can alleviate the negative externality induced by health shocks. To this end, we rely on an administrative reform that reallocated decisional authority on social policies across Danish municipalities as an exogenous source of variation in welfare support. We document that a decrease in the generosity of social security fosters an increase in the sensitivity of crime to health shocks. Individuals who experience the largest reduction in economic subsidies due to the reform increase crime rates by roughly twice as much following cancer.

This paper makes four main novel contributions. First, we document a causal effect of health shocks on criminal behavior. Hitherto, health events have been mostly overlooked by the crime economics literature with a few exceptions. Otsu and Yuen (2020) and Schroeder, Hill, Haynes, and Bradley (2011) find a contemporaneous negative correlation between self-reported measures of health status and criminal behavior. Furthermore, Corman, Noonan, Reichman, and Schwartz-Soicher (2011) show that men are more likely to commit crime if they have a child born in ill health. However, given the large number of plausible co-determinants of crime and health, previous papers fall short of establishing a causal link.

Second, our article complements a growing body of research that empirically identify turning points, i.e., pivotal moments in life that drive individuals away from crime (as proposed in sociology; see, e.g., Sampson and Laub 1995). For example, Dustmann and Landersø (2021) and Massenkoff and Rose (2020) find that events that lead to family formation such as childbirth or marriage are such turning points. Adverse health events can be viewed as "negative" turning points, as they drive individuals with a clean record to crime

(similar to job loss see, e.g., Dix-Carneiro, Soares, and Ulyssea 2018, Khanna, Medina, Nyshadham, Posso, and Tamayo 2021). Importantly, marriage, childbirth, and job loss are events that, on average, take place at a relatively young age (in Denmark at ages 34, 31, and 40, respectively). By contrast, our findings are important to explain changing crime-age profiles. In fact, after excluding people who are old enough to retire, the average age of individuals who are diagnosed with cancer is 51. Relatedly, Bell, Costa, and Machin (2021) document a change in crime-age profiles focusing on ages from 15 to 24.

Third, our study provides empirical support for rational theories of crime. These theories emphasize two main factors: income and punishment (e.g., Becker 1968, Ehrlich 1973). A stream of papers finds support for such theories by investigating the effect of job loss and access to the labor market on crime (Bennett and Ouazad 2020, Britto, Pinotti, and Sampaio 2021, Grönqvist 2011, Öster and Agell 2007, Pinotti 2017, Rose 2018, Yang 2017). Yet, the effect of job loss on crime is typically short-lived (see, e.g., Bennett and Ouazad 2020 and Rose 2018) compared to the more persistent long-run effects of health shocks. Regarding punishment, previous literature focuses mainly on the implications of a greater likelihood of apprehension (e.g., Ayres and Levitt 1998, Di Tella and Schargrodsky 2004, Draca, Machin, and Witt 2011). We provide novel evidence by documenting the role of survival probabilities in affecting the expected cost of punishment.

Finally, our paper adds to the literature on the consequences of health shocks. The conventional approach in this literature is to consider the implications for the affected individual and her close family (e.g., Dobkin, Finkelstein, Kluender, and Notowidigdo 2018, Fadlon and Nielsen 2019, Kvaerner 2019, Oster, Shoulson, and Dorsey 2013). Understanding whether health shocks are essentially private events or, on the contrary, have broader repercussions on the rest of society, is however critical to the design of optimal welfare

policies. We contribute to this literature by showing that the effect of health shocks extends beyond the personal sphere and generates a negative externality on society.

The remainder of the paper is structured as follows. Section 2 presents the institutional background and the data. Section 3 describes the empirical methodology. Section 4 documents the effect of health shocks on crime. Section 5 discusses the possible mechanisms. Section 6 presents additional robustness results and Section 7 concludes.

2. BACKGROUND AND ADMINISTRATIVE DATA

We explore the linkages between health shocks and crime using a combination of several administrative data on crime, health, income, and wealth, as well as demographic information. In this section, we briefly describe the institutional features of the Danish health and social security system and present our data.

2.1. *Institutional setting*

Two types of insurance are critical when a person experiences a severe health shock: i) health insurance, which provides coverage of medical care expenses, and ii) income insurance, which covers the loss of future income streams resulting from poor health. Health insurance is universal in Denmark and taxes pay for all medical treatment expenses during hospitalization. Post-treatment out-of-pocket health expenses are limited to co-payments for post-treatment prescription drugs and non-essential health services. Furthermore, Danes receive income insurance compensation, including both short-term sick pay and state-funded sickness benefits. We include a detailed description of the components of income insurance in Online Appendix A and we return to it in Section 5.4 when we consider the role of welfare policies.

2.2. *Administrative registry data*

We combine data from several different administrative registers made available to us through Statistics Denmark. Our data set covers the entire Danish population and contains demographic, labor, education, income, wealth, health, and crime information.

We obtain data on criminal offenses from the *Danish Central Crime Registry* maintained by the Danish National Police. The data contain records of all criminal offenses, legal charges, convictions, and non-trivial fines. All records are registered at the individual level by personal identification number and contain information about the nature of the crime, the police district, and the associated legal outcome.

Health data are from the *National Patient Registry* and from the *Cause of Death Registry*. The National Patient Registry records every time a person interacts with the Danish hospital system (e.g., for an examination or treatment). It covers all inpatient hospitalizations (1980–2018) and outpatient hospitalizations (1994–2018), in both private and public hospitals. The registry contains data on examination, treatment, and detailed diagnoses according to the International Statistical Classification of Diseases and Related Health Problems (ICD), which is a medical classification list by the World Health Organization. The Cause of Death Registry contains data on the exact cause and date of death.

All monetary values are expressed in nominal Danish kroner inflated to 2018 prices, unless stated otherwise. In 2018, the exchange rate was about DKK 6.2 per $1.

2.3. *Analysis sample*

To construct our sample, we start from the universe of individuals who are diagnosed with cancer in Denmark between the years 1980 and 2018 and retain only the [–10,+10]-year interval around the first cancer diagnosis. Furthermore, we limit our sample to people aged between 18 and 62, since during most of our sample period people over 62 could

retire and would, therefore, experience the adverse economic impact of cancer to a different degree. Table I Column 1 reports that the average individual in our sample is 48 years of age, has 13 years of education, and earns DKK 320,414 ($51,680) per annum. Roughly 60% of the observations in our sample are women. This is for two reasons. First, in our sample, women are comparatively more likely to develop cancer. Second, women tend to survive for longer periods after they have been diagnosed, thereby remaining in our sample for more years. Notably, some of the people in our sample are unlikely to break the law in a given year, as they are either re-hospitalized because of cancer after the year of initial diagnosis (the average of $Cancer\ recurrence$ is 6.19%) or in prison for more than half of the year (0.19%). In total, we have 5,007,687 observations for 368,317 distinct individuals who are diagnosed with cancer at different times over our sample period.

For comparison, Table I Columns 3 and 4 report the summary statistics for a matched sample of individuals who have never been diagnosed with cancer. Specifically, we match cancer patients on gender and age at diagnosis to compare their demographic and economic characteristics with those of healthy individuals. Overall, we find that people who are diagnosed with cancer differ to some degree from people that do not develop cancer. This evidence motivates our empirical strategy of focusing only on the former.

2.4. *Classifying criminals*

We have detailed data on charges, convictions, and penalties in terms of fines and prison sentences, as well as the type of crime committed. Figure 1, Panel A shows the distribution of convictions by age calculated for the entire Danish population in the years 1980-1985 and 2015-2018. The percentage of crimes committed by individuals aged 45 and above has increased from 13% to 22%, thereby contributing to a general flattening of age-crime profiles. This pattern is even more pronounced in the United States (Panel B). Table I shows

that the probability of being convicted of a crime in a given year for the people in our sample is 0.69%.

The richness of the data allows us to explore further the different mechanisms governing the crime–cancer relation. To that end, we classify crimes as *Economic Crimes* or *Non-economic Crimes* based on whether they are likely to be economically motivated or not. Online Appendix Table J.I illustrates how the different types of crimes map into these categories and reports the crime conviction summary statistics. The most common crime by number of convictions is store theft (9.5% of all convictions). After that, holding drugs, other theft, and minor violent offenses are the most frequent criminal offenses.

2.5. *Classifying cancer diagnoses*

We classify cancer diagnoses using ICD8 from 1980 to 1993 and ICD10 from 1994 onwards. The ICD list contains codes for diseases, signs and symptoms, abnormal findings, complaints, social circumstances, and external causes of injury or diseases. We define cancer as a malignant neoplasm, which we further classify into 15 broad categories based on its origin. Figure 2 shows that about 40% of individuals (60% of couples) face cancer during their lifetime.

3. EMPIRICAL IDENTIFICATION OF CAUSAL EFFECTS

3.1. *Staggered adoption design*

Estimating a causal response of crime to health shocks presents two identification challenges. First, the evolution of a person's health is to a large extent path dependent: people in poor health today are more likely to remain in states of poor health tomorrow. Second, health shocks are not randomly assigned to individuals. Individuals who experience health

shocks are different along a number of observable and unobservable dimensions. These covariates, in turn, may correlate with the propensity to engage in criminal activities. For example, individuals who grow up in bad neighborhoods are more likely to both develop bad health *and* violate the law (see, e.g., Kling, Ludwig, and Katz 2005 and Ludwig, Duncan, Gennetian, Katz, Kessler, Kling, and Sanbonmatsu 2012). Overall, empirical specifications that regress measures of criminal activity on health status yield biased coefficients.

To mitigate the concern that health shocks may be anticipated, we focus exclusively on cancer diagnoses. While genetics, dietary habits, smoking, exposure to pollutants, and physical exercise correlate with the likelihood of getting cancer, most risk factors have poor predictive power at the individual level. In particular, some persons in the "low risk" category will develop cancer at some point in their lives, whereas most of those who are considered at risk will remain healthy (Rockhill, Kawachi, and Colditz 2000).

In our analysis, we adopt a staggered adoption design in which we focus only on people who develop cancer and, therefore, reveal to be similar in terms of the (unknown) determinants of the health shock. Furthermore, we account for the impact of age and personal traits by including year-by-age and person fixed effects. With this procedure, we seek to compare individuals who are born in the same year but have different realizations in terms of the timing of the health shock.[3] Our identifying assumption is that the exact timing of the cancer diagnosis is unpredictable, conditional on invariant personal traits, having the same age, and on developing cancer at some point. We conduct three sets of tests to support this assumption. First, we test for the presence of pre-trends in criminal activity (see below). Second, we show that a host of likely co-determinants of criminal behavior fail to predict

[3] A potential concern is that individuals who are diagnosed with cancer at a young age may differ from individuals who develop cancer at an old age along some unobserved dimension. This, in turn, would limit the comparability of treatment and control observations. To address this concern, we also conduct our analysis separately for young and old individuals. We find effects of similar magnitude in both subsamples.

the timing of the cancer diagnosis (see Online Appendix Table J.II). Third, we compare observables in the (same) pre-diagnosis year t for individuals who are diagnosed 1 and 10 years later, respectively. We find these individuals to be observationally equivalent in terms of the distribution of key covariates when accounting for time trends, age, and gender (see Online Appendix B and Online Appendix Figure J.1).

Our empirical design necessarily incorporates a tradeoff between comparability and the possibility of identifying long-run effects. Although individuals who are diagnosed fewer years apart are more comparable, a shorter window of analysis would preclude us from estimating the response to health shocks in the long run.[4] As a compromise, we consider individuals in the $[-10, +10]$-year interval around the cancer diagnosis. Given that we estimate within-year effects, this implies that we rely on differences in the timing of diagnoses up to a maximum of 20 years apart.[5] In Section 6.2, we confirm that our results are similar when we impose that treatment and control observations are diagnosed exactly 6 years apart (following the approach of Fadlon and Nielsen 2019). Notably, this alternative estimation method is less efficient, as it uses a smaller number of valid comparisons.

3.2. *Baseline specification*

We estimate a semi-dynamic specification to recover the average treatment effect (ATE) rather than relying on the more commonly used static specification in which one dummy variable takes a value of one after a person is treated. This is because, when the re-

[4]Consider the example in which we compare two individuals who are diagnosed, respectively, in year t and year $t + 3$ (i.e., 3 years apart). This allows us to estimate treatment effects only for years $t + 1$ and $t + 2$, as in year $t + 3$ both individuals are treated.

[5]Comparison between individuals diagnosed 20 years apart are actually rare in our data, due to the high mortality rate post cancer and the fact that we truncate the age of the individuals in our sample at 18 and 62. In practice, our methodology over-weighs comparisons between individuals diagnosed close in time to each other, and under-weighs comparisons between individuals diagnosed far apart (see details below).

search design involves a multitude of treatment events, the static specification recovers the *weighted* average of all treatment effects with weights that may lack economic interpretability (see Athey and Imbens 2022, Borusyak, Jaravel, and Spiess 2021, De Chaisemartin and D'Haultfœuille 2020, Goodman-Bacon 2021, Sun and Abraham 2021). To overcome this issue, we estimate a semi-dynamic specification with a full set of post-treatment variables. Under the assumptions of lack of pre-trends (verified below) and homogeneity of treatment effects across cohorts over time (relaxed in Section 6.4), we recover the causal effect of health shocks on crime by estimating the following linear probability model:

$$C_{i,t} = \alpha_i + \beta_{t,a} + \sum_{\tau=0}^{10} \gamma_\tau \mathbb{1}\{T_{i,t} = \tau\} + \lambda X_{i,t} + \epsilon_{i,t}, \tag{1}$$

where i indexes individuals, a their age, t the calendar year, and τ the event time (i.e., the calendar year minus the diagnosis year). $C_{i,t}$ is an indicator that takes a value of one if individual i is convicted of a crime committed in year t, and $\mathbb{1}\{T_{i,t} = \tau\}$ are indicator variables for being treated. γ_τ captures the effect of cancer on crime at event time τ. We then recover the average treatment effect post cancer as the weighted average of these coefficients, $ATE = \sum_{\tau=1}^{10} w_\tau \times \gamma_\tau$, where we define each weight w_τ as the share of treated observations in each event year.[6]

We also add a number of controls. α_i are person fixed effects and $\beta_{t,a}$ are year-by-age fixed effects. The inclusion of person fixed effects allows us to estimate how a person *changes* her propensity to commit crime over time, accounting for time-invariant determinants (e.g., personality, IQ, genetic heritage, childhood experiences). Year-by-age fixed

[6]The empirical design may raise concerns including potential misspecification of the choice model and attrition bias (which we examine in detail in Section 6.2). Using simple Monte Carlo simulations of different data-generating processes, we find that our linear probability model recovers close to the true treatment effects (see Online Appendix C).

effects restrict the comparison to individuals who are born in the same year (as they are the same age a in the same year t).[7] In our baseline specification, the vector $X_{i,t}$ accounts for circumstances that limit the possibility of committing crime including $In\ prison$ and $Cancer\ recurrence$ controls. Importantly, we exclude from our sample the last available year ($t = 2018$), as all observations are treated, and the first cohort diagnosed in 1980, since those individuals are always treated.

3.3. *Testing for parallel trends*

Our approach relies on the identifying assumption that, conditional on (un-)observable time-invariant and observable time-varying controls, crime rates for the treatment and control groups would run parallel in the absence of a health shock. The plausibility of such an assumption boils down to whether the timing of the cancer diagnosis is as good as random in our window of analysis. We empirically test this parallel trend assumption by including a set of lead indicators in specification (1). Notably, we need to exclude at least two lead variables to avoid multicollinearity. We omit the event year before treatment ($\tau = -1$) and a number of leads distant from the treatment ($\tau < -6$). Figure 3 shows that there is no statistically significant difference in criminal activity between the treatment and the control group before the cancer diagnosis. We corroborate this claim by running an F-test on the pre-trend dummies in the model. The test cannot reject the null hypothesis that the pre-event coefficients are jointly equal to zero (F-statistic $= 0.35$, p-value $= 0.89$).[8]

[7] Notably, it is important to include year-by-age effects in our models, as crime progressively declines over time (see, e.g., Donohue III and Levitt 2001), whereas the number of people diagnosed with cancer increases, thereby inducing a spurious negative correlation between the two variables. Furthermore, age is strongly correlated with both cancer and crime (e.g., Freeman 1996, 1999 indicate that young people are more likely to break the law).

[8] We run a host of robustness checks in Online Appendix D: i) We explicitly estimate the coefficient $\tau = -1$; ii) We allow for the possibility of heterogeneous treatment effects by estimating separate coefficients for different cohorts (following Sun and Abraham 2021); iii) We estimate lead coefficients using untreated observations only (following Borusyak, Jaravel, and Spiess 2021); iv) We use the procedure of De Chaisemartin and D'Haultfœuille

The findings above validate our empirical design and mitigate concerns that individuals in our sample anticipate cancer diagnoses. Note that the coefficients in Figure 3 should only be used to evaluate the absence of pre-trends, as the specification including leads does not estimate the treatment effects efficiently (Borusyak, Jaravel, and Spiess 2021). Therefore, in the remainder of the paper we exclude all pre-treatment indicators and estimate semi-dynamic specifications in which the average pre-diagnosis crime rate is our baseline. We re-estimate all specifications in the paper including pre-treatment indicators and report F-tests on the pre-event coefficients in Online Appendix Table J.III.

4. MAIN RESULTS

4.1. *The effects of cancer on crime*

Table II reports the estimates for the effect of cancer on crime. Column 1 reports the coefficients estimated using Equation (1). In the year of the cancer diagnosis ($\tau = 0$) criminal activity declines relative to the pre-cancer period. The main reason for this initial decrement is intuitive: undergoing cancer treatment is physically strenuous and forces a cancer patient to visit or remain at the hospital for long periods. Furthermore, savings accumulated before the diagnosis may delay adverse economic repercussions. Overall, in the short run, health shocks reduce the likelihood of engaging in criminal activities.

However, we find a positive and economically substantial long-term impact of cancer on crime, which more than compensates for the initial reduction. Our estimates of Equation (1) indicate that, after event time $\tau = 0$, the probability of violating the law surges

(2021) to estimate placebo treatments. These results are reported in Online Appendix Figure J.2. Finally, v) we follow the procedures outlined in Rambachan and Roth 2021 and test the robustness of our results against deviations from a linear trend (Online Appendix Figure J.3). In all cases, we find no evidence of differential trends prior to the cancer diagnosis.

progressively, becoming higher than the pre-cancer baseline two years after the diagnosis (statistically significant at a 5% significance level). From event time $\tau = +3$ onward, the effect on crime is statistically significant at the 1% level and ranges from 0.08 to 0.20 percentage points. The effect increases sharply in the first five years after the diagnosis and stabilizes thereafter. To summarize these effects, we calculate the average treatment effect (ATE) post diagnosis as the average of all post-event coefficients weighted by the sample size of the observations treated at each corresponding event period. We obtain a value of 0.085 percentage points (significant at the 1% level): cancer patients are thus 12% more likely to commit a crime after they are diagnosed with cancer with respect to the baseline of 0.69 percentage points. This finding indicates that health shocks are trigger events that foster criminal behavior. In Section 6, we consider that criminal ability might decrease after cancer and evaluate the impact of attrition.

The findings above have direct implications for the change in crime-age profiles documented in Figure 1. Table II shows that, on average, an individual needs to survive two years or longer post diagnosis for us to find an increase in criminal behavior. Thanks to a series of landmark discoveries and advances in cancer treatment, the percentage of patients who survive at least 2 years post diagnosis has increased substantially over time. Specifically, it went from 57% in the first years of the sample to 74% in the final years of the sample. Over the same period, the generosity of social policies has diminished substantially, exacerbating the adverse economic effects of cancer.[9] In line with these arguments, we find that the ATE is 0.06 percentage points in the first half of the sample and 0.10 percentage points in the second half (Online Appendix Table J.IV). In Section 6.4, we consider that

[9]The average income increase for the 10 years after cancer relative to the 5 years before went from 15% to 5%.

the intensity of the treatment may be heterogeneous across diagnosis cohorts by estimating separate coefficients for each cohort.

We further explore whether health shocks have spillover effects on the criminal behavior of the spouse of the diagnosed individual. Column 2 of Table II shows that the cancer diagnosis increases the likelihood that the healthy partner breaks the law by 0.05 percentage points (statistically significant at the 5% level), roughly half the magnitude of the baseline estimate. This finding is in line with the literature that shows that families mitigate negative income shocks, for instance, because the spouse may increase her labor supply to compensate for the diminished income at the household level (Fadlon and Nielsen 2021).

To provide an estimate of the overall impact of cancer on crime, we consider single individuals and couples jointly in Column (3).[10] The dependent variable takes a value of one if the cancer patient (irrespective of whether single or married) *or her spouse* is convicted of a crime. We find that a cancer diagnosis increases the likelihood that a member of the household is convicted by 0.11 percentage points. This translates to 8.2 additional criminal convictions per year for each thousand cancer diagnoses (details are in Online Appendix E). In terms of criminal offenses, each thousand diagnoses lead to 14 additional crimes annually (as, in Denmark, criminals are convicted of 1.7 crimes on average). Under the assumption that cancer patients are equally likely to be convicted as non-cancer patients, this number can be inflated to 130 additional crimes each year, as only 10.6% of crimes lead to convictions (from our own calculations).

4.2. *Extensive vs intensive margin*

Do individuals with a clean record start violating the law because of cancer? To estimate the extensive margin effect of cancer diagnoses, we run a specification that replaces our

[10]The number of observations is larger than in Column (1) due to the presence of widows.

baseline crime variable with a first-time crime indicator variable ($First\ Crime$). Column 1 of Table III shows that after a cancer diagnosis individuals are 0.027 percentage points more likely to commit their first infraction (9% more than the average of 0.29 percentage points).

We measure the intensive margin effect by estimating how the cancer diagnosis impacts crime *conditional on being a criminal* (i.e., we exclude all observations from individuals who are never convicted).[11] This implies that we estimate the average crime rate—the number of years during which crimes were committed over the number of years in the sample—of criminals before vs. after the cancer diagnosis. Column 2 of Table III reports the coefficients for the average impact of cancer. We find an effect of 0.41 percentage points: 7% more than the average rate among criminals of 6.02 percentage points. Overall, we conclude that health shocks elicit a response both at the intensive and the extensive margin. Yet, relative to the baseline, the effect is larger for people who have never violated the law before. This evidence supports the claim that health shocks are trigger events.

5. WHY DOES CANCER PROMPT CRIME?

Guided by the theoretical framework that we develop in Online Appendix F, we conjecture that a number of different mechanisms concur in explaining the effect of health shocks on crime. A financial motive may induce individuals to mitigate the loss in human capital by seeking illegal revenues (economic mechanism). This motive emerges as cancer has a

[11] Notably, we do not restrict our sample to individuals who commit a crime *before* the cancer diagnosis only. In fact, if crime were randomly distributed over time and we would truncate the sample in this way, we would negatively bias our coefficient of interest. This is easily illustrated by an example. Consider individuals who all live the same number of years and are all diagnosed with cancer after they live half of their lives. Let us also assume that one crime is randomly assigned to each individual in a given year. If we restricted our sample to those individuals who (by chance) have a crime assigned in the first part of their life, we would incorrectly estimate that cancer decreases their probability of committing crime to zero (as we would exclude from the sample all people who commit crime after cancer).

long-lasting effect on income: Figure 4 shows that *Total income* declines on average by about DKK 13,000 in the diagnosis year and never fully reverts to the pre-cancer trajectory (broadly in line with findings from Dobkin, Finkelstein, Kluender, and Notowidigdo 2018, Fadlon and Nielsen 2021, and García-Gómez, Van Kippersluis, O'Donnell, and Van Doorslaer 2013).[12] Furthermore, decreased survival probabilities might increase time discounting and therefore reduce the expected cost of future punishment (survival probabilities mechanism). Finally, cancer may alter risk preferences (preference mechanism). For instance, some individuals may become less averse to risk or perceive risk differently. Recall that in our setting all cancer patients have medical insurance. Therefore, an out-of-pocket-medical-expense channel—i.e., a scenario in which cancer patients violate the law in order to pay their medical bills—is highly unlikely.

5.1. *Economic mechanism*

To disentangle the scenarios outlined above, we separate the broadest definition of crime into two narrower categories: *Economic* and *Non-economic Crime*. The former includes only crimes that are likely motivated by economic reasons (e.g., theft, burglary, or drug dealing). The latter consists of crimes that are unlikely to be motivated by a monetary incentive (e.g., sexual violence or vandalism). Our empirical design is motivated by the following consideration: if our findings were solely the result of an economic motive, the effect should be driven by an increase in economic crimes, while non-economic crime after cancer should either decline or remain steady.

The results in Table IV, Panel A document an increase of both economic and non-economic crime. Of the additional crimes prompted by cancer, economic crimes are three

[12] The average treatment effect is a drop in income of about DKK 6,000 per year. Notably, conditional on facing a decline in income, the effect is substantially larger.

times more prevalent: we find a 0.054 percentage point increase in economic crimes vs. a 0.014 percentage point increase in non-economic crimes.[13] Online Appendix Table J.III reports the results of F-tests obtained from a similar econometric specification that is, however, augmented with a set of lead variables, to exclude the presence of pre-trends. In Panel B, we employ a classification framework of Statistics Denmark through which crimes are sorted into three categories: i) *Property crime*, such as burglary, theft, and fraud; ii) *Violent crime*, including homicide, simple violence, and assault; and iii) *Sexual crime*, such as, rape, incest, and sexual offenses against children. Panel B reports that property offenses increase significantly, while the incidence of sexual and violent offenses does not change.

Next, we explore the heterogeneity in responses to cancer on the basis of the socio-economic background of the cancer patient. Figure 5 shows that there is little difference in crime rates between people who have above- and below-median income *levels* in the year before treatment. We further sort people based on whether they experience a decrease in average income in the first 6 years after cancer with respect to the pre-diagnosis year.[14] Individuals who experience a loss of income are more likely to commit crime after cancer. This finding further supports the existence of an economic mechanism and suggests that the individuals whose human capital is affected the most seek additional revenues in the illegal labor market.

Furthermore, Figure 5 reports that the increase in criminal activity is driven by individuals who do not own a home and have below-median financial wealth before the diagnosis, thereby suggesting that financial wealth and home equity provide a cushion (in line with

[13] Notably, we make sure that we are not just picking up an increase in non-economic crimes that are committed jointly with economic crimes by excluding convictions that involve both economic and non-economic crimes. Results remain analogous, see Online Appendix Table J.V.

[14] This analysis is potentially prone to endogeneity concerns, as we sort individuals on the basis of their income post health shock. We address this concern in Section 5.4.

Gupta, Morrison, Fedorenko, and Ramsey 2018). Furthermore, we find larger treatment effects for lower-educated, non-married men. Together with our finding that cancer patients' spouses break the law, this latter finding suggests that, in couples where a woman is affected by a health shock, the healthy man carries out the criminal offense. Finally, we find economically significant results for both younger and older individuals and that the treatment effect is stronger for individuals who have previous exposure to crime through a family member (parent, sibling, partner, child, or in-law) who violated the law. This result is consistent with Case and Katz (1991) who show a link between youths' propensity for crime and the criminal activity of older family members.

Overall, these results suggest that the decline in human capital following cancer is of first-order importance in explaining the increased incidence of crime. This is in line with the theoretical work that posits that lower human capital reduces the opportunity cost of crime (Becker 1968 and Ehrlich 1973). From a policy perspective, addressing the economic rationale behind health-shock-induced behaviors can mitigate the incidence of crime. In Section 5.4 we develop this argument further by exploring the effect of a change in social assistance schemes on the cancer–crime relationship. We also investigate in greater detail the increase in non-economic crimes by considering a psychological distress channel (see Section 5.3).

5.2. *Survival probabilities mechanism*

Health shocks negatively impact survival probabilities. In a dynamic crime and punishment framework in which crime today is discouraged by punishment tomorrow, a lower survival probability leads to discounting at a higher rate the long-term consequences of breaking the law (see Online Appendix F). A natural implication of this argument is that a sharper decline in survival probabilities should result in a stronger incentive to violate

the law. We investigate the importance of this channel by exploiting cancer's differential impact on survival probabilities based on the type of cancer and individual characteristics. More severe types of cancer reduce survival probabilities to a larger extent and, therefore, should elicit a stronger response in terms of criminal activity.

To investigate this channel, we predict declines in 5-year survival probabilities on the basis of the type of cancer, the period of the cancer diagnosis, and the age, gender, and marital status of the diagnosed individual.[15] We then conduct our analysis separately on two subsets of individuals who face high (respectively low) survival probabilities at diagnosis. Importantly, we rely on different thresholds by gender to define the two subsets, resulting in an equal share of men and women in both subsets. This is to avoid picking up a gender effect, as men are comparatively more likely to face a large decline in survival probability than women. Furthermore, we control for the effect of income in our specifications to shut down the economic mechanism outlined above. Specifically, we want to make sure that we are not capturing the fact that more severe types of cancer lead to a larger decline in income. We discuss the procedure for the estimation of survival probabilities in detail in Online Appendix G.

Figure 6 shows that the crime reduction is larger for those individuals whose survival probabilities are affected the most in the year of diagnosis.[16] Yet, in the long run, these are the only individuals who commit more crimes. Notably, the long-term increase in criminal propensity more than compensates for the initial decline. This set of results supports the existence of a survival probabilities channel and confirms the importance of the perceived cost of punishment as a deterrent against crime. Our finding complements previous

[15] We consider a five-year period because this is standard in the medical literature.

[16] In Figure 6 standard errors are clustered at the person level. In Online Appendix Figure J.4, we present results obtained with bootstrapping to account for the fact that estimates are based on a two-stage procedure.

research that establishes that a police presence discourages criminal behavior (Di Tella and Schargrodsky 2004, Draca, Machin, and Witt 2011, Lochner 2007) by showing that delayed punishment may, in turn, prompt criminal activities.

5.3. *Preference mechanism*

Health shocks are dramatic events that can influence personal preferences. In line with previous research on the impact of traumatic events (Hanaoka, Shigeoka, and Watanabe 2018, Voors, Nillesen, Verwimp, Bulte, Lensink, and Van Soest 2012), cancer may lead to a change in risk attitudes. To explore the presence of a preference channel, we link our registry data with experimental individual-level data on preferences measured in 2003/2004 and 2009/2010. Both experiments are incentivized, and the subjects, who are representative of the Danish adult population, perform between 25 and 90 tasks specifically designed to elicit risk preferences. We use as a proxy for risk aversion a dummy that equals one if the person makes a risk-averse choice in more than half of the tasks. These experiments form the basis of Andersen, Harrison, Lau, and Rutström (2008) and Andersen, Harrison, Lau, and Rutström (2014), to which we refer the reader for a detailed description of the experimental design.

Using this pooled cross-sectional data, Table V shows the relation between health shocks and risk preferences for 39 individuals who have already been diagnosed with cancer (treatment group) or will be in the future (control group). $Post\ cancer$ equals one if a person has been diagnosed in any of the previous 10 years. We find no significant relation when estimating jointly for men and women the relation between cancer and risk aversion (Column 1). However, when including a separate indicator variable for cancer interacted with the male dummy, our results indicate that women become *more* rather than *less* risk averse after cancer, whereas this effect is muted for men (broadly in line with Hanaoka, Shigeoka,

and Watanabe 2018). Hence, decreased risk aversion does not appear to be a relevant mechanism in our setting. In Online Appendix Figure J.5, we also show the effect of cancer on the likelihood of receiving speeding tickets—as an alternative proxy of decreased risk-aversion—but do not find evidence of increased risky behaviors. Overall, we do not find empirical support for a preference channel in our data.[17]

Notably, the mechanisms considered above emerge from incorporating health shocks into the Becker-Ehrlich framework (see Online Appendix F). In Online Appendix Table J.VI, we explore an additional channel that does not arise from that framework: psychological distress. Namely, cancer could prompt crime through an effect on a person's mental health. We find that, in the aftermath of the cancer diagnosis, diagnosed individuals are more likely to seek psychological help. Furthermore, we find that the cancer-crime relation for individuals who seek psychological help is 2.5 times stronger compared to those who do not receive any help. Together with the finding that part of the additional crimes due to cancer are not economically motivated, this evidence suggests that there is a psychological distress mechanism behind some of the crimes.

5.4. *The role of welfare programs: Evidence from the 2007 Danish municipality reform*

In Denmark, social policies are administered at the municipality level. In particular, local authorities can provide cancer patients with sickness benefits, pay permanent disability subsidies, allow early retirement, and/or conduct policies to reintegrate people into the labor force. The decentralization of welfare policies implies that similar people—who face the same health shock—will, to some degree, experience economic hardship differentially based on where they reside. However, as the choice of where to reside is itself endogenous,

[17] We also examine the effect of health shocks on time preferences using experimental data. While we find that health shocks are associated with higher time discounting, the estimated coefficient is not statistically significant, possibly due to lack of statistical power. This result is unreported.

the presence of local heterogeneity is not sufficient in itself to identify whether welfare policies mitigate the adverse effect of cancer on crime.

In the following, we exploit a change in the generosity of welfare policies *within municipality* to assess how it alters the economic incentives of cancer patients. On January 1, 2007, a local administrative reform went into effect, drastically reorganizing the Danish public sector. As an outcome, several administrative units were aggregated together: the previous 271 municipalities were consolidated into 98 new ones. The main rationales underlying this policy decision were the desire to increase the autonomy of local economic policy and seek efficiency gains. Yet, a byproduct of the reform was the reallocation of decisional authority on social matters across the country. We take advantage of this exogenous reallocation to explore how welfare policies mitigate the effect of health shocks on crime.

We conduct this analysis in two steps. First, we measure the municipality-level change in social support to cancer patients induced by the reform. Second, we explore how the sensitivity of crime to cancer changes for people who experienced large reductions in social support.

The generosity of each municipality is estimated pre- and post-reform on the basis of the average income replacement obtained by cancer patients residing there (we describe the estimation procedure in detail in Online Appendix H). We define as "stingy" ("generous") the municipalities with below (above) median income-replacement after cancer. Figure 7 illustrates the geography of generosity across municipalities pre- (Panel a) and post-reform (Panel b). Comparing the panels, it is immediately evident that the reform had relevant effects in a number of locations. For example, the former Vallø municipality in the eastern part of the country (see arrows) was merged with the municipality of Stevns to become the

new Stevns municipality. As a result, residents in Vallø went from being part of a generous municipality before 2007 to being part of a stingy municipality post 2007.

We explore how a reduction of welfare benefits impacts the incentive to commit crime for cancer patients. An empirical challenge stems from the fact that the reform affected undiagnosed individuals as well through, for instance, a reduction of subsidies unrelated to health conditions (e.g., maternity support). In other words, the reform impacts both our treatment group (already diagnosed individuals) and control group (individuals who have not yet been diagnosed), even though arguably to a different extent. We address this problem by estimating separate coefficients for the effects of the cancer diagnosis and the change in municipality's generosity:

$$\begin{aligned} C_{i,t} \;=\; \alpha_i \;+\; \beta_{t,a} \;+\; \sum_{\tau} b_\tau \left(\mathbb{1}\{T_{i,t}=\tau\} \times S_{t,m}\right) \;+ \\ \delta\, S_{t,m} \;+\; \sum_{\tau} \gamma_\tau \mathbb{1}\{T_{i,t}=\tau\} \;+\; \lambda X_{i,t} \;+\; \epsilon_{i,t}, \end{aligned} \tag{2}$$

where m indexes municipalities. $S_{t,m}$ is a dummy variable that takes a value of one from 2007 onwards for municipalities that become stingy, defined as municipalities in which the difference between pre- and post-reform income replacement for cancer patients falls below the sample median. The coefficient δ is the average effect on crime of living in a stingy municipality while healthy. γ_τ measures the effect of being diagnosed with cancer on crime in municipalities that are not stingy. The main parameter of interest is b_τ, which captures the *additional* effect of being diagnosed with cancer when living in a stingy municipality.

Table VI reports our findings. To increase readability, we report the coefficients b_τ in Column 2 and the coefficients γ_τ in Column 1 even though they are obtained as output of the same regression. Column 2 shows that a worsening of social support considerably

increases the effect of health shocks on crime. Specifically, while the effect of cancer on crime in municipalities that did not cut welfare is 0.07 percentage points, a reduction in social support policies fosters an additional increase of 0.08 percentage points. Hence, the cancer-crime relation is twice as strong in municipalities with less generous social policies.

Notably, in this empirical setting, we have to validate the additional assumption that, in the periods leading to the reform, there were no differential trends in how individuals responded to cancer (to mitigate the concern that cancer-induced crime had an impact on how the new municipalities were delineated). Online Appendix Figure J.6 confirms that there were no pre-trends in municipalities that cut welfare. Furthermore, in Online Appendix Table J.VII, we address the possibility that cancer patients strategically relocate to a better municipality by excluding movers from the analysis.

In general, the previous literature points to the fact that the costs of incarceration are such that prevention policies are socially desirable (Freeman 1996). An adequate welfare system appears to play an important role in this context. Our results indicate that policies that target the adverse economic consequences of health shocks are a useful tool to mitigate the effect of cancer on crime.

6. ROBUSTNESS

6.1. *Change in criminal ability*

As a number of criminals escape conviction, our dependent variable $C_{i,t}$ necessarily underestimates crime in our sample. Potentially problematic is the possibility that—by decreasing criminal ability—health shocks increase the chances of an arrest rather than the incentive to violate the law. In other words, our findings may be driven by an increase of convictions rather than an increase in crime.

Our first argument to attenuate this concern is embedded in previous results. As social welfare variations directly affect the economic incentive to commit crime, our results from the municipality reform confirm our main conclusion that health shocks prompt criminal activity. In fact, there is no reason to expect that *less* generous welfare programs should lead to *more* convictions unless crime rises too. Likewise, our finding that (healthy) spouses of individuals diagnosed with cancer also increase their supply of criminal activity is not consistent with an explanation based solely on a differential ability to avoid detection post-diagnosis.

We further run a battery of tests to attenuate concerns of a change in criminal ability explaining our findings. First, we reproduce our main results controlling for proxies of criminal ability based on the diagnosed individuals' physical and psychological condition (see Figure 8). Second, we show that there is no relationship between having had cancer and how long the criminal manages to avoid getting caught, which we proxy by the time that passes between infraction and apprehension (see Online Appendix Table J.VIII). Third, we compute the percentage of reported crimes that remains unsolved in each municipality and show that this fraction is unrelated to the number of cancer diagnoses per capita in the same municipality, thereby suggesting that cancer patients are not disproportionally more likely to be apprehended (see Online Appendix Table J.IX).

6.2. *Attrition*

Our estimates are potentially biased by selective attrition if post-cancer mortality rates are correlated with crime. Importantly, we find that the effect of cancer on crime is driven by individuals who experience a larger reduction of survival probabilities post diagnosis (see Section 5.2). Therefore, attrition should bias our estimates downward, as criminals are more likely to leave the sample. We attempt to assess the magnitude of this effect by

simulating the data generating process while mimicking our sample moments. With this procedure, we find that this bias is negligible for plausible levels of correlation between death probability and crime (see Online Appendix C). Furthermore, Figure 8 shows that our results are similar when using a balanced sample obtained by imposing that treatment and control observations are diagnosed precisely 6 years apart (following the approach of Fadlon and Nielsen 2019). We explain this balanced difference-in-differences analysis in detail in Online Appendix Section I.

6.3. *Further robustness checks*

We conduct a number of additional tests. Health shocks may drive diagnosed individuals out of the labor force, thereby leaving them with more free time to commit crime (Jacob and Lefgren 2003 and Rose 2018). Figure 8 shows that the magnitude of the effect is comparable to the baseline for people who likely had a similar amount of free time before and after cancer (i.e., those individuals who were either working or not working both before and after the diagnosis). We further address the possibility that local shocks lead to a spurious correlation between cancer and crime by adding municipality and municipality $\times$ year fixed effects to our baseline specification. Finally, we entertain the possibility that judges show more leniency towards cancer patients, thereby being more reluctant to convict. We replace our dependent variable based on crime convictions with one based on crime charges and find that, following cancer, people are 0.10 percentage points more likely to be charged with a crime (an increase of 13% relative to the sample average of 0.75 percentage points). Overall, all specifications produce qualitatively similar results.

6.4. *Heterogeneous treatment effects*

In our main specification, we implicitly assume treatment effects to be homogeneous across year-of-diagnosis cohorts as we estimate one coefficient for each relative time period. Each of these coefficients represents the *weighted* average of different treatment cohort effects. However, in the presence of time-varying intensity of treatment, weights can be non-convex and estimated coefficients can be biased (Callaway and Sant'Anna 2021, Goodman-Bacon 2021, Sun and Abraham 2021). We tackle this problem by estimating separate coefficients for different diagnosis cohort; we then recover the treatment effect as the weighted average across cohorts following Sun and Abraham 2021. Coefficients are reported in Online Appendix Figure J.2 panel (c) and are almost identical to those that we estimate with our main specification. Alternatively, we employ the differences-in-differences (DID) methodology developed by De Chaisemartin and D'Haultfœuille (2020, 2021). This methodology recovers the event study coefficients as weighted averages of DID estimators.[18] Results remain qualitatively similar (see Online Appendix Figure J.2 panel d).

6.5. *Placebo analysis*

A concern in our setting is the potential presence of unobserved events that affect individuals around the time when they are diagnosed with cancer. To mitigate this concern, we run a separate analysis in which we assign placebo cancer diagnoses to healthy individuals. Specifically, we draw random samples equal to the number of people in our main dataset from the segment of the Danish population that never develops cancer. We then assign placebo cancer diagnoses to this healthy set of individuals at the exact same age as the diagnosed individuals in our main sample. We replicate this procedure 50 times and estimate

[18] Note that this methodology does not allow to estimate all the relative time periods included in our baseline.

the average treatment effect of the (placebo) cancer diagnosis on crime in each random sample. We fail to reject the hypothesis that the ATE of the placebo cancer is greater than zero (p-value: 0.99).

7. CONCLUSION

In this paper, we provide evidence that health shocks elicit criminal behavior. Exploiting the random timing of cancer diagnoses, we establish that people who suffer severe health shocks are more likely to either commit their first offense or increase the frequency of convictions (if they did not previously have a clean record). The documented effect is subdued in the short run but increases over time as the individual recovers from medical treatment. In addition, we provide evidence of sizeable spillover effects on the crime behavior of the healthy spouses. In terms of magnitude, we estimate that each thousand cancer diagnoses lead to eight additional convictions and 14 additional crimes each year. Notably, the effect is stronger for more recent cohorts in line with the hypothesis that cancer contributes to explain flattening crime-age profiles. Overall, the results show that health shocks have negative externalities that lie outside of the private sphere.

Motivated by the rational models of crime of Becker (1968) and Ehrlich (1973), we further examine the mechanisms governing this empirical relationship. First, we find that an economic incentive motivates individuals to attenuate the loss of income by seeking illegal revenues. This is particularly the case for those individuals who are financially more at risk before cancer, because they have no supporting spouse, no home equity, and little financial wealth. The importance of an economic channel is further corroborated by the finding that the crimes committed are largely economically motivated, though also non-economic crime increases. Second, we find evidence that the increase in criminal activity is driven by

those individuals whose survival probabilities are impacted the most by the health shock and thus face lower expected cost of punishment. Finally, we test the hypothesis that cancer prompts criminal behavior through a change in personal preferences. However, we find no empirical support for this mechanism in our data. Importantly, the adverse effects of health shocks on society can be mitigated through welfare policies.

Steffen Andersen, Copenhagen Business School and CEPR

Gianpaolo Parise, EDHEC Business School and CEPR

Kim Peijnenburg, EDHEC Business School and CEPR

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TABLE I

SUMMARY STATISTICS[a]

	Main sample		Never-treated sample	
	Mean	SD	Mean	SD
	(1)	(2)	(3)	(4)
Crime (in %)	0.686	8.253	0.640	7.974
Economic crime (in %)	0.408	6.376	0.371	6.081
Non-economic crime (in %)	0.053	2.308	0.054	2.314
Sexual crime (in %)	0.014	1.170	0.015	1.222
Property crime (in %)	0.377	6.126	0.343	5.844
Violent crime (in %)	0.096	3.093	0.089	2.978
Other crimes (in %)	0.073	2.700	0.071	2.656
Partner's crime (in %)	0.587	7.639	0.571	7.538
Household's crime (in %)	0.992	9.912	0.950	9.700
Crime charge (in %)	0.751	8.636	0.704	8.360
First crime (in %)	0.289	5.368	0.273	5.220
Cancer recurrence (in %)	6.191	24.098	0.000	0.000
In prison (in %)	0.193	4.391	0.145	3.809
Male	0.406	0.491	0.414	0.493
Married	0.641	0.480	0.658	0.474
Age	47.763	9.656	48.410	9.624
Education in years	12.708	3.119	12.781	3.140
Home-owner	0.464	0.499	0.476	0.499
Total income (in 1,000 DKK)	320.414	641.368	331.944	318.254
Financial wealth (in 1,000 DKK)	156.637	367.173	169.107	383.329
Mortgage-to-income ratio	0.861	1.495	0.913	1.542
Doctors' fees (in DKK)	1953.401	2695.868	1819.600	2578.102
Psychological fees (in DKK)	83.210	660.037	73.813	659.729
Physiotherapy fees (in DKK)	193.494	1428.462	181.712	1396.461

[a]This table reports summary statistics for our main sample (Columns 1 and 2) and for a random selection of individuals who do not develop cancer in the period covered by our data (Columns 3 and 4). Individuals in the non-treated sample are matched with cancer patients on age and gender in the diagnosis year. $Mortgage\text{-}to\text{-}income\ ratio$ and $Financial\ wealth$ are winsorized at the 1st and 99th percentile. $Doctors'\ fees$, $Psychological\ treatment\ fees$, and $Physiotherapy\ fees$ are annual fees paid by the state to the health professional for the health care treatments provided to the patient. The main sample consists of 5,007,687 observations.

TABLE II

EFFECTS OF CANCER ON CRIME[a]

Years from diagnosis	Crime (1)	Partner's crime (2)	Household's crime (3)
0	-0.139***	0.041**	-0.107***
	(0.015)	(0.018)	(0.018)
+1	-0.045**	0.018	-0.027
	(0.018)	(0.020)	(0.022)
+2	0.042**	0.043**	0.056**
	(0.020)	(0.022)	(0.024)
+3	0.084***	0.026	0.080***
	(0.022)	(0.023)	(0.026)
+4	0.109***	0.038	0.111***
	(0.025)	(0.026)	(0.028)
+5	0.127***	0.067**	0.147***
	(0.027)	(0.028)	(0.031)
+6	0.152***	0.080***	0.180***
	(0.029)	(0.030)	(0.033)
+7	0.151***	0.056*	0.164***
	(0.031)	(0.032)	(0.035)
+8	0.170***	0.090**	0.213***
	(0.034)	(0.035)	(0.038)
+9	0.165***	0.069*	0.197***
	(0.036)	(0.037)	(0.041)
+10	0.198***	0.071*	0.224***
	(0.039)	(0.040)	(0.043)
ATE	0.085***	0.050**	0.110***
	(0.019)	(0.021)	(0.024)
Observations	5,007,687	3,770,262	5,566,410

[a]This table reports event study estimates for criminal activity changes in response to cancer diagnoses using Equation (1). The dependent variable is $Crime$ (Column 1), $Partner's\ crime$ (Column 2), and $Household's\ crime$ (Column 3), respectively. The average treatment effects (ATEs) are obtained as linear combinations of the post-diagnosis coefficients weighted by the relative size of the treatment group. The empirical models include person, year-by-age, in prison, and cancer recurrence fixed effects. All coefficients are multiplied by 100. Standard errors are clustered at the person level and presented in parentheses. ***, **, * indicate statistical significance at the 1%, 5%, and 10% levels, respectively.

TABLE III

NEW CRIMINALS VS RE-OFFENDERS[a]

Years from diagnosis	First crime (1)	Re-offenders (2)
0	-0.044***	-1.315***
	(0.010)	(0.130)
+1	-0.021*	-0.560***
	(0.012)	(0.164)
+2	0.030**	0.123
	(0.014)	(0.184)
+3	0.023	0.455**
	(0.015)	(0.204)
+4	0.039**	0.635***
	(0.017)	(0.223)
+5	0.049***	0.727***
	(0.018)	(0.243)
+6	0.048**	0.952***
	(0.019)	(0.260)
+7	0.052**	0.865***
	(0.021)	(0.278)
+8	0.048**	0.984***
	(0.022)	(0.299)
+9	0.037	0.899***
	(0.023)	(0.313)
+10	0.034	1.119***
	(0.024)	(0.336)
ATE	0.027**	0.412**
	(0.013)	(0.164)
Observations	5,007,687	570,502

[a]This table reports event study estimates for criminal activity changes in response to cancer diagnoses. Column (1) presents the effect of cancer on the first criminal conviction. Column (2) presents the effect of cancer on crime estimated on the subset of people who commit a criminal offense between 1980 and 2018. The average treatment effects (ATEs) are obtained as linear combinations of the post-diagnosis coefficients weighted by the relative size of the treatment group. The empirical models include person, year-by-age, in prison, and cancer recurrence fixed effects. All coefficients are multiplied by 100. Standard errors are clustered at the person level and presented in parentheses. ***, **, * indicate statistical significance at the 1%, 5%, and 10% levels, respectively.

TABLE IV

ECONOMIC MECHANISM—EFFECTS OF CANCER ON DIFFERENT TYPES OF CRIME[a]

	Panel A: Economic crime?		Panel B: Property, sexual, or violent crime?		
Years from diagnosis	Economic (1)	Non-economic (2)	Property (1)	Sexual (2)	Violent (3)
0	-0.098***	-0.005	-0.087***	-0.002	-0.019***
	(0.011)	(0.004)	(0.011)	(0.002)	(0.006)
+1	-0.018	0.003	-0.014	0.001	-0.013*
	(0.014)	(0.005)	(0.013)	(0.003)	(0.007)
+2	0.033**	0.011*	0.033**	0.004	-0.004
	(0.016)	(0.006)	(0.015)	(0.003)	(0.008)
+3	0.053***	0.008	0.053***	0.000	0.012
	(0.018)	(0.006)	(0.017)	(0.003)	(0.009)
+4	0.069***	0.012*	0.068***	-0.000	0.012
	(0.019)	(0.007)	(0.018)	(0.003)	(0.009)
+5	0.087***	0.012	0.084***	0.003	0.019*
	(0.021)	(0.007)	(0.020)	(0.004)	(0.011)
+6	0.080***	0.015*	0.073***	0.002	0.027**
	(0.022)	(0.008)	(0.021)	(0.004)	(0.011)
+7	0.093***	0.031***	0.083***	0.009*	0.023*
	(0.024)	(0.009)	(0.023)	(0.005)	(0.012)
+8	0.091***	0.023**	0.092***	0.008	0.025*
	(0.026)	(0.009)	(0.025)	(0.005)	(0.013)
+9	0.085***	0.034***	0.082***	0.005	0.018
	(0.027)	(0.010)	(0.026)	(0.005)	(0.013)
+10	0.127***	0.037***	0.125***	0.009	0.040***
	(0.030)	(0.011)	(0.029)	(0.006)	(0.015)
ATE	0.054***	0.014***	0.053***	0.003	0.010
	(0.015)	(0.005)	(0.014)	(0.003)	(0.007)
Observations	5,007,687	5,007,687	5,007,687	5,007,687	5,007,687

[a]This table reports event study estimates for changes in different categories of crime in response to cancer diagnoses using Equation (1). Panel A shows results for the dependent variables *Economic crime* (Column 1) and *Non-economic crime* (Column 2). Panel B shows results for the dependent variables *Property crime* (Column 1), *Sexual crime* (Column 2), and *Violent crime* (Column 3). The average treatment effects (ATEs) are obtained as linear combinations of the post-diagnosis coefficients weighted by the relative size of the treatment group. The empirical models include person, year-by-age, in prison, and cancer recurrence fixed effects. All coefficients are multiplied by 100. Standard errors are clustered at the person level and presented in parentheses. ***, **, * indicate statistical significance at the 1%, 5%, and 10% levels, respectively.

TABLE V

PREFERENCE MECHANISM—EFFECTS OF CANCER ON RISK ATTITUDES[a]

	(1)	(2)	(3)
Post cancer	0.208	0.214	0.417**
	(0.173)	(0.173)	(0.162)
Male		-0.0522	0.102
		(0.161)	(0.176)
Post cancer × Male			-0.420***
			(0.153)
Observations	39	39	39

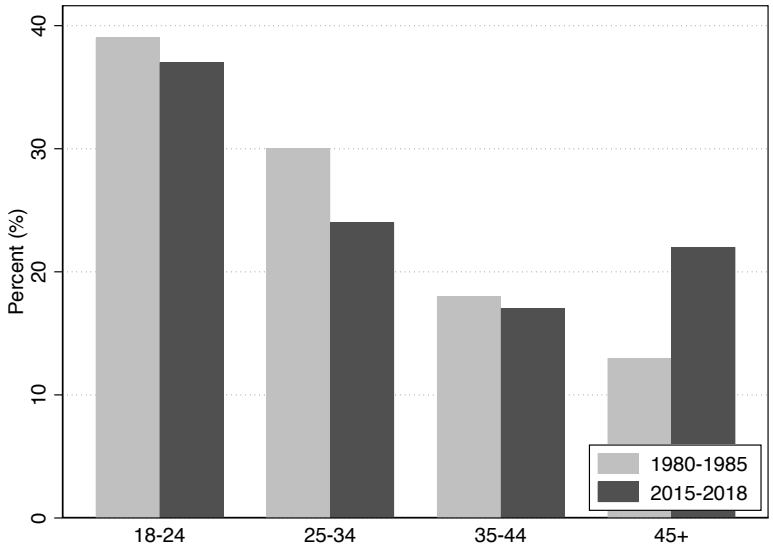
[a]This table reports cross-sectional estimates for the relation between risk aversion and having had a cancer diagnosis. Our main sample is matched with risk aversion measures obtained from experiments conducted in 2003/2004 and 2009/2010. The dependent variable $Risk\ aversion$ is a dummy that equals one if the respondent makes a risk-averse choice in more than half of the tasks. $Post\ cancer$ takes a value of one if a person has been diagnosed with cancer, and zero if a person has not yet been diagnosed with cancer but will be in the future. Standard errors are presented in parentheses. ***, **, * indicate statistical significance at the 1%, 5%, and 10% levels, respectively.

TABLE VI

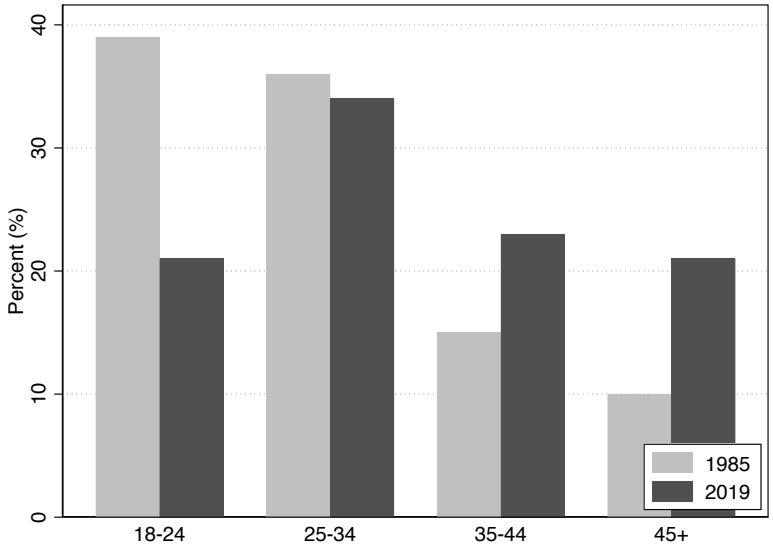
CHANGE IN WELFARE GENEROSITY AND THE EFFECT OF CANCER ON CRIME[a]

Years since diagnosis	Years from diagnosis indicator (1)	Years from diagnosis indicator × $S_{t,m}$ (2)
0	-0.151***	0.088*
	(0.022)	(0.045)
+1	-0.047**	0.019
	(0.019)	(0.043)
+2	0.030	0.080
	(0.023)	(0.054)
+3	0.082***	0.016
	(0.022)	(0.055)
+4	0.093***	0.107*
	(0.026)	(0.061)
+5	0.123***	0.022
	(0.027)	(0.059)
+6	0.125***	0.164**
	(0.034)	(0.065)
+7	0.120***	0.180**
	(0.032)	(0.076)
+8	0.143***	0.150*
	(0.036)	(0.079)
+9	0.137***	0.151**
	(0.036)	(0.073)
+10	0.176***	0.106
	(0.041)	(0.086)
ATE	0.072***	0.081**
	(0.020)	(0.040)
Observations	5,007,687	—

[a]This table reports event study estimates for the effect of the 2007 municipality reform on the relation between cancer and crime using Equation (2). $S_{t,m}$ is a dummy variable that takes a value of one from 2007 onwards for municipalities that become stingy, defined as municipalities in which the difference between pre- and post-reform income replacement for cancer patients falls below the sample median. Columns (1) and (2) report coefficients for two different sets of independent variables obtained from the same estimation. The independent variables in Column (1) are the years from diagnosis indicators and the independent variables in Column (2) are the years from diagnosis indicators interacted with $S_{t,m}$. The average treatment effects (ATEs) are obtained as linear combinations of the post-diagnosis coefficients weighted by the relative size of the treatment group. The empirical model includes person, year-by-age, in prison, and cancer recurrence fixed effects. All coefficients are multiplied by 100. Standard errors are clustered at the municipality level and presented in parentheses. ***, **, * indicate statistical significance at the 1%, 5%, and 10% levels, respectively.



(a) Denmark



(b) United States

FIGURE 1.—Offenses by age. *Notes*: Panel (a) shows the distribution of criminal convictions in Denmark by age for the periods 1980-1985 and 2015-2018, respectively. Panel (b) shows the distribution of arrestees in the United States by age in 1985 and 2019 using data from the FBI arrest statistics.

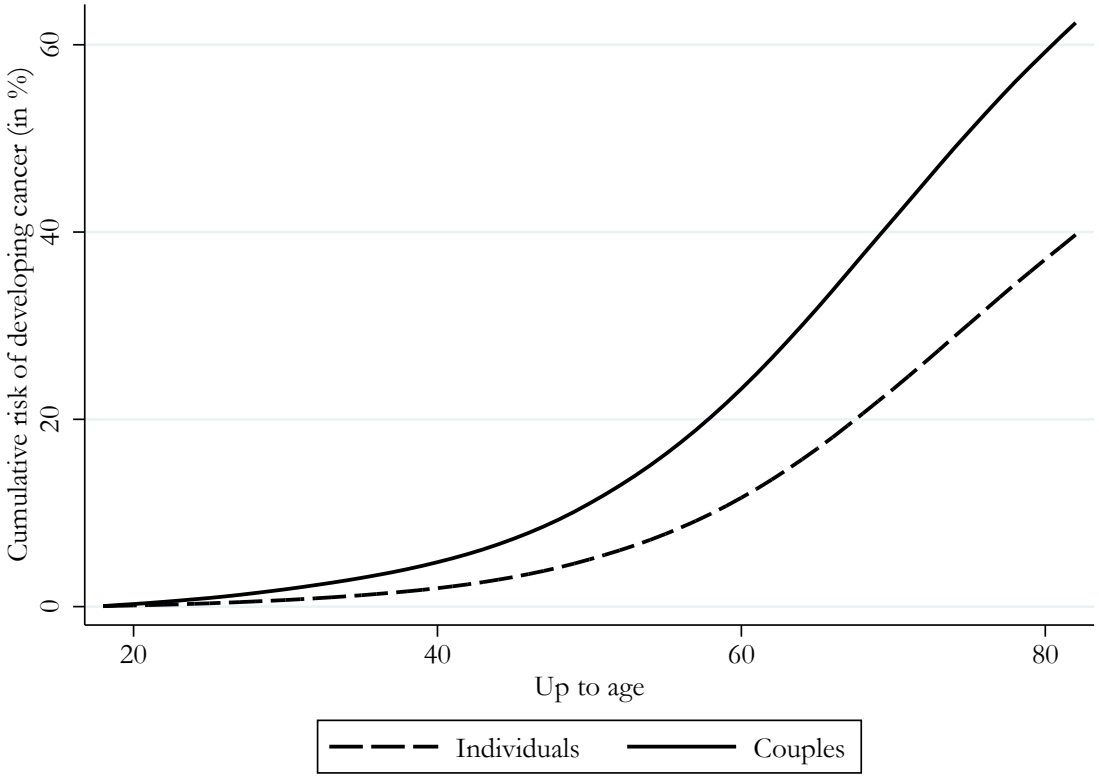


FIGURE 2.—Risk of developing cancer by age. *Notes*: This figure reports the cumulative probability of developing cancer by age. The dashed line shows the risk of developing cancer over time for an individual and the solid line for either of the partners in a couple.

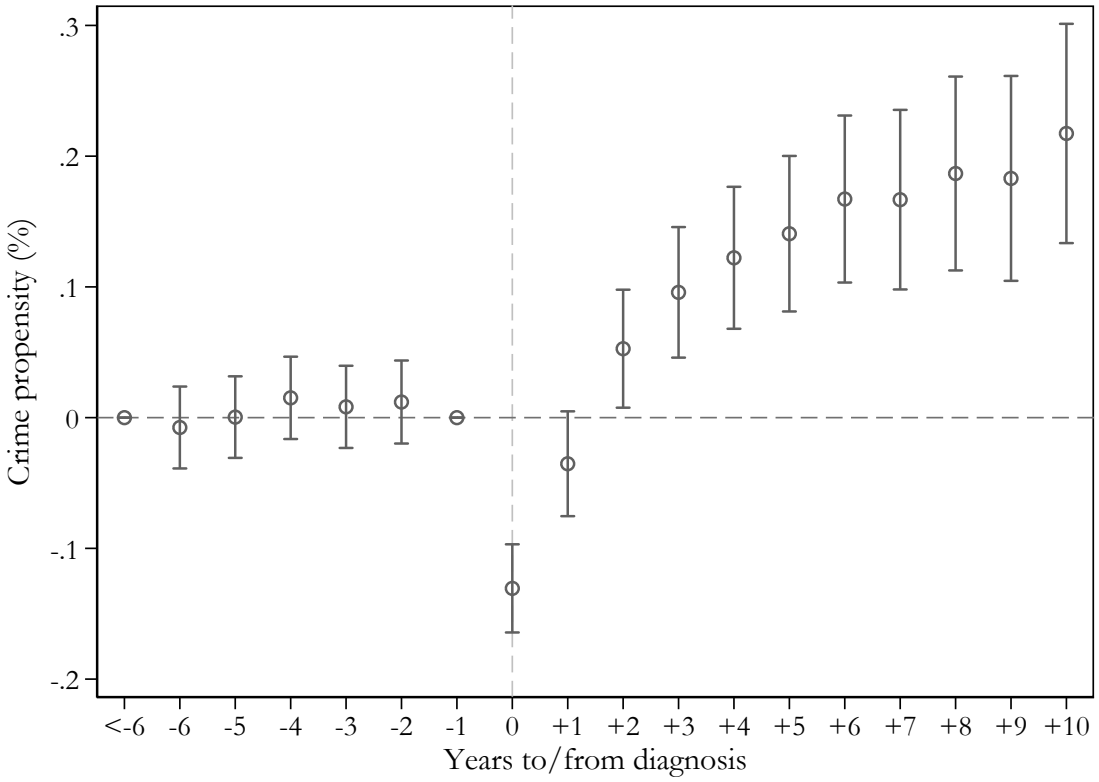


FIGURE 3.—Test for pre-trends in the relation between cancer and crime. *Notes*: This figure reports event study estimates for criminal activity changes in response to cancer diagnoses. The figure plots the estimated coefficients along with their 95% confidence interval. The x-axis denotes time with respect to the year of diagnosis. The y-axis denotes crime propensity in percentage points. The empirical model includes person, year-by-age, in prison, and cancer recurrence fixed effects. The number of observations is 5,007,687.

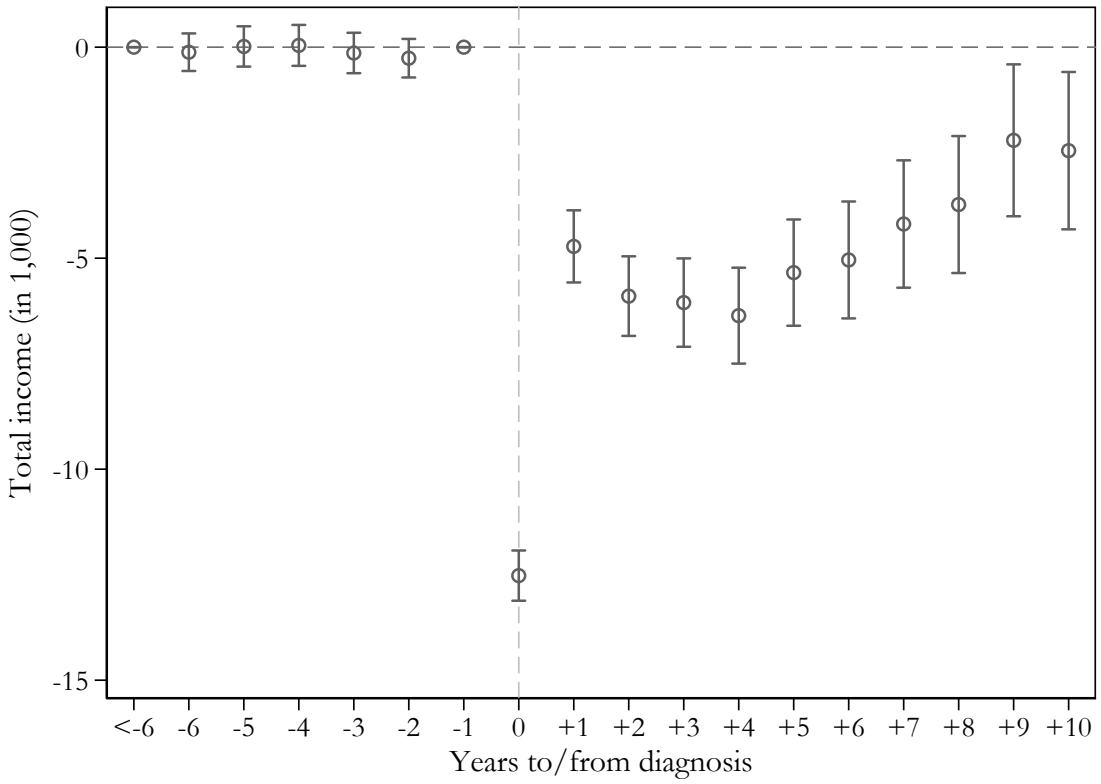


FIGURE 4.—Effect on cancer on income. *Notes*: This figure reports event study estimates for total income in response to cancer diagnoses. The figure plots the estimated coefficients along with their 95% confidence interval. The x-axis denotes time with respect to the year of diagnosis. The y-axis denotes total income. The empirical model includes person, year-by-age, in prison, and cancer recurrence fixed effects. Standard errors are clustered at the person level.

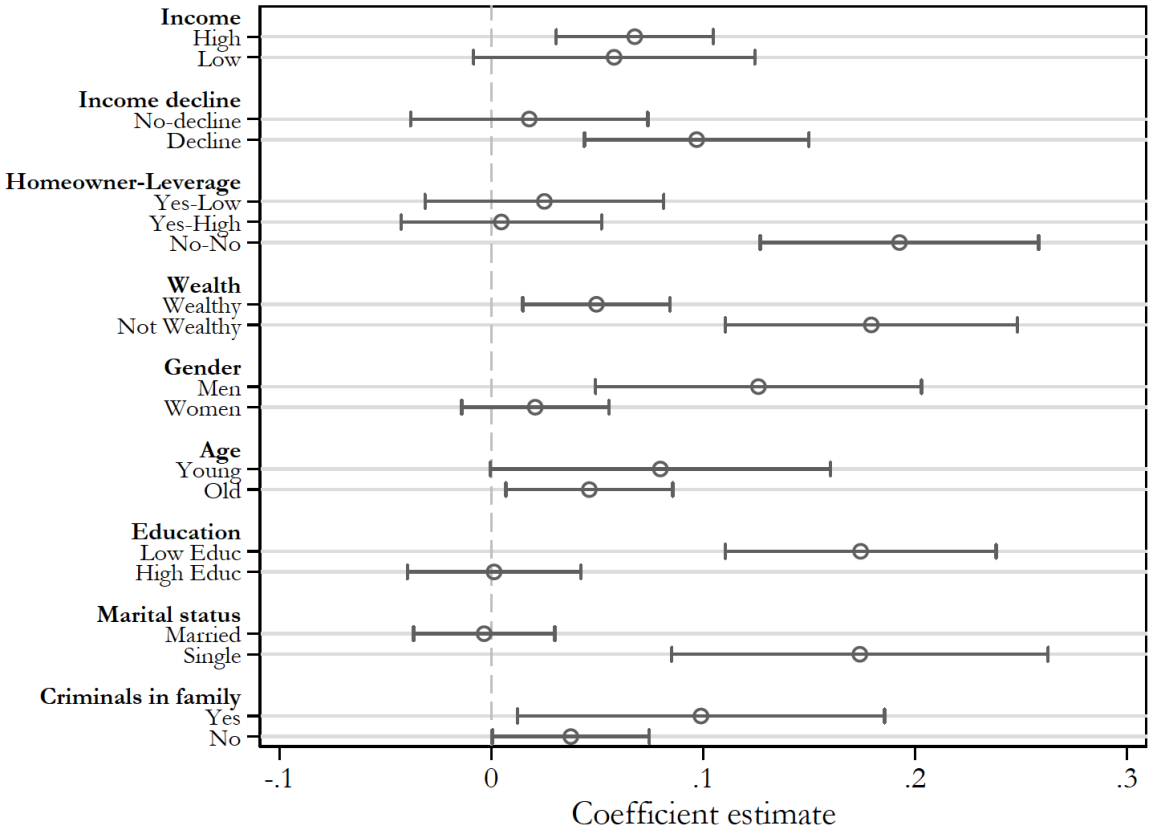


FIGURE 5.—Heterogeneous effects. *Notes*: This figure reports average treatment effects (ATEs) obtained as linear combinations of event study estimates for criminal activity changes in response to cancer diagnoses weighted by the relative size of the treatment group. Individuals are sorted into 1. above- (respectively below-) median income level in the year before the cancer diagnosis; 2. average income in the 6 years following the cancer diagnosis above (respectively below) the income in the year before the cancer diagnosis; 3. no home equity, high mortgage–to-income ratio, and low mortgage-to-income ratio in the year before the cancer diagnosis; 4. above- (respectively below-) median financial wealth in the year before the cancer diagnosis; 5. gender; 6. above- (respectively below-) median age in the year before diagnosis; 7. above- (respectively below-) median length of education in the year before diagnosis; 8. married (non-married) in the year before diagnosis; and 9. (no) criminals in the family in the year before diagnosis. Criminals in family is defined as having at least one family member (parent, sibling, partner, child, or in-law) who violated the law before diagnosis. The empirical models include person, year-by-age, in prison, and cancer recurrence fixed effects. All coefficients are multiplied by 100. Standard errors are clustered at the person level.

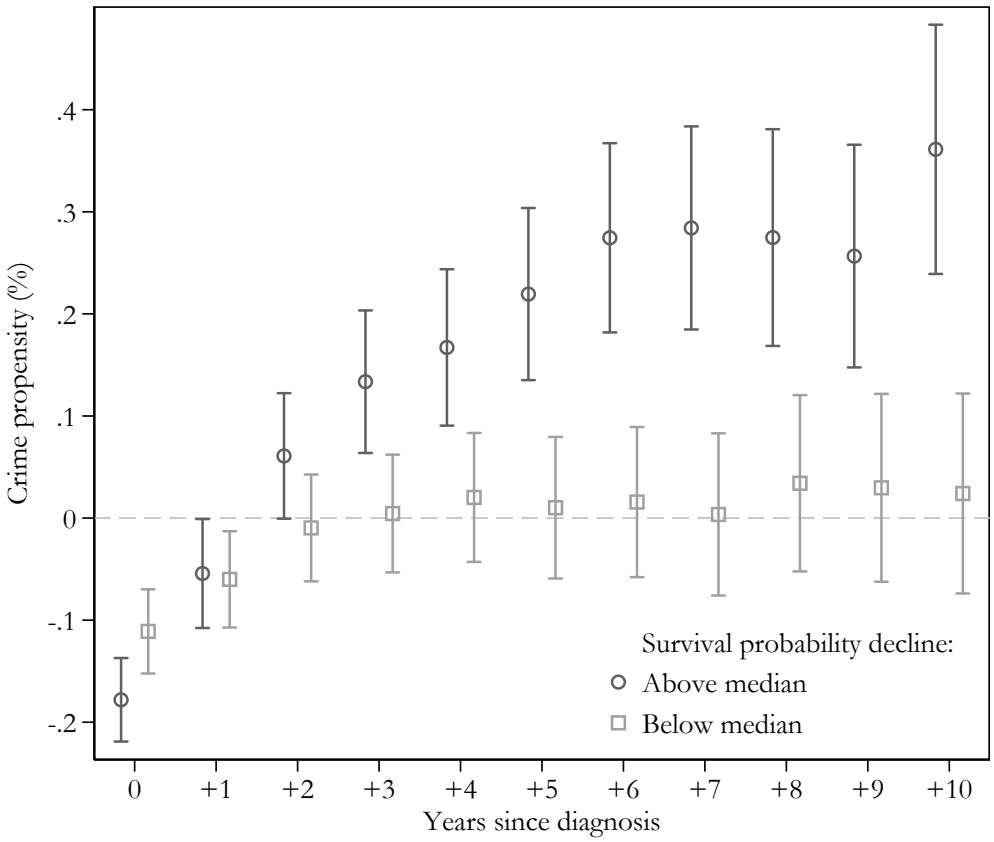
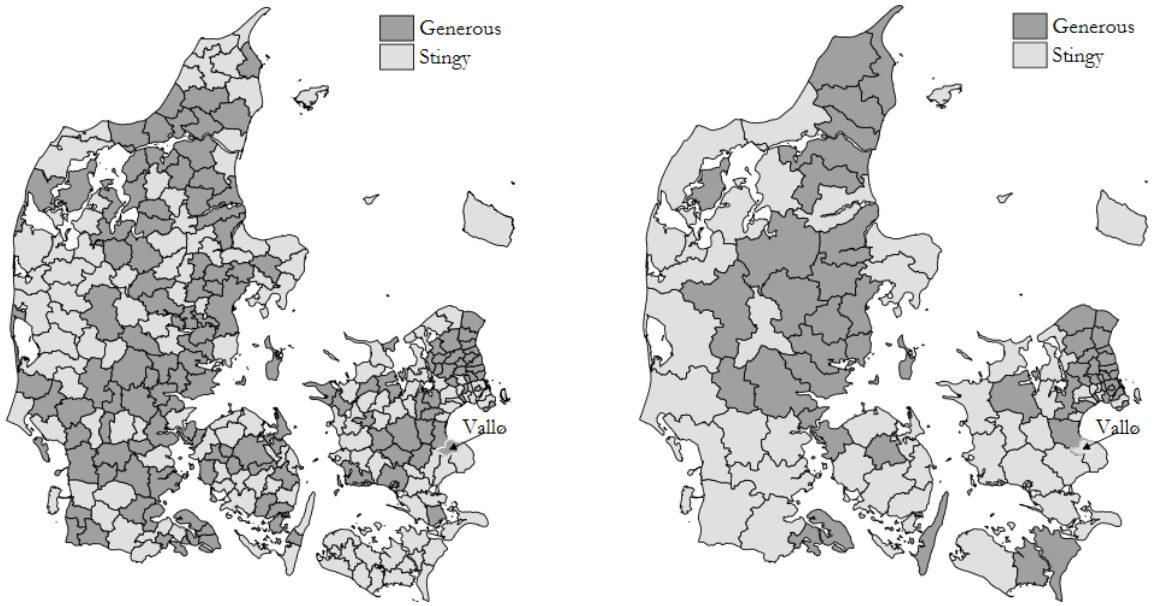


FIGURE 6.—Survival probability mechanism. *Notes*: This figure reports event study estimates for criminal activity changes in response to cancer diagnoses. The figure plots the estimated coefficients along with their 95% confidence interval. The x-axis denotes time with respect to the year of diagnosis. The y-axis denotes crime propensity in percentage points. Individuals are sorted based on whether they face an above- (respectively below-) median decline in survival probability due to cancer, using a different median threshold for men and women. The empirical model includes income controls ($Total\ income$ and $Income\ rank$) and person, year-by-age, in prison, and cancer recurrence fixed effects. Standard errors are clustered at the person level.



(a) Before reform (b) After reform

FIGURE 7.—Welfare generosity before and after the municipality reform. *Notes*: This figure illustrates the generosity of Danish municipalities before and after the implementation of the January 1, 2007 municipality reform. A generous (stingy) municipality is a municipality with above- (below-) median generosity towards people diagnosed with cancer in our sample. Values are obtained by estimating the average income replacement for cancer patients in each municipality before and after the reform. Details are presented in Online Appendix H.

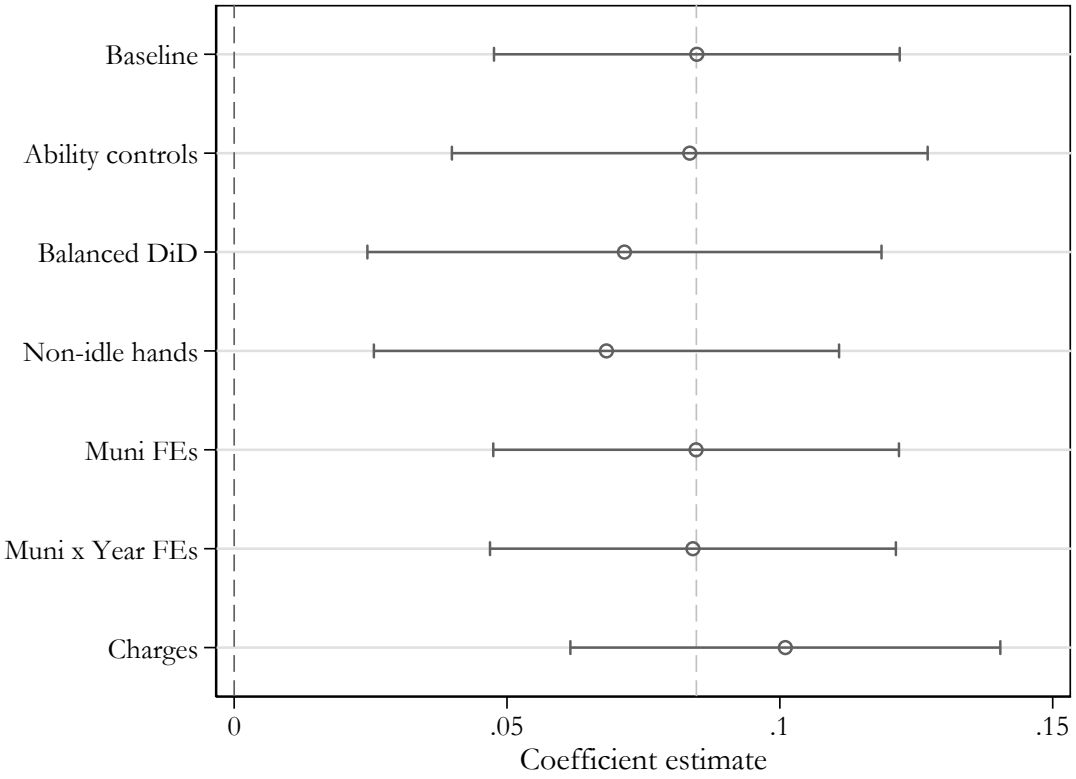


FIGURE 8.—Robustness tests. *Notes*: The figure shows average treatment effects (ATEs) obtained as linear combinations of event study estimates for criminal activity changes in response to cancer diagnoses weighted by the relative size of the treatment group. The light gray dotted line denotes our baseline ATE estimate, which is shown with confidence intervals at the top of the figure for comparison purposes. *Ability controls* includes additional controls proxying for the ability to commit crime: doctors' fees, psychological treatment fees, physiotherapy fees, and the log of these controls. These payments are made by the state to the doctor(s). *Balanced DiD* reports estimates of a balanced stacked difference-in-differences, which imposes that treatment and control observations are diagnosed exactly 6 years apart (as in Fadlon and Nielsen 2019; details are in Online Appendix I). *No idle hands* shows estimates for people who do not have more free time post diagnosis, as they are either working or not working both before and after the diagnosis. *Charges* shows the effect of cancer on $Crime\ charge$, which takes a value of one when a person allegedly commits a crime for which she is then charged but not necessarily convicted. All empirical models include person, year-by-age, in prison, and cancer recurrence fixed effects. *Muni FEs* further includes municipality fixed effects, and *Muni x Year FEs* includes municipality $\times$ year fixed effects. All coefficients are multiplied by 100. Standard errors are clustered at the person level.